

National Kidney Foundation 2010 Spring Clinical Meetings Abstracts

Acute Kidney Injury and ICU Medicine

Diabetic Myonecrosis – Rare Cause of AKI

Avinash Aravantagi, Suman Shekar, Bharat Sachdeva

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Acute Renal Failure: A Case of Burkitt's Lymphoma

J. Toby Arnold, Ana Lia Castellanos, Hanna Mawad, Bonnie Mitchell, Peter Sawaya, University of Kentucky, Lexington, KY.

Perinatal Characteristics of Premature Infants with Acute Kidney Injury

David Askenazi, Rajesh Koralkar, Akhil Maheshwari, Namasivayam

Ambalavanan, U of Alabama at Birmingham, Birmingham, AL

Eight Cases of Acute Kidney Injury Due to Vancomycin Toxicity

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Hemodialysis is Effective for Treatment of Hypothermia

Rebecca M Dahlberg, Laura Maursetter, R Michael Hofmann, Micah R Chan, University of Wisconsin, Madison, WI, USA

A Challenging Case of Partial Nephrogenic Diabetes Insipidus

Mirela Dobre, Kavita Jyotula, Andrei Brateanu

Huron Hospital, a Cleveland Clinic hospital, Cleveland OH, USA

Acute Kidney Injury is Associated with the Use of Intravenous Vancomycin in Hospitalized Patients

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Elevated BUN with Normal CR at ICU Admission Predicts Short Term and Long Term Mortality

Sabitha Eppanapally, Domingo Chang, Karthik Mahadevappa, Kevin Beier, Heidi S. Bazick, Clare Horkan, Fiona K. Gibbons, Kenneth B. Christopher. Brigham and Women's Hospital, Boston, MA

Energy Enhancement to Severe Toxicity Requiring a Transplant – A Case of Ephedra Toxicity

Monika Gandhi, Muhammad Yaqub. Division of Nephrology, Indiana University, Indianapolis, IN, USA

Acute Kidney Injury After Robotic Assisted Laproscopic Radical Prostatectomy (RARP), How Real Is It?

Sajid Melvin George, Chidi Okafor, Charles Brooks.

University Of Virginia Health System, Charlottesville, VA

Utility of SCUF in Hypotensive ICU Patients

Mohit Gupta, Rouzbeh Afsari, Stephanie Tzarnas, Neha Bansal, Simi

Shahabdeen, Nauman Shahid, Prasanna Srinagesh., Ziauddin Ahmed

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Dilemmas for a Nephrologist in a Case of Acute Leukemia

Saurabh Gupta, Sonika Puri, Beje Thomas, University of Connecticut,

Farmington, CT, USA

Acute Renal Failure Secondary to Carbidopa/Levodopa Induced Retroperitoneal Fibrosis

Hemalatha Gutta, Jorge Lamarche, Alfredo Peguero, Craig Courville, University of South Florida and James A. Haley Veterans Hospital, Tampa, FL, USA

CMV Nephritis in Native Kidneys of a Patient with Mycosis Fungoides

Amanda Hernandez, Kanwardeep Sachdeva, Charles Nguyen, Amber Podoll.

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Clinical and Immunohistological Analysis of 28 Cases of Biopsy-Proved Acute Interstitial Nephritis

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Gemcitabine Induced Hemolytic Uremic Syndrome

Atipon Kangwanpornisiri, Barbara Healey; Bassett Healthcare, Cooperstown, NY

Occupational Hazard: Leptospirosis and Acute Kidney Injury

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“We Would, and We Would Not”: A Tragic Case of Kell

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Acute Renal Failure in Poisonous Snake Bites

Thiruvengadam Muniraj, A.J. Pinevich (UPMC Mercy, Pittsburgh, PA, USA), S Chandrasekaran (Coimbatore Medical College, Coimbatore, Tamilnadu, India)

Acute Kidney Injury Secondary to Bilateral Occlusive Main Renal Artery Emboli: A Rare Presentation of A-FIB

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Spontaneous Perinephric Hematoma

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Increased Serum Adiponectin Levels With Reduced Expression of Renal Adiponectin Receptor-1 in Acute Kidney Injury (AKI)

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Spontaneous Kidney Rupture in Pregnancy Due To Cocaine Intoxication

Chike Nzerue, Mary Fleming, Sandra Torrente, Glenfield Knight, Marquette Faulkner. Dept of Medicine, Obstetrics & Radiology, Meharry Medical College, Nashville, TN, USA

Nondilated Obstructive Uropathy – An Unrecognized Cause of Acute Renal Failure in Hospitalized US Patients: Three Case Reports Seen Over Six Months in a North-Western Wisconsin Nephrology Practice

Macaulay Onuigbo¹, Kayode Lawrence², Mayo Clinic, Rochester, MN, USA & Midelfort Clinic, Eau Claire, WI, USA¹; Mount Sinai Services of the Mount Sinai School of Medicine, NY, USA²

Recurrent Acute Tubular Necrosis – A Rare Presentation of Hypercalcemia

Neena Penagaluru¹, Dayanand Makey¹, Ushir Patel¹, Sushil Mehandru¹, Neptune, NJ USA. Vivette D'Agati², New York, NY USA

Renal Failure in Critically Ill Patients with Influenza A (H1N1) Infection

Rupesh Raina, Sevag Demirjian, S.Navneethan, M. Schreiber and J. A Guzman Cleveland Clinic Foundation, Cleveland, OH

Acute Kidney Injury in H1N1/ Influenza A Virus Infection Related Deaths in Four Adults

Syed Saghir, William B. Cundiff, Mahmoud El-Khatib, N. Ganesh Yadlapalli, Charuhas Thakar. University of Cincinnati, Ohio

Biopsy Proven Acute Tubular Necrosis Due to Vancomycin Toxicity

Farheen Shah-Khan, Yashpal Kanwar, Cybele Ghossein, Chicago, Illinois, U.S.A.

Urinary Lipocalins: New Biomarkers That Predict Outcomes In Patients With Acute Kidney Injury (AKI)

Melissa Lamb Shannon, Page Moore, Prajwal Chevireddy, Cindy Davis, Ellen Satter, Kiran Nagothu, Didier Portilla, Div. of Nephrology Dept. of Internal Medicine, Dept. of Biostatistics, University of Arkansas for Medical Sciences, and Central Arkansas Veterans Healthcare System (CAVHS) Little Rock, AR, USA

Comparison of RIFLE and AKIN Classification Applied to ICU Patients in Need of Renal Replacement Therapy

R.A. Shingarev, K. Aaron, A. Tolwani. University of Alabama at Birmingham, Department of Medicine, Birmingham, AL, USA

Renal Injury In Critically Ill Patients Infected With Pandemic H1N1 Influenza A

Manish M Sood, Claudio Rigatto, Ryan Zarychanski, Paul Komenda, Amy R Sood, Joe Bueti, Martina Reslerova, Dan Roberts, Julie Mojica and Anand Kumar, Winnipeg, Manitoba, Canada

Spontaneous Tumor Lysis Syndrome Due to a Neuroendocrine Tumor

Kumar Sujeet, Alfred Anushayanathan, Hariprasad Trivedi, Medical college of Wisconsin, Milwaukee, WI

Role of Preoperative Angiotensin Converting Enzyme Inhibitors and Angiotensin Receptor Blockers in Acute Kidney Injury After Surgery

Shervin Yousefian, Silvester Kagunye, Viresh Patel, Lisa Inchani, Vincent DeBari, Chandra Chandran. Saint Joseph's Regional Medical Center, Paterson, NJ

Chronic Kidney Disease

Depression Predicts Outcomes in Diabetics but Not Non-Diabetics with CKD

Masoud Afshar¹, Robert Toto¹, Madhukar Trivedi¹, S. Susan Hedayati^{1,2}; ¹Univ of Texas Southwestern, and ²Dallas VA Medical Centers, Dallas, TX, USA

Access to Health Care in Adults Evaluated For Chronic Kidney Disease: Findings from the Kidney Early Evaluation Program

Varun Agrawal, Pranav S. Garimella, Bernard G. Jaar, Laura Plantinga, Jiuming Ye, Peter A. McCullough. Springfield MA, USA

Silent Myocardial Ischemia and Chronic Kidney Disease: The Fatal Dance

Ammar Almehti*, Mike Broce†, James B. Wetmore*

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Progressive Secondary Polycythemia in a Patient on Chronic Hemodialysis

Osama Amro, Ghayyath Sultan, Ahmed Ibrahim, Samer Alhindi, Salman Malick, Charity kankam, Jon Reisman, Case Western Reserve University/ St.Vincent Charity hospital, Cleveland, Ohio, USA

Dosing Trends over Time of Epoetin Alfa Utilization in Chronic Kidney Disease Patients Not On Dialysis: A Pharmacy Benefit Perspective

Robert A. Bailey² François Laliberté¹, Francis Vekeman¹, Mekré Senbetta², R. Scott McKenzie², Patrick Lefebvre^{1,1} Groupe d'analyse, Ltée, Montréal, Québec, Canada; ²Centocor Ortho Biotech Services, LLC, Bridgewater, NJ, USA

Drug Utilization Patterns and Costs for Erythropoiesis-Stimulating Agents in Adult Patients with Chronic Kidney Disease

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Epoetin Alfa Dosing Trend Over Time In Adult Patients With Chronic Kidney Disease: A Medical Benefit Perspective

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Epoetin Alfa (EPO) Utilization Trends in Medicare Patients with Chronic Kidney Disease (CKD) Not On Dialysis

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Recent Erythropoiesis Stimulating Agent (ESA) Utilization and Costs in Medicare Patients with Chronic Kidney Disease (CKD)

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Hemoglobin Decline Following Hematide™ Dose Interruption

Anatole Besarab^{1,2}; E. Martin²; L Ardelean²; P Pergola²; V De Silva²; S Zeig²; F Whittier²; R Zabaneh²; A Covic²; M Kaplan²; A Wiecek²; B Schiller²; R Leong³; A M Duliege³; C Francisco³; I C Macdougall²
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A Single Dose Study of Denosumab in Patients with Various Degrees of Renal Impairment

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Racial Differences in Kidney Function among Individuals with Obesity and Metabolic Syndrome: Results from the Kidney Early Evaluation Program (KEEP)

Andrew S. Bomback (New York, NY), Abhijit V. Kshirsagar (Chapel Hill, NC), Adam T. Whaley-Connell (Columbia, MO), Shu-Cheng Chen (Minneapolis, MN), Suying Li (Minneapolis, MN), Philip J. Klemmer (Chapel Hill, NC), Peter A. McCullough (Royal Oak, MI), George L. Bakris (Chicago, IL)

A Quality Improvement Project to Implement K/DOQI Guidelines at the Internal Medicine Clinic

Vivian Chukwuani, Sumeet Chavan, Olgun Esra, Shivani Bishnoi, Mahesh Borhade, Kavitha Kesari, Parul Sud. Department of Internal Medicine, McLaren Regional Medical Center, Flint, MI.

Associations of Serum Alkaline Phosphatase with Elevated CRP in Chronic Kidney Disease Are Independent of Serum 25 OH Vitamin D Levels

Sriharsha Damera², Bradley Baird², Tom Greene^{1,2} and Srinivasan Beddhu^{1,2,1} VA Healthcare System,² Department of Medicine, University of Utah School of Medicine, Salt Lake City, UT

Safety and Immunogenicity of a Novel Hepatitis B Vaccine Adjuvanted With Immunostimulatory Sequence (ISS) In Renal Predialysis and Dialysis Patients

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Corrected Serum Calcium but Not Phosphate Correlates With Change in Serum PTH in Treatment of Stages 3-5 CKD

J Ennis¹, J Asplin¹, S Donahue¹, E Worcester², and F Coe²; Litholink Corp¹ and University of Chicago², Chicago, IL, US

Physicians Using A Computer Guidance System Based On KDOQI Guidelines Appear To Regulate Systolic Blood Pressure Around The Recommended Target Of 130 mmHg

J Ennis¹, J Asplin¹, S Donahue¹, E Worcester² and F Coe², Litholink Corp¹ and University of Chicago², Chicago IL, US

Efficacy and Tolerability of Oral Iron as Initial Treatment for Iron Deficiency in Pre-Dialysis Anemia of CKD

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Chronic Kidney Disease in Hispanics: Baseline Characteristics of the Hispanic Chronic Renal Insufficiency Cohort (HCRIC) Study

Michael J. Fischer¹, Lynn Ackerson², Janet Cohan¹, Harold Feldman², Alan Go², Claudia Lora¹, Alejandro Mercado¹, Ana Ricardo¹, James Lash¹. ¹Jesse Brown VA/U. Illinois and ²CRIC Study Group, USA

Effects of Vitamine D2 Repletion in Patients with Chronic Kidney Disease Stages 3 And 4

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Is There a Difference between the Glomerular Filtration Rate (GFR) of Patients With HBV and HCV Chronic Hepatitis and Patients with Cirrhosis?

C. Gluhovschi, G. Gluhovschi, I. Sporea, S. Velciov, R. Buzas, V. Trandafirescu, L. Petrica, G. Bozdog, F. Bob, F. Gadalean, D. Cioca, C. Vernic. Nephrology, U. of Med. and Pharmacy Timisoara, Romania

Within the Broad Range of Renal Function as Assessed by Serum Creatinine, the Higher the Creatinine, Higher the Homocysteine and the Lower is HDL Cholesterol, with Resultant Higher Risk for Cardiovascular Disease

Gowda M, Alvarado A, Ahmed W, Khan A, Wang P, Glueck CJ. Cholesterol Center, Jewish Hospital, Cincinnati, Ohio

Pitfalls of Vitamin D Replacement Therapy

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Pregabalin Therapy in Refractory Uremic Pruritus

Guru PK, Ganguli A, Bains AS, Chaudhary S, Bhargava A, Lund R. Omaha, Nebraska, USA and Mohali, Chandigarh, India

Hematologic Outcomes and Dosing Patterns in Anemic Patients with Pre-Dialysis Chronic Kidney Disease (CKD) Switching from Darbepoetin Alfa (DARB) to Epoetin Alfa (EPO)

Claudia Hura¹, James Jackson², Orsolya Lunacsek², Robert A. Bailey³, R. Scott McKenzie³

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Short-Term Effect of Dietary Phosphorus Restriction and Lanthanum Carbonate on FGF23 in Chronic Kidney Disease Patients

Tamara Isakova,¹ O Gutiérrez,² K Smith,¹ M Epstein,¹ N Patel,² H Jüppner and M Wolf.² Massachusetts General Hospital, Boston, MA,¹ and University of Miami Miller School of Medicine, Miami, FL, USA.²

Kidney-Related Effects of Emerging Medications for Heart Failure

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A Cost-Effectiveness Model of Phosphate Binders for the Treatment of Hyperphosphatemia in Chronic Kidney Disease (CKD)

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Role of Acidosis in the Management of Anemia of Chronic Kidney Disease

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EPO and Hematide™ Requirements Differ in ESA Hyporesponsive HD Patients

Peter Kotanko^{1,2}; B Schiller²; S Zeig²; P Pergola²; F Whittier²; R Zabaneh²; A Besarab²; M Kaplan²; A Covic²; I C Macdougall²; A M Duliege³; H Tang³; N Levin^{1,21} Renal Research Institute, New York, NY, USA; ²AFX01-03 and -07 Hematide Study Groups; ³Affymax, Inc., Palo Alto, CA, USA

Cardiovascular Disease Prevalence in the Hispanic Chronic Renal Insufficiency Cohort (HCRIC) Study

James Lash,¹ Ana Ricardo,¹ Matt Budoff, Claudia Lora,¹ Martin Keane,² Michael Fischer,¹ Tom Stamos,¹ Harold Feldman,² Alan Go²

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Kidneymobile: on the Road to Healthier Living

Nancy LePain, Laurie Ruggiero, Nicole Sisen, Willa Lang, Kate Grubbs-O'Connor, Chicago, IL USA

Warfarin Dosing in Patients with Impaired Renal Function

Nita Limdi, Aaron Anderson, Mohit Limdi, Larisa Cavallari, Mellissa Baird, Michael Allon, Mark Beasley
Altamont School and University of Alabama at Birmingham, AL.

A Retrospective Cohort Study of Trends in Hemoglobin (HB) Levels among Erythropoiesis-Stimulating Agent (ESA)-Treated Chronic Kidney Disease (CKD) Non-Dialysis (NOD) Patients in the Fresenius Medical Care North America (FMC-NA) CKD Data Registry

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Comparative Effectiveness of Paricalcitol versus Calcitriol Treatment in Chronic Kidney Disease [CKD] Patients with Secondary Hyperparathyroidism [SHPT]

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Elevated Human Chorionic Gonadotrophin (HCG) Levels in Patients with Chronic Kidney Disease

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Symptoms and Impacts Reported By Patients with Type 2 Diabetes and Nondialysis Chronic Kidney Disease Related Anemia

Mary-Claire Miller¹, Antonia V. Bennett¹, Shravanthi R. Gandra², Eldrin F. Lewis, MPH³, Mona L. Martin¹, Donald L. Patrick⁴
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Association of the Friesinger Score and Estimated Glomerular Filtration Rate in an Urban South Asian Patient Population

Nadkarni Girish^a, Javed Fahad^a, Khan Shahzeb A^b, Annapureddy Narender^a, Benjo Alexandre^a, Aziz Emad F^a, Herzog Eyal^a. St. Luke's-Roosevelt Hospital Center University Hospital of Columbia University College of Physician and Surgeons, New York, NY, USA^a, Department of Medicine/Pediatrics, Wayne State University, Detroit, MI, USA^b

Prevalence of Iron Deficiency in Chronic Kidney Disease. Analysis in a Single Outpatient Nephrology Setting

Ebima Okundaye, Jacob Paulose, Sarfaraz Nimra , Chaim Charytan, Bruce S. Spinowitz; New York Hospital Queens, Flushing, NY and Nephrology Associates PC, Bronx, NY

Chronic Kidney Disease Progression to ESRD: Smooth and Progressive Vs Uneven and Staccato Patterns? – A Mayo Clinic PBRN-Based 82-Month Analysis of 100 High-Risk CKD Patients – Implications for a Paradigm Change in Reno-Protection

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To Stent or Not To Stent: Renal Artery Stenosis – A Mayo Health System Hypertension Clinic 82-Month PBRN-Based Patient-Level Prospective Data Analysis of 26 High- Risk CKD Patients with Renal Artery Stenosis Presenting with Acute Kidney Injury

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Phase 2b/3 Trial Design and Baseline Patient Characteristics of a Study to Determine Effects of Bardoxolone Methyl (BARD) in Patients with Type 2 Diabetes (T2DM) and Chronic Kidney Disease (CKD)

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Factors Associated with Quality of Life in African Americans with CKD

Anna Porter,¹ Michael Fischer,¹Deborah Brooks, Marino Bruce, Jeanne Charleston, William Cleveland, Tonya Corbin, Donna Dowie, Marquetta Faulkner, Jennifer Gassman, Tom Greene, Leena Hiremath, Cindy Kendrick, John Kusek, Denyse Thornley-Brown, Xulei Wang, Keith Norris, Mark Unruh, James Lash¹ for the AASK Study Group. ¹Medicine, U. Illinois, Chicago, IL. USA

Effect of Calcium Acetate (CaAc) on Serum Phosphorus (P) Levels in Nondialyzed Patients with Advanced Stages of Chronic Kidney Disease (ND-CKD)

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Prevalence and Significance of Unrecognized Myocardial Infarctions in Chronic Kidney Disease

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Calciophylaxis Case Study with Response to Sodium Thiosulfate Treatment

Megan Robinson, Mike Francis, Hanna Mawad, University of Kentucky Medical Center, Lexington, Kentucky, USA

The Association of Serum Phosphorus and Pulse Pressure in Men and Women with Chronic Kidney Disease: Data from the Kidney Early Evaluation Program

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Comparison of Awareness of Chronic Kidney Disease Guidelines among Primary Care Physicians & Resident Physicians: A National Survey

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Outcomes in Patients with and Without Proteinuria in a Single Center HIV Cohort

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Aspects of IV Ferumoxytol Administration and Acute Adverse Events in CKD Patients

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Under-Measurement of Quantative Urinary Protein in Stage 3 Kidney Disease

Shayan Shirazian, Jai Radhakrishnan, Maya Rao, Herbert Chase. Columbia University Medical Center, New York, NY, USA

Paricalcitol Treatment in CKD Patients with Secondary Hyperparathyroidism is Associated With Better Health Outcomes When Compared with no Vitamin D Receptor [VDR] Activator Treatment

Raimund Sterz¹, Carla Frye², Samina Khan¹, Qing Harshaw², Paul Audhya¹, Katie Deering², Steven Marx¹

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Effect of Niacin on Phosphate Control in Chronic Kidney Disease

Ketki Tendulkar, Elizabeth R. Lyden, John Berger, Marius C. Florescu
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CKD-EPI Equation is more Accurate than MDRD-Equation in a Multi-Ethnic Asian Population

Boon Wee Teo, Hui Xu, Borys Shuter, Danhua Wang, Jialiang Li, Arvind Kumar Sinha, Pek Yee Chow, Sunil Sethi, Evan Lee; National University Health System, Singapore

Ethnic Coefficients are not Required for the MDRD-Equation in a Multi-Ethnic Asian Population

Boon Wee Teo, Hui Xu, Borys Shuter, Danhua Wang, Jialiang Li, Arvind Kumar Sinha, Sunil Sethi, Evan Lee; National University Health System, Singapore

Early and Late Native Kidney Biopsies in Orthotopic Liver-Alone Transplant (OLT) Recipients

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Automated Reporting of Estimated GFR Alters Referral Patterns to a Nephrology Clinic

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CKD and Older Adults: A Review and Implications for Social Work Practice and Research

Tiffany Washington, University of North Carolina, Chapel Hill, USA

Effects of Pulsed Electromagnetic Field Therapy (PEMF) on Reducing Proteinuria (P) in CKD

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Bardoxolone Methyl (BARD) Inhibits Inflammatory Signaling in Cultured Mesangial Cells

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Chronic Kidney Disease Awareness is Limited in Patients Seen by Nephrologists

Julie Wright, T. Alp Ikizler, Kerri Cavanaugh, Vanderbilt University, Nashville Tennessee, U.S.A.

An Ethnic Chinese Coefficient for the Re-Expressed MDRD Equation Using Standardized Creatinine

Hui Xu, Boon Wee Teo, Borys Shuter, Danhua Wang, Jialiang Li, Arvind Kumar Sinha, Sunil Sethi, Evan Lee; National University Health System, Singapore

Standardized Serum Creatinine with Cystatin C Improves Accuracy of GFR Estimates in Asians

Hui Xu, Boon Wee Teo, Danhua Wang, Jialiang Li, Sunil Sethi, Evan Lee; National University Health System, Singapore

Three Years Experiences of Japanese Version of Kidney Early Evaluation Program (KEEP JAPAN)

Mitsuru Yanai, Kazuyoshi Okada, Susumu Takahashi
International Kidney Evaluation Association Japan, Tokyo, Japan

Diabetic Nephropathy

Role of Subclinical Inflammation in Early Diabetic Nephropathy

Gaurav Agarwal, Nirmal Kumar, S K Agarwal
John H Stroger Hospital of Cook County Chicago IL USA,

Factors in the Progression of Diabetic Nephropathy and its Complications: A Single Center Experience in Saudi Arabia

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Novel Antioxidants Block High Glucose-Induced Tubular Hypertrophy in Proximal Tubule Epithelial Cells

Terry Brown-Bryan, Susanne Nicholas, Keith Norris

Charles Drew University of Medicine & Science, and David Geffen School of Medicine, UCLA, Los Angeles, CA

In-Vivo Analysis of Glomerular and Proximal Tubule Handling of Age-Modified Albumin Using Two-Photon Microscopy

Monika Gandhi, Mark Wagner, Bruce Molitoris, Ruben Sandoval, George Rhodes. Division of Nephrology, Indiana University, Indianapolis, IN, USA.

Decreased Podocyte-Specific Protein Expression Correlates with Severity of Diabetic Nephropathy

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Urinary Excretion of Podocytes as an Early Marker of Diabetic Nephropathy

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Impact of Impaired eGFR On Hemoglobin A1C and Average Glucose Correlation in Diabetes Mellitus

Salman Waheed, Mark E. Williams, Renal Unit, Joslin Diabetes Center, Boston, MA, USA

Electrolyte and Acid Base

Serum Magnesium Concentration after Magnesium Citrate Bowel Preparation

Nisha Bhatt, George Bayliss, M. Rachel Sim, Suzanne Martin, Jacqueline Wolf, Roger Davis, Robert A. Cohen

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A Case of Peripartum Hypercalcemia: A Role for Prolactin?

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Hypercalcemia of Malignancy from Squamous Cell Carcinoma of Lung - A Therapeutic Challenge

Sajid Melvin George, Richard Hall, Kambiz Kalantarinia. University Of Virginia Health System, Charlottesville, VA

Diagnostic Value of Urine Sodium Concentration in Hyponatremia Due to Syndrome of Inappropriate Antidiuresis (SIAD) Versus Hypovolemia
Takashi Hato¹, Richard Hellman¹, Roland Ng². ¹Division of Nephrology, Indiana University, Indianapolis, IN, ²Division of Nephrology, University of Hawaii, Honolulu, HI

Bridging the Gap: The use of Spaced Education to Teach Medical Students Principles of Acid-Base and Fluid-Electrolyte Disorders
Michael Kern, Terry Wolpaw, Mahboob Rahman, Case Western Reserve University, Cleveland OH

Vitamin D Mediated Hypercalcemia & Recurrent Kidney Injury
Vijay Lapsia, Muna Canales, Univ of Florida at Gainesville, USA.

Elevated PTH and PTH-rp In a Patient with Hypercalcemia Of Malignancy
Laura Maursetter, Cate Ranheim, Micah Chan. University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin. USA

Hypernatremia in a Patient Treated with Sodium Polystyrene Sulfonate
Nepal M, Bucaloiu ID, Carnero G, Norfolk ER, Geisinger Medical Center, Danville, PA

Unique Etiology of Hypercalcemia in Male to Female Transgender Patients
Onyema Ogbuagu, Peter Soltani, Kayode Lawrence, Aaron Stern. Mount Sinai School of Medicine (Elmhurst), Elmhurst, NY, USA

An Unusual Case of Severe Hypokalemia Presenting with Hemiplegia
Okafor, Chidi, Lobo Peter; University of Virginia Health System, Charlottesville, VA, USA.

Bulimia as a Cause of Metabolic Alkalosis in a Patient with Chronic Kidney Disease
Praneetha Puskuri, Zaher Hamadeh, Anjali Acharya
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Fatal Hypercalcemia from Treating Hyperkalemia
Kanwardeep Sachdeva, Kantima Phisitkul, Amber Podoll, Kevin Finkel,
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Hypercalcemia Due to Granulomatous Activity of Pneumocystis Jiroveci Pneumonia in a Renal Transplant Patient
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Hypercalcemia Associated with Gluteal Silicone Injections: A Case Report

Salman Singapuri, Daniel Patel, Frederic Rahbari-Oskoui

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Hyponatremia and Steroid Resistance in HIV Disease-

Jiwan Thapa, Satinder Singh, Apurv Khanna, SUNY Upstate, Syracuse, NY, USA

Prevalence of Metabolic Acidosis and the Distribution between Anion Gap and Non-Anion Gap Acidoses

Sandeep Tiyyagura, Thinh Nguyen, Beth Stefanchik, Andrew Bohmart, and Godfrey Burns. St. Vincent's Catholic Medical Center, New York, NY and New York Medical College, Valhalla, NY

Hyponatremic Patients Demonstrate Improved Cognition after an Increase in Serum Sodium

Rick P. Vaghasiya, Maria V. DeVita, Georgia Panagopoulos, Michael F. Michelis, Division of Nephrology, Department of Medicine, Lenox Hill Hospital, New York, NY, USA

Severe Electrolyte Abnormalities after Cetuximab- Management Challenge

Rajiv Vij and Mala Sachdeva, Division of Nephrology, North Shore/Long Island Jewish Health System, NY USA

Management of Gitelman Syndrome in Pregnancy

Di Zhao and Matthew Trainor, Division of Renal Medicine, University of Massachusetts School of Medicine, Worcester, MA, USA

Glomerular Diseases

Small B Cell Lymphoma Manifesting Solely as Nephrotic Range Proteinuria. A Case Report

Waqas Ahmed, Naseer Khan, Madan Gowda, Muhammad A Khan, Timothy Braverman. Jewish Hospital Cincinnati, OH

Membranoproliferative Glomerulonephritis (MPGN) Secondary to Type III Cryoglobulinemia Associated with Small Lymphocytic Lymphoma Treated with Rituximab

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A Rare Case of Interstitial Nephritis with Secondary Membranous Glomerulopathy Due to Glimepiride

Sana Akbar, Melvin Goldblat, Sadanand Palekar, Sachin Sachdev, Adeem Akbar. Newark Beth Israel Medical Center, Newark, NJ

Anti-C1Q Antibody Levels are not Reliable Forecasters or Markers and Lupus Nephritis Flare

Joshua E. Bitter, Brad H. Rovin, Lee A. Hebert, Daniel J. Birmingham. Ohio State University, Columbus, OH USA

Chronic Bronchiectasis and Perinuclear-ANCA (pANCA) Crescentic Glomerulonephritis

William Chen, Andrew Chin. UC Davis Medical Center, Sacramento, CA, USA.

A Case of Podocyturia in a Patient Receiving Anti-VEGF Therapy with Sunitinib

Iasmina Craici, Steven Wagner, Aminah Jatoi, Joseph Grande, Eddie L. Greene, Vesna Garovic Mayo Clinic, Rochester, MN, USA

Coexistent P-ANCA and Anti-GBM Antibody Glomerular Disease Associated with Endometrial Cancer

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Idiopathic Nodular Glomerulosclerosis in a Non-Diabetic Young Caucasian Male with Metabolic Syndrome: A Case Report

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Cyclosporine for Treatment Membranoproliferative Glomerulonephritis (MPGN) Associated With Chronic Lymphocytic Leukemia (CLL)

Sajid Melvin George, Helen Cathro, Mitchell Rosner. University of Virginia Health System, Charlottesville, VA

Minimal Change Disease (MCD) in Patient of Ulcerative Colitis (UC), Adverse Reaction to Balsalazide or Disease Association? Literature Review & Case Report

Manish Gera¹, R.Jeevan¹, S.Dhar¹, P.S.Gera², C.L.Phillips³. Internal Med. Nephrology. Terre Haute, IN¹. Union Hospital Family Med. Residency. Terre Haute, IN². I.U.Pathology, Indianapolis, IN³

Membranous Nephropathy with Myeloperoxidase (MPO) Antineutrophil Cytoplasmic Antibody (ANCA) Associated Necrotizing and Crescentic Glomerulonephritis (NCGN)

Saraswathi Gopal, Alan Dubrow, Lawrence Kiss, James Winchester, Beth Israel Medical Center, New York, NY, USA

A Unique Case of Collapsing Glomerulopathy in a Patient with Mixed Connective Tissue Disease

Hemalatha Gutta, Stephen I Rifkin, Reji Nair, Christopher McFarren, Donald E Wheeler, Univ .of South Florida, Tampa, FL, USA

IGA Nephropathy in Cirrhosis; Incidence, Pathogenesis, and Clinical Significance: A Literature Review

Hilana Hatoum¹, Fadi Rzouq². ¹: McLaren Regional Medical Center, Flint, MI. ²: University of Washington, Seattle, WA

Uncommon Etiology of Cryoglobulinemic Renal Vasculitis

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Cryoglobulinemic MPGN in a Patient with Scleroderma and Sjogren's Syndrome

Ruth Indahyung, Mohamed Shafiu, Mark Segal
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Monoclonal Gammopathy of Uncertain Significance (MGUS) and Focal Segmental Glomerulosclerosis (FSGS)-A Case Report

Nishank Jain, Mary Rose Tantoco, Beje Thomas, Jeffrey Laut, University of Connecticut, Farmington, Connecticut, USA

MGN with ANCA-Associated RPGN: A Rare Dual Glomerulopathy

Zeeshan Khawaja, Pouneh Nouri. Georgetown University Hospital, Washington, DC

P-ANCA Associated Crescentic Glomerulonephritis in Setting of Renal Cell Carcinoma

Biresh Kumar, Sheldon Chaffer. Department of Nephrology, Scott & White Hospital, Texas A&M Health Science Center, Temple, TX, USA.

Membranous Nephropathy: A Complication of IgG4-Related Systemic Disease

Kelly Liang, Nidhi Jindal, Christopher Passero, Alyssa Krasinskas, Fiona Craig, Sheldon Bastacky, Dhiraj Yadav University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Renal Prognosis in HIV HCV Co-Infected Patients in the HAART Era

Sumit Mohan, Jennifer Tan, Jai Radhakrishnan, Jen-Tse Cheng. Dept of Medicine, Div of Nephrology, Harlem Hospital and Department of Medicine, Div of Nephrology Columbia University College of Physicians and Surgeons

Three Decades of Progress in Treating Childhood Onset Lupus Nephritis
Tanya Pereira, Carolyn L. Abitbol, Wacharee Seeherunvong, Jayanthi Chandar,
Michael Freundlich, Gaston Zilleruelo
University of Miami/Holtz Children's, Miami, FL

**"Will the Real Diagnosis Please Line Up"-A Rare Case of Anti-GBM
Negative Goodpasture's Syndrome**
Karthik Ramani, Pritesh Patel, Anderson Penuela, LSU Health Sciences Center,
Shreveport, LA

**A Case Report of HIV Associated Immune Complex Glomerulonephritis in
an African-American Male**
Margaret Rose, Roy Zent .Vanderbilt University Hospital, Nashville, TN USA

**Clinical Experience in the Therapeutic Challenge of Collapsing Focal
Segmental Glomerulosclerosis (FSGS)**
Andres Serrano, Lakshmi Nadimpalli, Mt. Sinai Hospital, Chicago, IL, USA

**Immune complex Glomerulonephritis with Lupus Like Features in a HIV
Positive Male**
Mohit Turagam, Suneetha Vysetti, Jean Holley. University of Illinois, Urbana, IL,
USA

Endocarditis and Kidney Mass in Wegener's Granulomatosis
Sreelatha Varkala, Carol Yuan, Luis Beltran Garcia, Nicholas Shah, Selvi
Gunasekharan, Elias Doumit. University of South Florida, Tampa, Florida, USA

First-Trimester Pregnancy Loss Associated with Plasma Exchange Therapy
Steven Wagner, Iasmina Craici, Carl Rose, Joseph Grande, Vesna Garovic
Mayo Clinic, Rochester, MN, USA

**IGA-Dominant Staphylococcus Infection- Associated Glomerulonephritis:
Two Case Reports and Review of the Literature**
Edgard Wehbe, Sankar D Navaneethan, Charbel A. Salem, James Simon, Marc A.
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OH, USA

Hemodialysis Anemia

**Epoetin Alpha Administration was Reduced to Once per Week without
Compromising Hemoglobin Targets**
Alexandre Ackad, Christopher Parisi. Hackensack University Medical Center,
Hackensack, NJ

Variant Hemoglobin May Affect Erythropoietin Response in African-Americans Receiving Hemodialysis

Vimal K. Derebail, Patrick H. Nachman, Nigel S. Key, Heather Ansede, Ronald J. Falk, Abhijit V. Kshirsagar

Parenteral Iron Therapy and Infection Related Hospitalizations in ESRD Patients on Hemodialysis

N Goel, R Khanna, M Awad, B Kanna, I Gnanasekaran, Lincoln Medical and Mental Health Center, Bronx, New York, USA

Computer-Based Model for Evaluation and Validation of Current Anemia Management Protocols in Hemodialysis

Emily Kenner, Zack Ernstberger, Michael Brier, Adam Gaweda, Louisville, Kentucky, USA

Administration of Ergocalciferol: Effect on Anemia Management in End Stage Renal Disease

Neenoo Khosla, Derek Larson, Junine DeGraf, Hongyan Du, Stacey Kirshenbaum, Stuart M. Sprague, Louisa Tammy Ho, University of Chicago (NorthShore), Evanston, IL, USA

Effects of Erythropoietin Stimulating Agent (ESA) Automated Adjustment Protocols on Hemoglobin (HGB) Levels and Mortality in ESRD Patients

Luiz M. Kolankiewicz¹, Michal L. Melamed¹, Marcos Rothstein², Marc S Weinberg³. ¹ Albert Einstein College of Medicine, Bronx, NY, ² DSI Renal, Inc. Nashville, TN and Washington University Medical School, St. Louis, Missouri and ³ Roger Williams Medical Center, Boston University School of Medicine, Providence, RI

Relationship between Statins Use and Erythropoietin Resistance in ESRD Patients

Salman R. Mallick, Codruta Rafiroiu, Ghayyath Sultan, Charity Kankam, St Vincent Charity Hospital Internal Medicine Residency Program, Cleveland, OH, USA

Ferumoxyl in Endstage Renal Disease: Repletion and Maintenance Dosing

Beckie Michael, Istvan Bogner, Joji Urlanda, Johnie Flotte, Charlene Garrison, Marcos Rothstein, DSI Renal, Nashville, TN

Vitamin D Supplementation to Improve Levels of Anemia in ESRD Patients

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Long-Term Clinical Experience with Hematide™

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²AFX01-03/09 Hematide Study Groups; ³Affymax, Inc., Palo Alto, CA, USA

Dose Conversion Analyses between Epoetin Alfa (EPO) and Darbepeotin Alfa in Hospital-Based Dialysis Centers (HBDCs)

Amit Sharma, MD;¹ Jerry Yee, MD;² Shravanthi R Gandra, PhD, MBA;³ Irfan Khan, PhD;³ Jeffrey Petersen, MD³

¹Boise Kidney and Hypertension Institute, Meridian, ID; ²Henry Ford Hospital, Detroit, MI; ³Amgen Inc., Thousand Oaks, CA

Evaluation of the Maintenance of Hb Control in HD Patients both During and After Hospitalization with Once-Monthly Hematide™

Raja Zabaneh^{1,2}; A Besarab²; V De Silva²; S Zeig²; E Martin²; L Ardelean²; P Pergola²; F Whittier²; A Covic²; A Wiecek²; B Schiller²; M Kaplan²; R Leong³; A M Duliege³; Gao H³; I C Macdougall²

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Hemodialysis: Mineral Metabolism

A Prospective Randomized Trial on Intermittent Post-Dialysis Dosing Of Cinacalcet: A Solution to Non-Compliance

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Post-Parathyroidectomy (PTX) Thyrotoxicosis in a Patient with ESRD. A Case for Caution

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Impact of Sevelamer on Hospitalization and In-Center Dialysis Utilization: A Modeled Study Based On DCOR

Lisa Bernard³, David Mendelssohn¹, Elizabeth Dunn², , Parisa Airia³, Daniel Grima³. ¹Humber River Regional Hospital, Weston, ON, Canada, ²Genzyme Corporation, Cambridge, MA, USA, ³Cornerstone Research Group, Burlington, ON, Canada

Tumoral Soft Tissue Calcification in a Dialysis Patient

Bucaloiu I D, Ashouian N, Bonebrake S R, Bowen T R, Hartle J E, Norfolk E R. Geisinger Medical Center, Danville, PA

Efficacy of Incorporating Criteria for Cinacalcet (Sensipar) Use Into a Paricalcitol (Zemlar) Protocol in Secondary Hyperparathyroidism

Barbara Clark, Bruno Lima, Piyush Lohiya, Dept of Nephrology, Allegheny General Hospital, Pittsburgh, PA, USA

Strategies to Improve Phosphorus Control in Patients with Chronic Severe Hyperphosphatemia

Barbara Clark, Ankur Patel, Lori Groves-Seaman, Jessica Palombine, and Richard Marcus. Nephrology and Hypertension, Allegheny General Hospital, Pittsburgh, PA, USA

Effect of Ergocalciferol Treatment on Mineral Metabolism in Chronic Hemodialysis Patients

Junine DeGraf, Derek Larson, Hongyan Du, Stacey Kirshenbaum, Stuart M. Sprague, Neenoo Khosla, Louisa Tammy Ho

Optimum Frequency of Parathyroid Hormone Monitoring in Chronic Dialysis Patients

Suneeta Gadde, Sheldon Greenberg, Murali Pagala, Miriam Greenberg, Ilya Shneyderman, KC Janga, Maimonides Medical Center, Brooklyn, NY

Bioequivalence of a Liquid vs. Solid Formulation of Calcium Acetate

Jonathan Greenberg, Laura Howson, Chiang Hong-Ho, Claudy Mullon, Claude Miller, Sarah Tuller, Anila Mico, Steve Houlihan, Vitaly Pirotsky, Jose Diaz-Buxo. Fresenius Medical Care NA, Waltham, MA, USA

Improved Survival among Paricalcitol Treated Hemodialysis Patients Compared with no Treatment for Secondary Hyperparathyroidism [SHPT] Is Independent of Baseline iPTH Levels

Samina Khan¹, Steve Marx¹, Allen Nissenson², Beverly Johns¹, Paul Audhya¹.
¹Abbott, Abbott Park, IL, USA, ²DaVita Inc., Lakewood, CO

Paricalcitol Treated Hemodialysis Patients Utilize Less Erythropoietin than Patients Not Receiving Treatment for Secondary Hyperparathyroidism [SHPT]

Samina Khan¹, Steve Marx¹, Allen Nissenson², Beverly Johns¹, Paul Audhya¹.
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Parathyroidectomy Trends in US Hemodialysis Patients, 1992-2007

Suying Li¹, Yen-Wen Chen², Yi Peng¹, Robert N. Foley¹, Wendy St. Peter^{1,2}.
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In-Center Nocturnal Hemodialysis Leads to Improved Serum Phosphorus (PO₄) Levels

Robert I. Lynn¹, Linda Francisco², Shane Simon², Abbe Volz², Karen Spach², Ronald Levine², Robert Provenzano²

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Dialysis Health Outcomes Associated with Pre-Dialysis use of Paricalcitol Compared with no Vitamin D Receptor (VDR) Activator Treatment

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Amount of Fluid Ingested with Phosphate Binders (PB) in Hemodialysis-Dependent Chronic Kidney Disease (HDD-CKD) Patients

Claudy Mullon, Elizabeth Sussman, Nancy Ginsberg, *Rosio Ramos, *Maria Tarallo, *Rebecca Apruzzese, *Olga Sergeyeva, Chiang-Hong Ho, Jose Diaz-Buxo. Fresenius Medical Care North America, Waltham, MA, USA. *Renal Research Institute, New York City, NY, USA

Reactive Thyrotoxicosis: Under Diagnosed Complication of Parathyroidectomy in the Dialysis Population

TH Naber, KD Jhaveri, M Sachdeva.

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Proton Pump Inhibitors and Efficacy of Phosphate Binders in Control of Serum Phosphorus Levels in Hemodialysis Patients

Okundaye, Ebima, Paulose, Jacob, Lockman, Carl, Spinowitz, Bruce, Charytan, Chaim

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Generalized Osteitis Fibrosa Mimicking Metastatic Bone Disease: A Case Report

Oluwaseun Opelami, Tinatin Narsia, Andrei Brateanu.

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The Effect of Sevelamer Carbonate and Lanthanum Carbonate on the Pharmacokinetics of Oral Calcitriol

David Pierce¹, Raymond Pratt², Patrick Martin², Lynne Poole¹. ¹Shire Pharmaceuticals, Basingstoke, UK. ²Shire Pharmaceuticals, Wayne, PA

Reduction of Dietary Phosphorus Absorption with Lanthanum Carbonate or Sevelamer Carbonate: A Balance Study

Raymond Pratt¹, Patrick Martin¹, Lynne Poole², Jeffrey Dragone¹. ¹Shire Pharmaceuticals, Wayne, PA; ²Shire Pharmaceuticals, Basingstoke, UK

Association of 25 Hydroxy Vitamin D with Health Related Quality Of Life and Physical Function in a Hemodialysis Population

Wilner Samson, Farmington, CT; Sharad Sathyan, Farmington, CT; Richard Feinn, Farmington, CT; Anne Kenny, Farmington, CT

Iron Absorption with Higher Doses of Ferric Citrate in Controlling Serum Phosphorus in ESRD Patients

Marvin Sinsakul¹, Steven Korbet¹, Tom Greene², Mohammed Sika³, Julia Lewis³ and the Collaborative Study Group ¹Rush University, Chicago, IL, ²University of Utah, Salt Lake City, UT, ³Vanderbilt University, Nashville, TN

The Safety and Tolerability of Higher Doses of Ferric Citrate (FC) In Controlling Serum Phosphorus (P) In ESRD Patients

Marvin Sinsakul¹, Steven Korbet¹, Tom Greene², Mohammed Sika³, Julia Lewis³ and the Collaborative Study Group ¹Rush University, Chicago, IL, ²University of Utah, Salt Lake City, UT, ³Vanderbilt University, Nashville, TN

Lanthanum Carbonate Offers Sustained Control of Serum Phosphorus

Michael Smyth, Lynne Poole. Shire Pharmaceuticals, Basingstoke, UK

Use of Kinetic Modeling to Achieve K/DOQI Phosphorus Target and Neutral Calcium Balance in HD Patients

Amanda K. Stennett¹, Norma J. Ofsthun¹, Peter Kotanko^{2,3}, Nancy Ginsberg², Jeff Maxwell¹, Frank A. Gotch¹ ¹Fresenius Medical Care, Waltham, MA ²Renal Research Institute New York, NY, ³Beth Israel Medical Center New York, NY

Calcific Uremic Arteriopathy in a Patient on Hemodialysis: Role of Pharmacotherapy

Chandraprakash Umapathy and Kelly Liang, Renal-Electrolyte Division, University of Pittsburgh, Pittsburgh, PA, USA

Rapid Reduction of Serum Phosphorus Levels by Lanthanum Carbonate in Patients on Dialysis

Rosamund Wilson,¹ Scharmen Confer,² Raymond Pratt.² ¹SPICA Consultants Ltd, Marlborough, UK; ²Shire Pharmaceuticals, Wayne, PA, USA

Hemodialysis: Other

How do Patients with Failed Kidney Transplants Fare once back on Dialysis? A Gulf Prespective

Samra Abouchacra¹, Ahmed Chaaban¹, Abdelkarim Saleh², Muna Rukhaimi³, Osman Furaih⁴, Naveed Haq¹, Suad Sajwani¹, Mohamad Osman¹, Nicole Gebran¹, ¹Tawam Hospital, UAE; ²SKMC, UAE; ³Dubai Hospital, UAE; ⁴KFSH RC, Saudi Arabia

Prevalence of Pulmonary Hypertension in Patients with High Fistula Flow Rates

R Agarwala, D Da-Rocha, S Mohan, H Anderson, A Clarke, I Fergus, V Pogue, JT Cheng .Harlem Hospital, Columbia University, NY

Serum Beta 2 Microglobulin Levels in Patients on Chronic Hemodialysis

Mohit Ahuja, Anita Basu, Geeta Gyamlani, Monique Moxey Department of Nephrology, G.V. Sonny Montgomery Veteran's Affairs Medical Center, Jackson, MS, USA

Regional Citrate Anticoagulation for Slow Continuous Ultrafiltration (SCUF) Complicated By Severe Metabolic Alkosis

Mourad Alsabbagh, Ahsan Ejaz, Edward A. Ross, Division of Nephrology and Hypertension, University of Florida at Gainesville, Gainesville, FL

Validation of GDS-15 as a Screening Tool for Depression in Elderly Hemodialysis Patients

Rasheed A Balogun¹, Faruk Turgut¹, Seki A Balogun², Suzanne Holroyd³ and Emaad M Abdel-Rahman¹

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Five-Year Follow up to the Hemodialysis Infection Prevention with Polysporin Ointment (HIPPO) Study

Marisa Battistella^{1,2}, Jayvee Guerrero¹, Cynthia Bohla¹, Charmaine E. Lok^{1,2}

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Reducing Behavior-Based Missed Hemodialysis Treatments

Stephanie Best, Bart Canny, Emily Averette, David Cameron, David Keaveney, Janel Anderson, Gemini Stroman, Jennifer Felts, David Lapinski, Helen Grammas, Hollie Russ
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Vitamin D [25(OH)D] and its Relationship to Risk Factors For Cardiovascular Disease in Maintenance Hemodialysis Patients

Debra Blair^{1,2}, Laura Byham-Gray¹, Stephen Sweet³, Emily Lewis², Susan McCaffrey², Scott Parrott¹, Alison Steiber⁴, Riva Touger-Decker¹, Diane Rigassio Radler¹. ¹University of Medicine and Dentistry of New Jersey, Newark, NJ; ²Fresenius Medical Care (FMC), Springfield, MA; ³Western New England Renal and Transplant Assoc., Springfield, MA; ⁴Case Western Reserve University, Cleveland, OH.

**Helping Adult Hemodialysis Patients Self-Manage Oral Medications:
Recommendations from the Literature**

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Limited Health Literacy Associated With Catheter Use for Chronic Hemodialysis

Kerri L. Cavanaugh, Russell L. Rothman, Julie A. Wright, T. Alp Ikizler.
Vanderbilt University Medical Center, Nashville, TN

Improving the Dialysis Experience with the Use of Technology

Crampton, Karen; University of Michigan Dialysis Services, Ann Arbor, MI

Cost Assessment of Patients with Excessive Hospital Stays in an Inner City Hospital

Egbosimba, Florence C. Rutgers University, NJ, Hannah Nelson, Praneetha Puskuri, Anjali Acharya, Jacobi Medical Center, Bronx, NY, USA

Conception of a National Registry for Renal Replacement Therapy (RRT) In Lebanon

Hafez Elzein, Salim Kabalan, Samir Mallat, Hilal Abuzeinab, Beirut, Lebanon

Pilot Study of Practices and Outcomes at Hemodialysis Centers in Lebanon

Hafez Elzein, sana ghaddar,ahmad abdallah. Beirut, Lebanon

Health Disparities/Inequities in End Stage Renal Disease

Ford-Anderson, Carla, Nephro-Care West, Inc., Brooklyn, NY

Adequacy Of Intermittent Hemodialysis (IHD) in an Inpatient Setting

Meghana R. Gaiki, Maria V. DeVita, Jordan L. Rosenstock,
Georgia Panagopoulos, Michael F. Michelis, Division of Nephrology, Department of Medicine, Lenox Hill Hospital , New York, NY, USA

Tenecteplase for the Improvement of Blood Flow Rate in Dysfunctional Hemodialysis Catheters

Jesse Goldman,¹ Steven Fishbane,² Matthew J. Oliver,³ Martha Blaney,⁴ Joan R. Jacobs,⁴ Susan M. Begelman⁴

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⁴Genentech, Inc., South San Francisco, CA, USA

Increased Oxidative Susceptibility of Low Density Lipoprotein (LDL) after Standard Hemodialysis Procedure

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Incidence of Vascular Access Type after Renal Allograft Failure

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Staphylococcus Lugdunensis Mitral Valve Endocarditis in a Patient on Chronic Hemodialysis

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Hemoperfusion (HP) vs Hemodialysis (HD) In a Case of Combined Overdose (OD) With Acetaminophen and Valproic Acid (VPA): Implications of Our Findings

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Improving Adherence in Adolescents with ESRD: A Case Study

Robin Hensley, Kathy Jabs; Vanderbilt University Medical Center, Nashville, Tennessee, USA

Extra-Articular Manifestations of Dialysis Related Amyloidosis: A Case Report

Nabeel Imam, George Muteema, Anjum Najeed, Mohammad Ahmed, Antoine Samaha, Good Samaritan Hospital, Cincinnati, OH, USA

Timing of Hemodialysis Vascular Access Placement Determines Access Outcomes

Kambiz Kalantarinia, Adam Campbell, Shadi Mourad, Judy Kauffman, Kim Deaver, University of Virginia Health System, Division of Nephrology, Charlottesville, VA

Evaluation of Medication Reconciliation Process in Preventing Medication Errors in ESRD Patients

Swapna Kamadana, Ravish Shah, Judy Hartman, Sheri S. Vancleef, Christopher Valentine. Division of Nephrology, The Ohio State Medical Center, Columbus, OH, USA

Adequate Estimation of Tumor Markers in Hemodialysis (HD) Patients

Miho Kando, Nozomi Okada, Daisuke Okita, Emi Kihara, Koichi Sasaki, Yasufumi Kiyota, Chiharu Kawamoto, Yuka Orita, Kazuko Arita, Katsutoshi Maeda, Hiroaki Oda Oda Medical Clinic, Hiroshima, Japan.

Analysis of Hemoglobin Stability with Hematide™ in Hemodialysis Patients

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Managing Workload on Dialysis

Sonja Kapun, Dialysis Center Nefrodial Krsko, Slovenia

Intradialytic Use of Heparin: Is Heparin Necessary for Stable Chronic Hemodialysis Treatments?

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Comparison of Mortality Risk between Incenter Nocturnal and Conventional Hemodialysis

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Depression is Associated with Higher Mortality Risk in Incident Hemodialysis Patients

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Incenter Nocturnal Hemodialysis Conversion-Associated Changes in Laboratory Markers

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Evaluating Deficiencies in Processes of Care in Incident Fistulas in a Veterans Administration Healthcare System

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Effect of Hemodialysis (HD) on Body Composition in ESRD Patients (PTS)

Neal Mittman, Meghna Desai, Rakesh Sheliya, Finian Oparah, Jyoti Chattopadhyay, and Morrell M. Avram. Avram Division of Nephrology, Long Island College Hospital, Brooklyn, New York, USA

Long-Term use of Ferric Citrate in ESRD Patients

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Citrate (ACD) Vs Heparin Locks: Comparing Catheter Outcomes for Short Term Inpatient Hemodialysis

Ruba Nijmeh, Samir Parikh, Udayan Bhatt, Anil Agarwal. The Ohio State University, Columbus, OH

Improving Medication Access to Reduce Disparities by a Large Dialysis Provider

Allen R. Nissenson¹, Josh Golomb¹, Joe Weldon¹, Steve Wilson¹
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Dialysis Related Transient Methemoglobinemia-A Case of Topical Prilocaine Induced Systemic Toxicity

Ebima Okundaye, Alla Goldberg, New York Hospital Queens, Flushing and Trude Weishaupt dialysis Satellite Unit, Flushing, NY

Bilateral Lower Extremity Sequential Compression Devices (SCDS) for the Management of Intra-Dialytic Hypotension – A New Approach to an Old Problem

Macaulay Onuigbo, Mayo Clinic, Rochester, MN, USA & Midelfort Clinic, Eau Claire, WI, USA

Time Averaged Variations in Hemodialysis Vascular Access Types and Their Determinants at an Inner-City Hemodialysis Facility

Anju A. Oommen, Khalid Bashir, Chamberlain Obialo, Khalid Mehmood, Christopher Phillips, Ernest Alema-Mensah, Morehouse School of Medicine, Atlanta, GA, USA

Impact of a Nurse-Driven Vascular Access Management Program on Achieving and Maintaining Optimal Vascular Access for a Chronic Hemodialysis (HD) Population

Abeth Overbey and Karen Bell, University of Colorado Hospital, Aurora, Colorado, USA

U.S. Investigations of Healthcare-Related Adverse Events in Hemodialysis Patients, 1999-2009

Priti R. Patel, Melissa K. Schaefer, Nicola D. Thompson, Matthew J. Arduino, Centers for Disease Control and Prevention (CDC), Atlanta, GA

The DOPPS Practice Monitor: A New Initiative for Timely Reporting of US Hemodialysis Practices

RL Pisoni, DS Fuller, JM Albert, DM Dickinson, BW Gillespie, RA Wolfe, FK Port, and BM Robinson, Arbor Research Collaborative for Health, Ann Arbor, MI USA

A Randomized Controlled Trial To Evaluate The Effects Of Cinacalcet Plus Low-Dose Vitamin D On Vascular Calcification In Hemodialysis Patients

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Effect of Medicaid Coverage in Dialysis

Simi Shahabdeen, Prasanna Srinagesh, Nauman Shaheed, Muhammed Rahman, Mohit Gupta, Ziauddin Ahmed
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K-DOQI Guideline Goal Attainment at Hemodialysis Initiation and First-Year Survival

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Streamline Bloodlines Improve KT/V While Lowering Dialysate Usage

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Replacement of 25-OH Vitamin D in an Indigent Hemodialysis Population

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Clinical Outcomes of Dialysis Catheter-Related Bacteremia (CRB) with Concurrent Exit Site Infection (ESI)

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Hematide™ Dose Adjustments in the Maintenance of Hb in Hemodialysis Patients

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Hereditary Kidney Disease

Renal Cell Carcinoma in Von-Hippel Lindau Syndrome: To Sever or to Spare

Samina Bhatti, Richlands, Virginia U.S.A., Titilayo Ilori, Chamberlain Obialo, Khalid Bashir, Abimbola Akomolafe, Atlanta, GA, USA

Comparing Mortality-Predictability of Hyper-Phosphatemia In Maintenance Hemodialysis Patients With And Without Polycystic Kidney Disease (PKD)

Lilia Lukowsky, Csaba P Kovesdy, Gabriel McNeill, Elani Streja, Jennie Jing, Mahesh Krishnan, Allen R Nissenson, and Kamyar Kalantar-Zadeh. Harold Simmons Center at Harbor-UCLA, Torrance, CA; VA Salem; DaVita Inc, El Segundo, CA

Secondary Hyperparathyroidism & Survival in Hemo-Dialysis Patients With & Without Polycystic Kidney

Gabriel McNeill, Elani Streja, Lilia Lukowsky, Csaba P Kovesdy, Jennie Jing, Mahesh Krishnan, Allen R Nissenson, Kamyar Kalantar-Zadeh. Harold Simmons Center at Harbor-UCLA, Torrance, CA; VA Salem; Salem, VA; DaVita Inc, El Segundo, CA

Home Hemodialysis

Catastrophic Hypercalcemia as a Technical Complication in Home Hemodialysis

Praveen Murlidharan, Christopher T. Chan, Joanne M. Bargman
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Hypertension

Analysis Of Renal Fibrosis And Weight Of Spontaneously Hypertensive Rats (SHRS) Pregnant Exposed To Physical Training

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Rational Synthesis of a Selective Renal Outer Medullary Potassium Channel (ROMK) Antagonist

Gautam Bhawe, Brian A. Chauder, Rishin Kadakia, Eric S. Dawson, Craig W. Lindsley, C. David Weaver and Jerod S. Denton
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Sodium (Na) Intake Varies Across the African Diaspora and is Associated With BMI

Alexander R Chang, Holly Kramer, Amy Luke, Guichan Gao, Ramon Durazo-Arvizu, Richard Cooper Loyola University Medical Center, Chicago, IL

The Association Between Sodium (Na) and Potassium (K) Intake And Systolic Blood Pressure (SBP) Across The African Diaspora

Frank Y Chen, Holly Kramer, Amy Luke, Guichan Cao, Ramon Durazo, and Richard Cooper. Loyola University Medical Center, Maywood, IL, USA

Correlation Between Routine Peri-Hemodialysis Blood Pressure Measurements and Ambulatory Blood Pressure Measurement in Patients with End-Stage Renal Disease

Adrian Cosmin., Nahid Islam, Catherine Wells, Tibor Fulop, Department of Internal Medicine, Division of Nephrology. University of Mississippi Medical Center, Jackson, MS

Effects of Aldosterone Receptor Blockers in Resistant Hypertension and Stage III Chronic Kidney Disease

Roberto Pisoni, Maria Czarina Acelajado, Suzanne Oparil, David Calhoun. University of Alabama at Birmingham, Birmingham, AL, USA

Renal Artery Stenting Works - In the Right Patient

Aastha Sethi, Ralph Daher, Sumeska Thavarajah, Mahmoud Malas, John Anderson; Johns Hopkins University, Baltimore, MD, USA

New Onset Refractory Hypertension and Hypokalemia Associated with Ectopic Renin Secretion during the First Trimester of Pregnancy

Bassam Shakil, Ammar Almekhi
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Nutrition

Validation of a Paper Based Dietary Potassium Test for Adults with Chronic Kidney Disease

Katherine Baczewski, Mary Julius, Elizabeth Kern, Louis Stokes Cleveland, Veteran Affairs Medical Center, Cleveland, OH

C-Reactive Protein (CRP) Predicts Response to Nutritional Enhancement in Hemodialysis Patients with Low Serum Albumin

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The Effect of Nutrition Counseling On Malnutrition and Azotemia in Adult Veterans with Chronic Kidney Disease (CKD)

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Assessment of Extracellular Fluid Volumes Obtained By Different Methods in Multi-Ethnic Asian Chronic Kidney Disease Patients

Pek Yee Chow, Hui Xu, Boon Wee Teo; National University Health System, Singapore

Improved Patient Phosphorus Outcomes with the Use of Lanthanum Carbonate

Rachel Gaston; DaVita Inc., Lakewood, CO, USA

Implementation of Vitamin D for Stage 3 to Stage 5 Chronic Kidney Disease Patients

Haewook Han, Robert F. Houser, Jeanne M. Wolfrum, Elizabeth Munroe and Bradley M. Denker; Harvard Vanguard Medical Associate, Department of Nephrology, Boston MA, USA

Pica: An Important and Unrecognized Problem in Pediatric Dialysis Patients

Chryso Katsoufis, Myerly Kertis, Judith McCullough, Wacharee Seeherunvong, Jayanthi Chandar, Gaston Zilleruelo, Carolyn Abitbol; University of Miami/Holtz Children's, Miami, FL

RCT of Personal Digital Assistant (PDA) Supported Dietary Intervention to Reduce Sodium Intake in PD

Michael Koprucki, Beth Piraino, Filitsa Bender, Linda Snetselaar, Beth Hall, Susan Stark, Mary A Seveck University of Pittsburgh, Pittsburgh PA, USA

Supermarket Tours as a Novel Tool for Teaching Renal Nutrition to CKD Stage 3, 4, and 5 Patients

Linda Lim; St. Paul's Hospital, Vancouver, British Columbia, Canada

Individualized Intradialytic Parenteral Nutrition (IDPN) Solutions Increase Albumin Levels in Patients with Chronic Kidney Disease (CKD) Stage Five

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¹NutrePletion Resources (NPR), also known as Home HealthCare Resources, Inc. (HHCR), Bensalem, PA, ²Accredo Health Group Inc., Memphis, TN, USA

Biotin Deficiency and Restless Legs Syndrome in Dialysis

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Effect of Protein Supplementation in Chronic Dialysis

Heidi D. Moretti, Andrea M. Johnson, Tammy J. Keeling-Hathaway, Missoula, Montana, USA

Association Of Dietary Phosphorus To Protein Ratio With Mortality In Hemodialysis Patients

Nazanin Noori, Kamyar Kalantar-Zadeh, Csaba P Kovesdy, Rachelle Bross; Debbie Benner, Joel D Kopple. Harold Simmons Center, Harbor-UCLA, Torrance; VA Salem; DaVita, El Segundo, CA

Walking Disabilities and Nutritional Status in Long-Term Hemodialysis Patients

Suzanna Rivero, Jennifer Ennis, Tammy Poma, Tiffany Perry, Nicole Stankus; University of Chicago Medical Center, Chicago, IL, USA

Beneficial Effect of Exercise on Body Composition-A Hemodialysis Case Study

Mary Sundell¹, Mary Lollar², Rita Dimmit²; ¹Vanderbilt University Medical Center, Nashville, TN, USA ²Fresenius Medical Care, North America (FMC-NA), Nashville, TN, USA

Innovative Collaboration Between a Dietetic Internship Program and the MTCRN to Enhance a Renal Diet Education Tool

Mary Sundell, Jane Greene, Dianne Killebrew, Jessica Cox, Holly Darnell Vanderbilt University Medical Center (VUMC), Nashville, TN, USA

Effects of Using an Interactive Approach to Phosphorus Education

Lindsay Weil, Fresenius Medical Care, Chicago, IL, USA

Nutritional Status of Peritoneal Dialysis (PD) Patients after Medical Nutrition Therapy at Singapore General Hospital PD Centre

Po Yee Yu, Singapore General Hospital, Singapore

Peritoneal Dialysis

Peritoneal Inflammation from Sterile Silicone Intraperitoneal Catheters in Rodents and Humans

Mohit Ahuja, Toni Peters, Xiarong Li, Zhi He, Michael Flessner; University of Mississippi Medical Center, Jackson, MS

CT Scans in Encapsulating Peritoneal Sclerosis (EPS): Comparing Two Scoring Methods

Saurabh Bansal, Nasir Siddiqui, Heena Sheth, Filitsa Bender, James Johnston, Beth Piraino, University of Pittsburgh School of Medicine, Pittsburgh, PA

Hemoglobin A1c And 5-Year Survival in 2,798 Chronic Peritoneal Dialysis Patients with Diabetes Mellitus

Uyen Duong, Rajnish Mehrotra, Csaba P Kovesdy, Jennie Jing, Mahesh Krishnan, Allen R Nissenson, Kamyar Kalantar-Zadeh. Harold Simmons Center, Harbor-UCLA, Torrance, CA; VA Salem; DaVita, El Segundo, CA.

The Use of Intravenous Sodium Thiosulfate for the Treatment of Calciphylaxis in an Elderly Peritoneal Dialysis Patient

Stefanie Finch, Irene Aspden, Lyn Johnson, Khalid Bashir Prince George, British Columbia, Canada

Peritoneal Dialysis Modalities and Sodium Removal. No Differences with Optimal Use of Icodextrin

Costas Fourtounas, Periklis Dousdampanis, Andreas Hardalias, Dimitrios Goumenos, Jannis G. Vlachoianis. Department of Internal Medicine-Nephrology, Patras University Hospital, Patras, Greece

Salvage of a Totally Occluded Peritoneal Dialysis Catheter by Laparoscopic Surgery

Costas Fourtounas¹, Ioannis Maroulis², Dimitrios Goumenos¹, Dionysios Karavias², Jannis Vlachoianis¹. Department of Internal Medicine - Nephrology¹ and Department of Surgery², Patras University Hospital, Patras, Greece

Chemical Peritonitis Following Intraperitoneal (IP) Sodium Thiosulphate (STS) For Treatment of Calciphylaxis

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Peritoneal Dialysis: An Old Therapy with Serious Complications

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Tuberculous Peritonitis Following Intravesical BCG Therapy in a Patient with Peritoneal Dialysis

Hilana Hatoum¹, Ali Owda¹, Jagdish Mirchandani¹, Nabil Zaki,¹ Fadi S Rzuq².¹: McLaren Regional Medical Center, Flint, MI. ²: University of Washington, Seattle, WA

Comparison of Mortality of Incident Peritoneal Dialysis (PD) and Hemodialysis (HD) Patients by Age and Diabetes in a National Cohort

Rulin Hechter, Kamyar Kalantar-Zadeh, Csaba P Kovesdy, Jennie Jing, Allen R Nissenson, Rajnish Mehrotra. Harold Simmons Center, Harbor-UCLA, Torrance, CA; VA Salem; DaVita, El Segundo, CA

Peritoneal TB in a Peritoneal Dialysis Immigrant Patient- A Diagnosis to Consider

Kayode Lawrence, Alan Hola , Saad Bhatti , Ellena Linden, George Coritsidis. Elmhurst Hospital Center, Elmhurst NY, USA

Chronic Abdominal Pain in a Patient on Maintenance Peritoneal Dialysis: A Case of Encapsulating Peritoneal Sclerosis

Rupesh Raina and Surafel Gebreselassie, Cleveland Clinic Foundation Cleveland, OH, USA

Systematic Differences Among Patients Initiated on Home Hemodialysis and Peritoneal Dialysis: The Fallacy of Potential Competition?

Jean-Philippe Rioux, Joanne M. Bargman and Christopher T. Chan
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Transplantation

Renal Autotransplantation for Loin Pain Hematuria Syndrome: A Single Center Experience

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Evaluation of Blood Pressure and Renal Function Before and After Renal Transplantation Associated with Cyclosporine Use

Cristhiane F Aguiar, Leandro C D Breda, Débora T R S Abate, Marlene A Reis, Ana C G Faleiros. Triangulo Mineiro Federal University, Uberaba, Minas Gerais, Brazil

A Case of Primary CNS PTLN in a Renal Allograft Recipient with IgG Lambda Monoclonal Gammopathy

Sana Akbar , Sadanand Palekar, Newark Beth Israel Medical Center, Newark, NJ

BK Viruria Incidence in Renal Transplant Recipients with Hepatitis C and CMV who had Delayed Graft Function and Received Thymoglobulin

Sana Akbar, Sadanand Palekar, Vivek Agarwal, Adeem Akbar, Sachin Sachdev, Kezia Alberto; Newark Beth Israel Medical Center, Newark, NJ

Fatal Alemtuzumab Associated Coagulopathy in a Renal Transplant Recipient

Sana Akbar, Sadanand Palekar, Indu Sabnani, Sachin Sachdev, Adeem Akbar
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Prospective BK Viral Screening in Pediatric (Ped) Renal Transplant (RTx) Recipients (Rec) Reduces Graft Dysfunction and Loss Associated With BK Nephropathy (BKN)

Anyaeibu, E I, Al-Akash, S I; Kidney Center, Driscoll Children's Hospital, Corpus Christi, TX, USA.

Use Of Intravenous Immunoglobulin (IVIG) in the Treatment of BK Viremia (TBKV) and Nephropathy in Pediatric Transplant (PTX) RECIPIENTS (REC)

Anyaeibu, E; Al-Akash, S. Kidney Center Driscoll Children's Hospital, Corpus Christi, TX, USA

Correlation Between Immune Cell Function (ICF) Assay and Rate of Rejection and Infection in Renal Transplant Recipients

Nasrin Ashouian, Rajesh Govindasamy, Dan Bucaloiu, Chintalapati Varma, James Hartle Geisinger Medical Center, Danville, PA

Single-Day Work-Up Protocol is Associated with a Shorter Time to Kidney Transplant: the Yale-New Haven Transplantation Center Experience

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Progression of Coronary Artery Calcification (CAC) in Incident Renal Replacement Therapy (RRT) Patients by Modalities of RRT

Subhasish Bose, Sylvia E Rosas. Renal-Electrolyte and Hypertension Division, University of Pennsylvania, Philadelphia, PA, USA

Isolated Renal Aspergilloma Causing Obstructive Uropathy in Renal Allograft- Case Report and Unique Approach to Management

Kellie Calderon, Rajiv Vij and Azzour Hazzan
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Capillary C4d Deposition in Kidney Allografts Biopsies: Relationship with Diagnosis, Immunoglobulines, Renal Function and Fibrosis

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Comparison of Proteinuria with Sirolimus and Tacrolimus in Renal Transplant Recipients

Saurabh Goel, Anas Al Rifai, Mohamed El-Ghoroury, Detroit, MI, US

T-Cell Lymphoma Early After Renal Transplant

Saurabh Goel, Mohamed El-Ghoroury, Detroit, MI, US

Renal Transplantation After Successful Treatment of Calciphylaxis with Sodium Thiosulfate

Deepika Jain, Preethi Yerram, Ramesh Khanna

A 57 Year Old Kidney Transplant Recipient with Skin Lesions, Polyneuropathy and Difficulty Walking

Rabih Kalakeche, Michael Sheehan, Muhammad S. Yaqub, Division of Nephrology, Indiana University, Indianapolis, IN

Hemophagocytic Lymphohistiocytosis in a Renal Transplant Patient

Binaya Khanal, Anjali Parajuli, Satinder Singh, Sri Narsipur SUNY Upstate Medical University, Syracuse, New York

Delayed Graft Function (DGF) in HIV+ Kidney Transplant Recipients Predicts Worse Long-Term Renal Function

Lissa Levin, Gregory Malat, Mohit Gupta, Muhammad Saeed, Snehanika Kulkarni, Sarosh Zafar, Alden Doyle, Ziauddin Ahmed, Karthik Ranganna, Mysore Anil Kumar Drexel College of Medicine/Hahnemann University Hospital Philadelphia, PA

Vitamin D Deficiency and Bone Disease in Renal Transplant Patients: A Missed Opportunity

Julie Ann Linatoc, John Leggat, Raza Qureshi, Bonnie Chapman, SUNY Upstate Medical University, Syracuse, NY, USA

Management of Persistent Hyperparathyroidism with Cinacalcet Following Kidney Transplantation: Thirty Six Month Follow Up

Patrick Lynch, Deep Patel, Nand K Wadhwa and Edward P Nord. Medicine/Nephrology, SUNY Stony Brook, Stony Brook, NY

Schistosomiasis and CMV Colitis in a Renal Transplant Patient

Uma Pakkivenkata, Michael Jin Casey, Division of Nephrology, Hypertension & Transplantation, University of Florida, Gainesville, FL, USA

Influence of Hepatitis C (HCV) Co-Infection on C4D Staining and Presence of DSA in HIV Positive Recipients

Aniruddha Palya, Prasanna Srinagesh, Gregory Malat, Shamin Vania, Usman Lone, Ziauddin Ahmed, Aldan Doyle, Mysore Kumar, Karthik Ranganna. Drexel University College of Medicine, Philadelphia, PA

Minorities Have Survival Advantage Among Rapamycin-Treated Renal Transplant Patients

Kantima Phisitkul, Aleksandra Dyk, Jacqueline Lappin, Hoang Nguyen, and Bhamidipati V.R. Murthy. Divisions of Renal Diseases and Transplantation, UT Medical School, Houston, TX, USA

Microangiopathic Hemolytic Anemia Complicating Renal Transplant

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Intravenous Immune Globulin Associated Nephropathy in Kidney Transplantation

Laleh Razavi, Ashfaq Balla, William Bastnagel, Maung Mya; University of Tennessee HSC, Memphis, TN

Renal Disease in Familial Dysautonomia: Two Case Reports of Successful Transplants

Yelena Rekhtman¹, Andrew S. Bombback¹, Martin A. Nash¹, Scott D. Cohen¹, Dominique Jan¹, Felicia B. Axelrod², Jai Radhakrishnan¹, Gerald B. Appel¹
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African American Race & Male Sex as Risk Factors for Sirolimus Adverse Effects in Renal Transplant Recipients

Sachin Sachdev¹, Adeem Akbar¹, Pallavi Batwar¹, Hiral Desai¹, John Madigan¹, Sadanand Palekar¹. ¹Newark Beth Israel Medical Center. Newark, NJ

Segmental Parasis, a Rare Presentation of Varicella Zoster in a Kidney Transplant Patient

Ankit Sakhuja, Joel Angel, Mohammad Saleh, Syed Hussain, Ehab Saad. Medical College of Wisconsin, Milwaukee, WI, USA

Posterior Reversible Encephalopathy (PRES) Related to Tacrolimus (TAC): A Case Report

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Severe Reversible Renal Allograft Failure from Anti-Mica Antibodies
Nauman Shahid, Simi Shahabdeen, Elizabeth Tecza, Robert McAlack, Alden
Doyle Drexel University College of Medicine Philadelphia, PA

De Novo Immunotactoid Glomerulopathy (ITG) in a Renal Allograft: A Case Report
Salman Singapuri and Sudha Tata, Emory University School of Medicine,
Division of Hypertension and Nephrology, Atlanta, GA

Association of Pre-Transplant Serum Creatinine as a Potential Muscle Mass Surrogate and 5-Year Patient and Graft Survival in 10,090 Hemodialysis Patients
Elani Streja, Csaba P Kovesdy, Jennie Jing, Mahesh Krishnan, Allen R Nissenson, Suphamai Bunnapradist, Gabriel M Danovitch, Kamyar Kalantar-Zadeh. Harold Simmons Center, Harbor-UCLA, Torrance, CA; VA Salem; UCLA Transplant, DaVita, El Segundo, CA

Hypomagnesemia Causing Rapid New Onset Type II Diabetes Post Transplant (NODAT)
D Torri, D Salhan and KD Jhaveri. Nephrology, NS/LIJ and Hofstra Medical School, Manhasset, NY

Bone Disease in Long Term Post Renal Transplant Patients: Single Center Experience
Rajiv Vij¹, Ashfaq Akhtar¹, Malluche HH²
Nephrology, North Shore/LIJ, NY¹ & University of Kentucky, KY²

Other Topic

A Rare Case Of Cocaine Induced Bilateral Renal Infarction
Waqas Ahmed, Madan Gowda, Naseer Khan, Piam Shanesaz, Puneet Bains.
Jewish Hospital Cincinnati, OH.

Iron Repletion Decreases Platelet Counts (PLT) In Non-Dialysis CKD Patients
A Besarab, S Rayamajhi, H Al-Sharif, S Frinak, Jerry Yee. Henry Ford Hospital, Detroit, MI, USA

Comparison of Radiocephalic Fistulas (AVF) Placed in the Proximal Forearm and In the Wrist
Rajeshkumar Bhalodia, Michael Allon , Alan M. Hawxby, Ivan D. Maya, Division of Nephrology, University of Alabama at Birmingham, Birmingham, AL and Department of Surgery, University of Mississippi, Jackson, MS

Trends in Stroke Rates Among Hemodialysis Patients

David T. Gilbertson, Craig Solid, Thomas Arneson, Stephan Dunning, Allan Collins. United States Renal Data System, Minneapolis, MN, USA

Febuxostat in Gout: Serum Urate Responses in Uric Acid Overproducers vs. Underexcretors

David S Goldfarb¹, Patricia MacDonald², Barbara Hunt², Lhanoo Gunawardhana²; 1. New York VAMC & NYU Langone MC, New York, NY, USA & 2. Takeda Research & Development Center, Inc., Deerfield, IL, USA

The Use of Internet as a Resource for Health Information among CKD Patients: A Clinic Based Study

Hooman Hajian, W Brian Reeves, Nasrollah Ghahramani. Penn State Hershey Medical Center, Hershey, PA, USA

Primary Amyloidosis Presenting as Acute Renal Failure with Coagulopathy

Julie Ann Linatoc, Samer Nasser, Sri Narsipur, SUNY Upstate Medical University, Syracuse, NY, USA

Sodium Thiosulfate: A Novel and Effective Treatment for Calciphylaxis

Lama Nouredine, Megan Landis, Nina Patel, Sharon Moe, Division of Nephrology, Indiana University, Indianapolis, IN

Hypertension Management Improved For In-Center Nocturnal Dialysis Patients Compared to Conventional Dialysis Patients

Robert Provenzano^{1,2}; Shane Simon², Ronald Levine², Karen Spach², Allen Nissenson^{1,2}; ¹Office of the Chief Medical Officer, DaVita Inc., Lakewood, CO ²DaVita Inc., Lakewood, CO

Effect of Low Serum Creatinine on Mortality/Morbidity in Hospitalized Elderly Population

Suresh Samson, J. Krishnakurup, Vivian Argento, Yaw Adjepong, Yale University Bridgeport Hospital, Bridgeport, CT, USA

Microalbuminuria and Lactate Dehydrogenase (LDH) as Predictors of Kidney Involvement in a Pediatric Sickle Cell Disease (SCD) Population

Kyla J Scarponi¹, Hilary Hotchkiss², Beth Savage¹, Richard Drachtman¹, and Sevgi Gurkan¹. ¹Pediatrics, University of Medicine and Dentistry of New Jersey(UMDNJ)-Robert Wood Johnson Medical School(RWJMS), New Brunswick, NJ, USA and ²Pediatrics, Mount Sinai School of Medicine, New York, NY, USA

A Case of Pathologic Polyarteritis Nodosa (PAN) in the Setting of Clinical Calciphylaxis

Aastha Sethi, Marzouq Qubti, Jin Park, Stuart Levine, Paul Segal, Ira Mandell, David Spector, Gary Briefel; Johns Hopkins University, Baltimore, MD, USA

IgA Nephropathy Presenting with Pulmonary Hemorrhage

Ebele Umeukeje; David Trochtenberg; Edith Henderson; Minaba Wariboko; Fatima Huma; Agnes Fogo; Chike Nzerue; Department of Internal Medicine, Meharry Medical College, Nashville, TN, USA; Department of Renal Pathology, Vanderbilt University, Nashville, TN, USA

Bilateral Extra-Adrenal Perirenal Myelolipoma Mimicking Retroperitoneal Hemorrhage

Sreelatha Varkala, Jorge Lamarche, Alfredo Peguero, Craig Courville; James A. Haley Veterans Hospital, University of South Florida, Tampa, FL, USA

Research Fellows/Young Investigators

Hepcidin in the Chronic Kidney Disease in Children (CKiD) Cohort Study

Meredith Atkinson¹, Chris Pierce¹, Mark Mitsnefes², Bradley Warady³, Cindy Roy¹, and Susan Furth¹. ¹Johns Hopkins University, Baltimore, MD, United States; ²CKiD Investigator, Cincinnati, OH, United States and ³CKiD Investigator, Kansas City, MO, USA

Activation of Proximal Tubule Sphingosine 1-Phosphate Receptor 1 Protects Kidneys from Ischemia Reperfusion Injury Independent of Lymphocytes

Amandeep Bajwa^{*,§}, Sang-Kyung Jo^{*,§,†}, Hong Ye^{*,§}, Liping Huang^{*,§}, Krishna R. Dondeti^{*,§}, Diane L. Rosin^{†,§}, Volker H. Haase[¶], Timothy L. Macdonald^{**}, Kevin R. Lynch[†], and Mark D. Okusa^{*,§} Departments of Medicine^{*}, Chemistry^{**}, Pharmacology[†] and the Center for Immunity, Inflammation and Regenerative Medicine[§], University of Virginia, Charlottesville, USA. Department of Medicine[¶], Vanderbilt University Medical Center, Nashville, TN, USA

Ergocalciferol Increases Circulating Levels of Human Cathelicidin (hCAP18)

Ishir Bhan, Hector Tamez, Jun Ye, Elizabeth Ankers, Ravi Thadhani Department Of Medicine, Nephrology Division, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA

A Novel Central Mechanism in Uremic Bone Disease

Wai Cheung, Chaim Vanek, Urszula Iwaniec, Russell Turner, Robert Klein, Robert Mak, Department of Pediatrics, University of California San Diego, La Jolla, CA

Mechanisms of Epithelial Cells Injury in Diabetes: Role of Signaling Pathways Mediated by NADPH Oxidases

Assaad Antoine Eid^{*}, Bridget Fagg, Yves Gorin, Karen Block, Jeffrey Barnes and Hanna Abboud, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

Role of Cofilin in Podocyte Actin Dynamics

Puneet Garg¹, Rakesh Verma¹, Leslie Cook¹, Abdul Soofi¹ and Lawrence Holzman^{1,2}. ¹University of Michigan, Ann Arbor, MI, USA, ²University of Pennsylvania, Philadelphia, PA, USA

Polycystins are Required for Endothelial Cell Morphogenesis

Carlo Iomini, Leila Teodora Tchelebi, Lorenzo Battini, Luca Gusella and Patricia Wilson Mount Sinai School of Medicine, New York, NY, USA

The KLK-1 DNA Promoter Methylation Pilot Study in Acute Kidney Injury

Sunwoo Kang, P. Betty Shih, Daniel T. O'Connor, University of California San Diego, San Diego, CA, USA

Lipopolysaccharide Induced Autophagy is Cytoprotective in Renal Tubular Epithelial Cells

Jeremy S. Leventhal¹, Zygimantas Alsauskas¹, Alexandra Snyder², Pengfei Gong¹, Bin Wang¹, Ronald Gordon³, Michael J Ross¹

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Lower Generation of Uremic Solutes P-Cresol Sulfate and Indoxyl Sulfate with a Vegetarian Diet

Frank Luo¹, Kajal Patel¹, Natalie Plummer¹, Laurie Steinberg¹, Thomas Hostetter², Timothy Meyer¹. Stanford University, Palo Alto, California¹ and Albert Einstein College of Medicine, New York, USA²

p120 Catenin Regulates Epithelial Tubulogenesis in Proximal Tubules

Denise K. Marciano¹, Paul R. Brakeman², Chao-Zong Lee¹, Natalie Spivak², Dennis J. Eastburn³, Keith E. Mostov³, Louis F. Reichardt⁴

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Novel Markers of Anemia Risk in Chronic Kidney Disease

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Physical Activity and Albuminuria

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Gd-IgA1: A Screening Test for Pediatric IgA Nephropathy?

John Sanders¹, Zina Moldoveanu², Grant Sommes³, M. Colleen Hastings¹, Wen-Qiang Huang², Sherry Walker⁴, Olivia Hancox⁴, Jan Novak² and Robert Wyatt¹
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Primary Cilia and Fluid Flow Establish the Orientation of Mitotic Spindles

Neeraj Sharma and Bradley K. Yoder
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Polycystin-1 Interacts With Arf4 and Rab GTPases to Traffic to Renal Primary Cilia

Heather Ward¹, Dusanka Deretic², Angela Wandinger-Ness¹. Departments of ¹Pathology and ²Surgery, University of New Mexico, Albuquerque, NM, USA

Non-Viral Gene Transfer for Renal Disease and Complications

Matthew H. Wilson, Michael E. DeBakey VA Medical Center and Department of Medicine, Center for Cell and Gene Therapy, Baylor College of Medicine, Houston, TX, USA

Novel Role of the TIM-4 Molecule in Alloimmunity

Melissa Y Yeung¹, Takuya Ueno¹, Hisaya Akiba², Hideo Yagita², Nader Najafian¹ and Mohamed Sayegh¹
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Bundled Payment for Dialysis Services Under Medicare

A Simplified Case-Mix Adjuster Model for the Proposed End Stage Renal Disease (ESRD) Prospective Payment System (PPS)

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Alternative Higher Predictive Model for the End Stage Renal Disease (ESRD) Prospective Payment System (PPS)

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Impact of Reductions in Infectious Complications, With the Use of the Hero Vascular Access Device, On Dialysis Provider Revenue in an Era of Bundling

Lesley Dinwiddie, Vascular Access for Hemodialysis, Cary, NC, USA

Failure to Replicate CMS Case Mix Adjusters and the Potential Impact on Dialysis Reimbursement

Tracy J. Mayne¹, Mary Burgess¹, Joe Weldon¹

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Potential for Racial Disparities in the Proposed Medicare Dialysis Prospective Payment System

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DIABETIC MYONECROSIS – RARE CAUSE OF AKI

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We report a complication of uncontrolled diabetes, diabetic myonecrosis and report in our knowledge, the first case of acute kidney injury (AKI) associated with diabetic myonecrosis. 46 year old female with 9 year history of type 2 diabetes mellitus on Insulin (HbA1c 10.4) presented to hospital with painful left thigh swelling for 1 week. She was diagnosed as having inflamed lipoma and discharged home. She revisited ED after 2 weeks with persistent pain. On examination, she was afebrile and had a firm, tender swelling on the medial aspect of left thigh measuring 8 x 20cm with no fluctuance/erythema, her contralateral thigh being normal. Muscle strength and motion were limited due to pain. Pedal pulses were present bilaterally and ABI was normal. Laboratory tests revealed elevated BUN and creatinine; 120 and 5.2 mg/dl respectively from baseline 12 and 1.2 mg/dl. Serum myoglobin was 1695 ng/ml. Urine analysis showed blood ++ and no RBC. Renal Ultrasound was unremarkable and Doppler of lower extremities was negative for DVT. CT of left Lower extremity showed diffuse edema and enlargement of “medial vastus” with no localized fluid collection confirming the muscle infarction. AKI was secondary to rhabdomyolysis caused by diabetic myonecrosis. After 5 days on supportive management, AKI improved. Follow up at 10 weeks showed complete resolution. Previous case series have shown Diabetes myonecrosis is seen in patients with poorly controlled and long standing diabetes and is often misdiagnosed as a neoplasm, an abscess, or myositis (In our case as an inflamed lipoma). Recurrence is seen in upto 50% of the cases; our patient returned with left lateral vastus (previously left medial vastus), muscle infarction after 2 months of prior episode. The current case is the first report of myonecrosis causing AKI. This case serves to remind us that although diabetic myonecrosis is a rare entity, it can present with rhabdomyolysis / acute kidney injury, should be kept in mind in a clinical setting of rhabdomyolysis in a patient with uncontrolled diabetes mellitus.

ACUTE RENAL FAILURE: A CASE OF BURKITT'S LYMPHOMA

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We are reporting a patient who presented with acute renal failure secondary to diffuse bilateral renal high grade lymphoproliferative infiltration by Burkitt's lymphoma (BL). The patient temporarily required hemodialysis and was aggressively treated with chemotherapy which lead to complete resolution of his renal failure.

A 41 year-old African American male presented to our hospital with a 3 day history of mental status changes, confusion, inability to care for himself, nausea, poor appetite, poor oral intake and left thigh pain.

Laboratory data disclosed a complete blood cell count with a WBC of 4.3 K/uL, PLT < 5,000 K/uL, HCT of 34.6 %, HGB of 12.7g/dL. Serum urea was 96mg/dL, serum creatinine 9.5mg/dL, sodium 130mmol/L, potassium 5.2mmol/L, chloride 94mmol/L, bicarbonate 16mmol/L, glucose 202mg/dL, calcium 9.3mg/dL and the anion gap was 20. Complement levels, ANA, ANCA, hepatitis serologies and HIV were normal or negative. His renal ultrasound showed bilateral kidney size near the upper limit of normal.

Bone marrow biopsy showed a hypercellular bone marrow (90%) involved by a high grade B cell lymphoproliferative disorder suggestive of Burkitt's lymphoma. Renal biopsy showed a high grade lymphoproliferative infiltrate compatible with Burkitt's lymphoma. The interstitium was grossly involved by a high grade mononuclear cell infiltrate.

Our patient who received hyperfractionated cyclophosphamide, vincristine, adriamycin and dexamethasone alternating with high dose methotrexate and cytosine arabinoside had complete improvement of his renal function back to baseline after 3 weeks of treatment and renal replacement therapy was discontinued.

With this case report we want to emphasize that acute renal failure in conjunction with constitutional symptoms, lymphopenia, elevated LDH and enlarged kidneys should suggest renal lymphoma and early diagnosis and treatment should be pursued. Treatment of the primary lymphoma is usually associated with recovery of the renal function. Patients presenting with limited stage disease have an excellent prognosis. Complete remission rates are very high, but the cure rate is generally less than that observed for diffuse large B-cell lymphomas. Rapid diagnosis and treatment are essential for better outcome and survival.

PERINATAL CHARACTERISTICS OF PREMATURE INFANTS WITH ACUTE KIDNEY INJURY

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Purpose: Acute kidney injury (AKI) is an independent predictor of mortality in many patient populations.

Evaluation of perinatal characteristics associated with AKI may yield insight into pathogenesis and prevention.

Methods: Infants with birth weight between 500-1500g and gestational age >25 weeks were enrolled from a regional quaternary care NICU. AKI was determined using a new proposed neonatal definition with AKI = SCr \geq 1.5 mg/dl in 1st week of life, or \uparrow SCr \geq 0.3 mg/dl from previous value, or \uparrow SCr \geq 150-200% from previous value.

Results: Over 18 consecutive months, 229/359 (65%) infants agreed to participate in the study.

Maternal	Infant
Age = 25.8 ± 6.2 ; p 0.19	GestA wks 25 ± 2 ;p 0.001
Prenatal care= 68% ;p0.68	Birth weight 702 ± 205 ;
Diabetes = 7.3% ; p 0.97	Cord pH 7.28 ± 0.10 ; p 0.28
Hypertension= 27%;p0.05	Apgar1min 3 SE 0.31 ;p0.02
Indomethacin =5% ;p 0.1	Apgar 5 min 6 SE0.24 ;p0.001
Cigar smoke =2.4%;p0.2	UmbilicalCatheter = 66 % ; p 0.03
Preeclampsia = 17%;p0.02	Ventilator = 88% ;p <0.0001
Mult. births =22 % ;p0.4	
Chorio = 2.4 % ;p 0.2	

Conclusions: AKI was associated with younger gestation, lower Apgar scores, and greater intensive care needs and inversely associated with maternal HTN and preeclampsia.

EIGHT CASES OF ACUTE KIDNEY INJURY DUE TO VANCOMYCIN TOXICITY

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Controversy exists as to whether Vancomycin (VAN) independently can be a causative agent for Acute Tubular Necrosis (ATN). Recent guidelines have recommended higher VAN trough levels for patients with severe infections. This change has led to utilization of higher VAN dosages.

We report eight patients who developed Acute Kidney Injury (AKI) presumed to be ATN, associated with supra-therapeutic levels of VAN in the absence of other nephrotoxin exposure. Patient #5 was biopsied which revealed ATN. Two of the eight patients required dialysis. All eight patients recovered after VAN was held.

With increased utilization of higher VAN doses, VAN alone could be considered nephrotoxic.

Table

Pt	Sex	Age yrs	Wt kg	Dose	Trough µg/ml	Initial Cr mg/dl	Peak Cr mg/dl	HD
1	F	49	96	2g q8hr	105.3	0.86	3.76	Y
2	M	53	134	2g q12hr	93.8	1.2	4.84	N
3	F	20	60	2g q8hr	54.3	0.46	4.19	N
4	F	46	79	1g q8hr	90.8	0.6	2.48	N
5	M	23	92	2g q12hr	64.7	0.97	8.41	Y
6	F	24	131	2g q8hr	56.0	0.68	3.23	N
7	F	65	58	1.5g q12hr	58.6	0.7	3.64	N
8	F	53	69	1g q8hr	38.2	0.8	4.64	N

HEMODIALYSIS IS EFFECTIVE FOR TREATMENT OF HYPOTHERMIA

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Hypothermia is a medical emergency. In severe cases, the gold standard for re-warming is cardiac bypass. This is not available at all hospitals. We present 2 cases of hypothermia treated with intermittent hemodialysis (HD) and a systematic review of similar cases.

CASE #1: A 22 year old female was found after a suicide attempt with a temperature of 25.1°C. Intermittent HD was initiated when it was 30.7°C. After 90 minutes it was 33.4°C.

CASE #2: A 45 year old male was found after falling from a height of 30 feet with a temperature of 26.6°C. Intermittent HD was initiated when it was 28.2°C. After 120 minutes it was 32.7°C.

A search of English language literature was conducted for studies of dialysis for the treatment of hypothermia (1960 - May 2009) using MEDLINE, PREMEDLINE, and CINAHL. Search terms for dialysis included continuous venovenous hemofiltration (HF)/hemodiafiltration (HDF), continuous arteriovenous HF/ HD, HD, and ultrafiltration. We reviewed in detail 25 articles, 9 articles describing 11 cases met our criteria and rate of change could be calculated. The 11 cases and our 2 cases are compared in the following table.

Author, Year	Modality	Rate of change (°C/hours)
Komatsu, 2007	CVVHDF	1.38
van der Maten, 1996 (2 cases)	CVVHD	1.11 & 2.2
Wagner, 2008	CVVHD	1.93
Brodersen, 1996	HF	1.33
Spooner, 2000	HF	4
Hernandez, 1993	HD	2.15
Murray, 1995 (2 cases)	HD	3.6 & 1.16
Owda, 2001	HD	1.9
Sultan, 2009	HD	1.9
Dahlberg, 2009 (2 cases)	HD	1.8 & 2.25

Renal replacement modalities appear to be effective and timely in speeding re-warming in hypothermia.

A CHALLENGING CASE OF PARTIAL NEPHROGENIC DIABETES INSIPIDUS

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Nephrogenic diabetes insipidus (NDI) is characterized by the inability of the kidney to concentrate urine in response to arginine vasopressin. Disturbance of the aquaporin-2 shuttle is the underlying molecular basis of the acquired NDI. The consequences are severe polyuria and polydipsia, often associated with hypertonic dehydration. We report a case of an acquired partial NDI in the setting of acute kidney injury, liver cirrhosis and protein malnutrition.

A 52 year old African American male with hypertension and chronic alcohol abuse, not taking prescription or over the counter medications was found unresponsive. For one week prior, the patient had been drinking alcohol and using intravenous heroin. He had icterus, dry oral mucosa, tenderness to palpation in the right upper quadrant and bilateral leg edema. On admission, laboratory data showed: serum creatinine 3.53 mg/dl, sodium 132 mmol/L, total bilirubin 17.9 mg/dl, albumin 1.9 g/dL. After two days of appropriate fluid challenge, the acute kidney injury resolved and the serum sodium normalized. The urinary output remained constant, between 3700 and 5000 ml/24H, regardless of the amount of intravenous saline solution administered. The serum sodium gradually increased to 161 mmol/L on the 7th day of admission. Concomitantly, the patient had a serum osmolality of 339 mOsm/kg, urine sodium 43 mmol/L and urine osmolality of 444 mOsm/kg. The patient received desmopressin, with no significant change in the urine sodium and osmolality (53 mmol/L and 497 mOsm/kg, respectively). After receiving hydrochlorothiazide, there was a partial response with the increase in the urine sodium and osmolality to 72 mmol/L and 520 mOsm/kg, respectively, and a significant decrease in the urinary output to 2200 ml/24H. The patient was diagnosed with partial NDI as opposed to the polyuric phase of acute tubular necrosis.

Partial NDI represents a diagnostic challenge in most cases. Differentiating between excessive diuresis of the acute kidney injury recovery and NDI is essential, since the therapeutic approach is different and failure to diagnose NDI could lead to severe and life-threatening hyponatremia and dehydration.

ACUTE KIDNEY INJURY IS ASSOCIATED WITH THE USE OF INTRAVENOUS VANCOMYCIN IN HOSPITALIZED PATIENTS

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Background: Vancomycin has largely ceased to be considered a major cause of drug-related Acute Kidney Injury (AKI) in the years since its reformulation. However, recent reports have suggested that increased levels of vancomycin, especially in combination with aminoglycoside use, may be associated with an increase in creatinine.

Purpose: To determine if an association exists between vancomycin use and AKI; and correlate serum vancomycin levels with plasma creatinine levels in hospitalized patients receiving vancomycin.

Methods: We expanded our previous retrospective chart review, to now include 768 hospitalized patients who had received vancomycin over a 2-year period. Those who had end stage renal disease, incomplete data or had received less than two doses of vancomycin were excluded (166). Data collected included patient demographics, comorbid conditions, and serial vancomycin levels and creatinine values. Data were analyzed using Student's t-test to compare mean maximum vancomycin trough levels in AKI vs. non-AKI patients, and linear regression analysis to elucidate dose-dependent relationship between maximum vancomycin trough levels and creatinine and change in creatinine.

Results: Our analysis included 602 patients for whom there were complete data, of whom 176 (29%) developed AKI as defined by an increase in creatinine by $> 0.5\text{mg/dL}$ or by $> 25\%$ of baseline. The mean maximum vancomycin trough level in the patients who did not develop AKI was 13.2 ± 5.9 mcg/mL, and that in the AKI group was 17.7 ± 8.0 ($p < 0.0001$).

Conclusion: AKI is associated with the use of intravenous vancomycin, especially at higher doses. Although dose-dependent toxicity could not be established, 29% of the patients developed AKI. Attention should be paid to drug levels, and the dose of vancomycin should be adjusted in a timely fashion in order to prevent AKI. Its use should be monitored closely.

**ELEVATED BUN WITH NORMAL CR AT ICU ADMISSION
PREDICTS SHORT TERM AND LONG TERM MORTALITY**

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We hypothesized that elevated BUN would be associated with all cause mortality independent of Cr in the critically ill. We performed an observational study of patients treated in 20 medical and surgical ICUs between 1997 and 2007 in two hospitals in Boston. The study cohort was 28,737 patients ≥ 18 years with Cr 0.80-1.30 mg/dl and BUN ≥ 10 mg/dl at ICU admit. The main outcome measures were 28, 90 and 365 day mortality. Cox proportional and Logistic regression models were performed. Adjustments included Cr, Charlson index and transfusions.

A stepwise increase in mortality occurs with increasing BUN. The multivariable hazard ratio for death of a 10 mg/dl BUN increase at ICU admission is 1.22 (95% CI, 1.19-1.24; $P < .0001$). Compared with BUN 10-20 mg/dl, the multivariable hazard ratio with BUN >20 to 40 mg/dl is 1.35 (95% CI, 1.3 to 1.4; $P < .0001$) and with BUN >40 mg/dl it is 2.55 (95% CI, 2.3 to 2.8; $P < .0001$).

BUN at ICU admission is predictive for short term and long term mortality. At 28 days following ICU admission, the multivariable odds ratio for death of 10 mg/dl BUN increase at ICU admission (OR) is 1.35 (95% CI, 1.30-1.42; $P < .0001$). At 365 days following ICU admission, the OR is 1.39 (95% CI, 1.34-1.44; $P < .0001$).

Elevated BUN at ICU admission is a significant predictor of short and long term all cause patient mortality independent of Cr.

ENERGY ENHANCEMENT TO SEVERE TOXICITY REQUIRING A TRANSPLANT – A CASE OF *EPHEDRA* TOXICITY

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Dietary supplements containing *Ephedra* alkaloids are commonly found ingredient in weight loss and energy enhancement supplements. In 2004, the FDA banned Ephedra-containing products because of numerous reports of adverse cardiovascular and neurological events. Although it can still be obtained as a traditional Chinese medicine, *Ma Huang* and synthetic ephedrine (*Synephrine*) have also been created as an over-the-counter medication for asthma and energy-boosting products.

We present the case of a previously healthy 34-year-old Caucasian male with a history of *Ephedrine* ingestion for “energy boost.” He was admitted 2 days after an ingestion of 10 pills of *Ephedrine* with complaints of nausea, vomiting and severe abdominal pain. He was found to have severe acute hepato-renal failure. AST–13,786 units/L, ALT–6,714 units/L with coagulopathy, with INR–6.45 and acute renal failure (ARF) with BUN–16 mg/dL, and Cr–5.19 mg/dL. He had anion-gap metabolic acidosis with acute respiratory alkalosis. His urine was dark on exam, positive for blood, hemoglobin and myoglobin with mildly elevated CK. He was also found to have hyperkalemia with K-6.6 mmol/L, hypocalcemia with Ca-7.4 mg/dL, and hyperphosphatemia with phosphorus-7.5 mg/dL consistent with acute rhabdomyolysis. Liver biopsy showed 90% necrosis of hepatocytes. He required temporary dialysis with continuous veno-venous replacement therapy. After emergent liver transplant he recovered his renal function and was discharged home without any further need of dialysis.

Our patient was thought to have toxic hepatitis with, ARF from rhabdomyolysis and hypoperfusion kidney injury from vasoconstrictive affects of *Ephedrine*. However, he had no prior history of exercise and yet was showing signs of acute rhabdomyolysis. This point towards the importance of extreme caution in using these supplements, as it is commonly used in the setting of weight loss, energy and performance enhancement, and often by younger generation without any knowledge of the severe toxicity involved, placing them at higher risk.

ACUTE KIDNEY INJURY AFTER ROBOTIC ASSISTED LAPROSCOPIC RADICAL PROSTATECTOMY (RARP), HOW REAL IS IT?

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The overall incidence of acute kidney injury is 24-30 cases/1000 hospital discharges. We present an interesting case of AKI whose management differed as the raise in creatinine was result of reabsorption of accumulated urinary creatinine.

62 y/o male presented 2 days after having a robotic prostatectomy with abdominal fullness & increased drainage from the JP drains. As urinary out put declined and creatinine increased to 4mg/dl nephrology was consulted. Abdominal girth increased. Urinary leak into peritoneal cavity was suspected. Creatinine sent on the abdominal fluid was 9.6mg/dl confirming spillage of urine into abdominal cavity. Paracentesis removed 10 liters of fluid. An urethro cystoscopy with urethrogram confirmed the leak at the posterior anastamotic site, and foley repositioned. The JP drains out put decreased and serum creatinine normalized.

Complications rate for RARP ranges from 2.3% to 21.6%. The elevation of serum creatinine as a result of reabsorption of urinary creatinine from urinary leak into abdominal cavity has not been clearly described in literature. Given the different mechanism contributing to AKI, the management differed. Such mechanism causing spurious AKI should be kept in mind while evaluating post operative urology patients, & testing the ascitic/abdominal fluid for creatinine will aid in confirming the diagnosis.

DILEMMAS FOR A NEPHROLOGIST IN A CASE OF ACUTE LEUKEMIA

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Renal failure is an uncommon manifestation of leukostasis. Unexplained renal failure associated with hyperleukocytosis and improvement in renal parameters with cytoreduction indicates a possible association. Hypokalemia due to inappropriate kaliuresis in a patient with myelomonocytic leukemia is due to increased lysozyme levels.

A healthy 29 year old male presented with fever, lymphadenopathy and hepatosplenomegaly. Lab work showed a WBC count of 161.7k/ μ l, BUN 17mg/dl and Cr 1.8mg/dl. Bone marrow biopsy revealed an acute monocytic leukemia with some lysozyme positive cells. The patient subsequently developed respiratory failure secondary to leukostasis. Therapeutic leukapheresis was initiated and the patient showed marked improvement after 3 cycles of leukapheresis. His serum Cr decreased concomitantly with a substantial fall in WBC count. Persistent hypokalemia was also seen. Calculated Trans Tubular Potassium Gradient (TTKG) was 5.1. Patient had increased serum lysozyme (>32g/ml) during this period. Lymph node biopsy showed lysosomal staining of leukemic cells. Eventually hypokalemia resolved coinciding with normalization of lysozyme levels and leukocytoreduction.

Our patient had tumor lysis syndrome (TLS) on admission. Serum Cr showed no improvement even after resolution of TLS suggesting another etiology. Myeloblasts are hyperviscous and hence hyperleukocytosis can cause renal failure by leukostasis. Improved renal function with reduced leukocyte load suggests a link between cell load and renal failure. Lysozyme interferes with tubular cell function leading to potassium wasting. A high TTKG of 5.1 reflects inappropriate kaliuresis. Concomitant normalization of the serum K⁺, lysozyme levels and reduction in leukocyte counts indicate a relationship.

Acute myelomonocytic leukemia can be associated with renal failure secondary to leukostasis. Hypokalemia due to renal K⁺ wasting with high lysozyme levels is also a unique feature of this leukemia subtype.

UTILITY OF SCUF IN HYPOTENSIVE ICU PATIENTS

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Despite severe volume overload sometimes it is difficult to remove fluid by Continuous VenoVenous Hemofiltration (CVVH) in some ICU patients. Different methods have been tried to maintain hemodynamic stability such as use of vasopressors (VP), decreasing ultra filtration (UF) rate, isovolemic CVVH etc. Slow continuous UF (SCUF) was performed in two patients who needed volume removal but could not tolerate even isovolemic CVVH due to hypotension.

Data was collected on 2 such patients in the ICU which included the mode of therapy (CVVH vs SCUF), use of VP, volume removed per day, blood pressure (BP), blood flow rate and dialysate flow rate.

The first patient a 64 year old female with ESRD (end stage renal disease) on Hemodialysis was admitted to the ICU due to sepsis. The CVVH was started due to hypotension. After 2 days, despite being on isovolemic CVVH, patient exhibited hypotension, increase in VP and oxygen requirement. CVVH was switched to SCUF and in next 24 hours 6.3 liters of fluid was removed, VP requirement decreased with improvement of BP. CVVH was reintroduced due to hyperkalemia after 2 days of SCUF and the patient again became hypotensive. The modality was then switched to SCUF with successful removal of 3.9 to 4.2 liter of fluid per day. During this time the antibiotic regimen was re-adjusted, the patient became alert and was extubated. CVVH was resumed for hyperkalemia and the patient at this time tolerated the CVVH.

The second patient a 58 year old female presented with septic shock and was placed on CVVH and antibiotics. Patient had significant volume overload, however she became hypotensive, requiring increased VP despite being on isovolemic CVVH. SCUF was utilized twice and both the times 3.4 and 5.2 liters volume per day was removed with subsequent improvement in BP and decreased need for VP. This patient also subsequently tolerated CVVH.

From the above observation it appears that there can be an interim role of SCUF in critically ill patients when CVVH is not tolerated. A larger study is needed to confirm this finding.

ACUTE RENAL FAILURE SECONDARY TO CARBIDOPA/LEVODOPA INDUCED RETROPERITONEAL FIBROSIS

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Retroperitoneal Fibrosis (RF) is an uncommon but treatable cause of obstructive nephropathy. RF presents insidiously making the diagnosis difficult. Prompt diagnosis is essential in order to avoid sequelae such as ESRD. Dopamine agonists such as bromocriptine have been reported as having an association with developing RF. In the present report, a 59 y/o male with history of benign prostatic hypertrophy (BPH) was initiated on carbidopa/levodopa to treat restless leg syndrome. Four weeks after initiating treatment, the patient presented with testicular pain and low back pain of 2 weeks duration. The serum creatinine was 1.7 mg/dl. His baseline creatinine was 0.8 mg/dl. A renal ultrasound revealed bilateral hydronephrosis. He underwent a cystoscopy, which was normal, and discharged with a foley catheter as it was presumed his renal failure was secondary to BPH. One week later his serum creatinine was 10.8 mg/dl. A CT scan of the abdomen and pelvis revealed findings suggestive of RF. He was offered biopsy to rule out malignancy as a cause but he declined. Bilateral nephrostomy tubes (NT) were placed and oral prednisone was initiated. In 4 days his serum creatinine was 2.4 mg/dl. One year later the RF has almost completely regressed and his serum creatinine is 1.2 mg/dl.. To our knowledge this is the first reported case of a temporal association between carbidopa/levodopa and RF.

CMV NEPHRITIS IN NATIVE KIDNEYS OF A PATIENT WITH MYCOSIS FUNGOIDES

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CMV is a common infection seen in transplant recipients and immunosuppressed patients. Prior studies have reported CMV-associated renal injury limited to allograft recipients and autopsy samples. There have not been any reports of CMV nephritis in native kidneys. We present a case of CMV nephritis in the native kidneys of a woman with newly diagnosed Mycosis Fungoides (MF).

A 48 year old woman with psoriasis refractory to immunosuppressive therapy was admitted to the hospital with failure to thrive and acute renal failure. Because of the worsening rash and overall clinical condition, MF was suspected and confirmed by a repeat skin biopsy. The patient's renal function continued to decline despite adequate fluid resuscitation. Given her active urine sediment and 1.8 grams of proteinuria, a kidney biopsy was done which showed acute tubulointerstitial nephritis with CMV viral inclusions. The patient was treated with IV Gancyclovir and her creatinine stabilized. However, due to other comorbidities she continued to deteriorate and eventually died of septic shock.

CMV is one of the most common infectious complications seen in renal allograft recipients. Its role as a causative agent for direct renal injury remains controversial because CMV viral inclusions are seen in only 1% of biopsies. There have been no proven cases of CMV causing direct injury in native kidneys even in immunocompromised patients. We present for the first time a patient with newly diagnosed MF who had previously been on immunosuppressive therapy and subsequently developed acute renal failure secondary to CMV nephritis. Previous reports suggest that over 97% of MF patients are seropositive for CMV compared to only 57% of healthy controls. Patients with MF develop severe immunodeficiency and CD8⁺ T cell depletion as their disease progresses. The combination of immunosuppressive therapy and immunodeficiency associated with MF likely led to reactivation of CMV infection in this patient.

CLINICAL AND IMMUNOHISTOLOGICAL ANALYSIS OF 28 CASES OF BIOPSY-PROVED ACUTE INTERSTITIAL NEPHRITIS

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Acute interstitial nephritis (AIN) is an often reversible cause of kidney injury induced by drug hypersensitivity reactions, systemic diseases, or infections. While drug exposure history, eosinophiluria, rash, and fever are felt to be indicative of the disorder, renal biopsy remains the gold standard for diagnosis of AIN. The disease can progress to fibrosis and potential ESRD. We hypothesize that mast cell degranulation may be implicated in the development of tissue fibrosis in AIN, and that the involvement of mast cells may differ depending on the etiology of AIN.

We reviewed all native kidney biopsies performed at our institution between 2002 and 2007, and identified 28 cases of acute interstitial nephritis. Clinical history was reviewed, and renal biopsy material was stained for β -tryptase, IL-16, CD-3, and IgE.

Twenty cases (72%) were medication-induced, 4 were idiopathic (14%), and 4 were associated with systemic diseases (14%). Eosinophiluria was assessed in 25 patients, and found to be present in only 8 (32%). Rash was present in 5 of 28 (18%), and fever was present in 4 of 24 (17%). 24 of 28 (86%) were treated with systemic corticosteroids. Five patients required dialysis; 2 progressed to ESRD. Medication-induced disease accounted for both cases of ESRD and 2 of 3 cases requiring short term dialysis. β -tryptase staining revealed a mean of 21 cells/HPF, with IgE staining an average of 0.68 cells/HPF. IL-16 stained an average of 28% of the interstitium, and CD3 stained 33% of the interstitium. There was no difference between groups in staining for β -tryptase ($p=0.59$), IL-16 ($p=0.37$), IgE ($p=0.26$), or CD3 ($p=0.32$). Stains were not predictive of death or dialysis requirement.

In this study, we have demonstrated a potential role of mast cells and IgE in acute interstitial nephritis. The expression pattern of β -tryptase, IgE, CD3, and IL-16 did not differ depending on the etiology of acute interstitial nephritis.

GEMCITABINE INDUCED HEMOLYTIC UREMIC SYNDROME

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Hemolytic uremic syndrome (HUS) is characterized by the clinical triad of acute kidney injury, microangiopathic hemolytic anemia and thrombocytopenia. We report a 43-year-old man who developed HUS secondary to Gemcitabine, which has been recognized but is relatively rare.

A 43-year-old man with history of adenocarcinoma of the pancreas was admitted with a three-week history of shortness of breath. Four months prior, the patient was started on chemotherapy with Gemcitabine. He was on a regimen three weeks on one week off. Gemcitabine was continued until 2 weeks prior to hospitalization with a total dose of 15,640 mg administered. The tumor marker, CA19-9, decreased from 914 to 239 U/mL. Pertinent findings are signs of volume overload and petechial hemorrhage. Laboratory testing showed an increase creatinine level of 2.9 mg/dL from 1.1 mg/dL as a baseline. The platelet count was $83 \times 10^3 /\mu\text{L}$ with serum lactate dehydrogenase (LDH) of 949 U/L and fibrinogen of 342 mg/dL. A peripheral blood smear showed microangiopathy. The clinical and laboratory findings were consistent with a diagnosis of HUS. The patient was promptly treated with plasmapheresis and dexamethasone. Stool culture for *E. coli* O 157: H 7 was negative. Biopsy was not done due to comorbidities. Despite plasmapheresis, kidney injury did not recover; hemodialysis was initiated and continued as an outpatient upon discharge.

This case proposes that direct endothelial injury and dysregulation of thrombotic processes from complement-fixing circulating immune complexes are the key features. Endothelial injury from circulating immune complexes leads to platelet aggregation, microvascular thrombosis and eventually leads to acute kidney injury. Plasmapheresis should promptly initiate and continue until circulating immune complexes become undetectable with normal serum complement levels. Because this complication is critical, awareness and early recognition of the etiology are warranted.

OCCUPATIONAL HAZARD: LEPTOSPIROSIS AND ACUTE KIDNEY INJURY

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Leptospirosis is caused by spirochetes of the genus *Leptospira* and by exposure to water contaminated by urine from infected animals. Disease manifestations vary from mild influenza-like illness to fatal multi-organ failure.

A 36 year old man complains of fever and calf pain for 5 days. He had recently been hunting and his symptoms progressed to generalized myalgias, nausea, vomiting, and blood tinged urine. Mosquito bites were noted on physical exam and labs suggested acute renal failure with BUN 84mg/dL and creatine 4.2mg/dL. Total bilirubin was 7.2mg/dL. Urine showed proteinuria, blood, urobilinogen, leukocytes, and granular casts.

A fluid trial was started; however, he developed anuria and respiratory failure. He was intubated and continuous renal replacement therapy started along with empiric antibiotics. Ultrasound showed enlarged kidneys. Immunologic work-up was negative and studies for Influenza, Lyme, HIV, West Nile, and Hepatitis were negative. Leptospirosis antibody was positive and antibiotics changed to penicillin and ceftriaxone. He remained on CRRT for a week. After 1 month he had complete recovery of his renal function.

90% of leptospirosis cases are mild self-limited anicteric forms. However, those that progress to the more severe form, Weil's syndrome, mortality can be as high as 55%. The triad of Weil's syndrome includes jaundice, renal failure, and hemorrhage. Renal involvement is common because the organism colonizes the renal tubules, leading to renal failure in 60% of severe cases. Seroconversion, antibody titer, and leptospiral culture become positive late into the illness. Clinicians need to recognize the clinical presentation so as to start urgent and appropriate therapy thereby preventing progression to multi-organ failure and increased mortality.

“WE WOULD, AND WE WOULD NOT”: A TRAGIC CASE OF KELL

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Acute Hemolytic Transfusion Reaction (AHTR) is a medical emergency requiring immediate intervention. Classic triad of fever, flank pain, and hemoglobinuria is rarely seen; fever and chills may be the only manifestation.

A 44-year-old African American woman was admitted for vaginal hysterectomy (VH) due to a 4 month history of menometrorrhagia. Medical history was significant for Systemic Lupus Erythematosus, controlled on hydroxychloroquine sulfate. Vital signs were unremarkable and pre-op hematocrit was 30.8%. She was A positive but had an anti-Js^b antibody. The surgical team was informed that no appropriately typed blood was in the hospital. Due to the patient's urgent condition a VH was performed without any initial complications. Post-op, she decompensated with a hematocrit of 19.3%. Laparotomy showed bleeding from surgical sites. Left oophorectomy and bilateral hypogastric artery ligation were performed. She was resuscitated with product including 12 units Js^{b+} blood. Follow-up hematocrit was 40.8%. Clinical findings and labs were consistent with AHTR. Due to severe heme pigment-induced acute tubular necrosis, continuous renal replacement therapy was initiated but not continued due to hypotension. No signs of lupus exacerbation or disseminated intravascular coagulation were found. Despite receiving Js^{b-} blood, anemia worsened and post-op day 6 she succumbed to multisystem organ failure.

Js^b is a member of the Kell system. It is seen on RBC of nearly 100% Caucasian and 99% African descent individuals. Js^{b-} phenotypes are not seen on RBC of Caucasians and only 1% of African descent individuals. Most patients suffer from mild to moderate hemolytic anemia that resolves after supportive therapy. To our knowledge this is the first case of a Js^{b-} individual receiving 12 units of Js^{b+} blood at one time who eventually died due to AHTR.

ACUTE RENAL FAILURE IN POISONOUS SNAKE BITES

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We sought to determine clinical factors associated with development of acute renal failure (ARF) in poisonous snake bites.

Our method involved prospective study of 3128 patients with poisonous snake bites who presented to Coimbatore Medical College Hospital, India from January 2004-January 2007. "Poisonous" was defined by presence of characteristic fang marks, presence of severe cellulitis and lymphadenopathy, or direct visualization of the culprit snake. Anti Snake Venom (ASV) was administered to patients who showed signs of envenomation. Initial evaluation included identification of the snake, evidence of envenomation such as fang marks, cellulitis, regional lymphadenopathy, abnormal clotting time, hypotension and ptosis. ARF was defined by serum creatinine >50% above baseline or urine output < 0.5ml/kg/hr for >12 hours. No afflicted patients were known to have chronic renal failure.

ARF was seen in 588 patients (18.8%). Independent factors associated with increased risk of ARF were regional lymphadenopathy (91%), cellulitis (95%), prolonged clotting times (76%), DIC (32%), and hypotension (22%). Mortality was 12% in ARF vs 4% without ARF. ARF patients average 18.6 hours between bite to ASV, vs. 6.5 hours in pts without ARF.

We conclude that initial clinical findings which are more likely to be associated with ARF in poisonous snake bite include lymphadenopathy, cellulitis, prolonged clotting times, DIC, and hypotension. A delayed bite-to-ASV time is also associated with higher likelihood of ARF.

ACUTE KIDNEY INJURY SECONDARY TO BILATERAL OCCLUSIVE MAIN RENAL ARTERY EMBOLI: A RARE PRESENTATION OF A-FIB

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TH Naber, A Kontamwar, M Sachdeva, MD Gitman.

Acute bilateral renal artery occlusion by emboli is a rare cause of acute kidney injury. Early diagnosis and intervention is crucial for favorable renal outcomes. Bilateral renal artery occlusive thrombo-emboli is seen more often after blunt trauma to the abdomen, antiphospholipid/ hypercoagulable syndrome, heparin induced thrombocytopenia, and paradoxical large emboli; but has been rarely reported as a clinical presentation of new onset atrial fibrillation.

We report a 69 year-old man without significant past medical history who was admitted with one day duration of bilateral flank pain, gross hematuria, and found to have new onset atrial fibrillation. LDH on admission was 2418U/L, CT-Angio (figure 1) was performed which confirmed the diagnosis with bilateral global renal hypo-perfusion and infarction. Urgent selective renal artery thrombolysis was performed by injecting r-TPA with restoration 80-90% of the renal blood flow. There was no evidence of renal artery stenosis or hypercoagulable state. Patient was started on anticoagulation, and his kidney function improved and his creatinine stabilized at 2mg/dl.

Because patients with renal artery emboli present with abdominal or flank pain that mimic other conditions, such as nephrolithiasis and pyelonephritis, the diagnosis is frequently missed or delayed. LDH, in the appropriate clinical setting, is strongly suggestive of renal infarction and should be always investigated. There are no reports comparing outcomes with untreated patients. In addition, the duration of complete renal arterial occlusion beyond which thrombolysis would no longer be of benefit is unknown. We reinforce clinicians to consider renal occlusive disease as part of their differential and to immediately intervene once the diagnosis is reached.

SPONTANEOUS PERINEPHRIC HEMATOMA

Vidya Nadig, Deven Patel, Rupesh Raina, Alan Lichtin, Bernard Silver, Robert Heyka.

Spontaneous perinephric hematoma (SPH) is rare and has been described in association with conditions such as angiomyolipomata, renal cell carcinoma, renal artery aneurysm and metastatic renal melanoma. However, SPH has not been described in association with primary amyloidosis. A 34 year old African American male presented to the ED with a sudden onset of left flank pain, radiating to the left testicle. Medical history was significant for hypertension since the age of 14. Spontaneous splenic capsular bleeding that required splenectomy occurred 2 months prior to current presentation. CT scan of the abdomen performed in the ED showed a large left perinephric hematoma. Left renal angiogram showed active extravasation from the lower pole branch of the left renal artery. Gelfoam embolization was performed with resolution of bleeding. Acute renal failure developed within one day of admission, due to renal artery embolization, perinephric hematoma and administration of contrast. It resolved rapidly over the ensuing 48 hours with supportive therapy. He also developed spontaneous bilateral subconjunctival hemorrhage on day two of his hospital stay. Admission laboratory results were significant for a slightly prolonged PTT. Further workup revealed low factor X levels. Urine protein electrophoresis showed an elevated abnormal homogeneous band in the lambda region. Bone marrow biopsy revealed plasmocytosis with an excess of lambda light chains and aberrant cyclin D expression. Stomach and duodenal biopsies confirmed amyloid deposits by Congo red staining. Spontaneous perinephric hematoma due to vascular angiopathy of amyloidosis is rare. However, when associated with abnormal coagulation studies or bleeding at other sites, amyloidosis should be considered because it has protean systemic manifestations and potentially can be cured with chemotherapy.

INCREASED SERUM ADIPONECTIN LEVELS WITH REDUCED EXPRESSION OF RENAL ADIPONECTIN RECEPTOR-1 IN ACUTE KIDNEY INJURY (AKI)

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Adiponectin is a hormone secreted by adipocytes that improves insulin sensitivity and possesses anti-inflammatory and anti-atherosclerotic properties. Previous studies have documented increased serum adiponectin levels in chronic kidney disease (CKD) patients, however, the potential effects of AKI on serum adiponectin levels and expression of renal adiponectin receptors has not been previously examined. Our previous studies demonstrated increased accumulation of neutral lipids in the proximal tubule during ischemia/reperfusion and cisplatin (CP)-mediated AKI. In the present study we measured the effects of CP on epididymal adipose tissue mass, serum adiponectin, and the expression of renal adiponectin receptor-1. We found that CP causes a significant reduction ($43\pm 5\%$) of epididymal fat tissue mass when compared to saline-treated mice. In addition, CP induced a 10-fold increase in serum adiponectin levels including low and high molecular weight forms. When we quantified protein levels of renal adiponectin receptor-1 we found that CP treatment was accompanied by a dramatic reduction (93%) in protein levels of renal adiponectin receptor-1 when compared to saline treated mice. Administration of fibrates on the other hand caused a 15% increase in protein levels of renal adiponectin receptor-1. Our studies showing loss of adipose tissue mass, as well as increased serum adiponectin levels with reduced expression of renal adiponectin receptor-1 during AKI, may represent a state of “resistance to adiponectin” as recently described in CKD patients. This phenomenon could be the result of adiponectin dysfunction or down regulation of adiponectin receptor-1 with a counter-regulatory increase in adiponectin secretion. In addition, our findings of fibrate-mediated increased expression of renal adiponectin receptor-1 may explain our observations of reduced renal lipotoxicity and amelioration of renal function by the use of fibrates during AKI.

SPONTANEOUS KIDNEY RUPTURE IN PREGNANCY DUE TO COCAINE INTOXICATION

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Spontaneous Kidney rupture is rare in pregnancy. Cocaine has multiple toxic effects on the kidney. We report a case of spontaneous rupture of the kidney pregnancy due to cocaine abuse. A 36-year-old woman at 18 weeks gestation presented to ED with sudden right flank abdominal pain unassociated with trauma, fever, dysuria nor frequency. She had a BP of 165/95, a hemoglobin of 6.7g/dL, BUN of 6mg/dl & creatinine of 1.0mg/dL. CT scan and renal US showed large right perinephric hematoma with positive urine drug screen for cocaine. She was transfused 4 units of blood, and conservatively managed due to pregnancy and absence of active bleeding on Doppler US. The patient left hospital against medical advice after 4 days, but returned again at 29 weeks gestation with ARF, preeclampsia, cocaine intoxication and intrauterine fetal demise. A cesarean section was done, and repeat US showed reduction of in size of subcapsular hematoma. Kidney rupture has been previously reported in a male cocaine user. Our case shows that cocaine use can cause kidney rupture, ARF and preeclampsia in pregnancy.

NONDILATED OBSTRUCTIVE UROPATHY – AN UNRECOGNIZED CAUSE OF ACUTE RENAL FAILURE IN HOSPITALIZED US PATIENTS: THREE CASE REPORTS SEEN OVER SIX MONTHS IN A NORTH-WESTERN WISCONSIN NEPHROLOGY PRACTICE.

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The syndrome of non-dilated obstructive uropathy (NDOU) in the causation of acute renal failure (ARF) is well reported. However, the impression in the literature is that this syndrome is a rare phenomenon.

We experienced three cases of NDOU causing symptomatic ARF, requiring HD in one patient, between March 2009 and October 2009. These experiences have caused us to reconsider the reality that this syndrome may not be that rare after all.

There were two males, one female. They all presented with recent symptomatic ARF. Renal imaging revealed no dilatation (Table).

Age /Sex	Peak Ser Creatinine (mg/dL)	New Ser Creatinine (mg/dL)	Cause of Non-dilatation	HD	Rx
56/ M	10.3	1.2	Metastatic vesical carcinoma	Yes	Ureteric stent + Left Perc Nephrostomy
59/ F	4.4	1.08	Metastatic uterine cervical carcinoma	No	Left Perc Nephrostomy
67/ M	4.0	1.8	Short –duration obstruction	No	Foley Catheter

We note the dramatic recovery of renal function following relief of obstruction in all 3. Only a very high index of suspicion, coupled with a very high level of collaboration between Nephrology, Urology and Interventional Radiology, would lead to more diagnosis of NDOU. We submit that NDOU may not be rare after all. Another learning point is that in ARF with dual kidneys, both kidneys must be assumed to be obstructed without prejudice to any radiological findings.

RECURRENT ACUTE TUBULAR NECROSIS – A RARE PRESENTATION OF HYPERCALCEMIA

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Renal disorders due to hypercalcemia most commonly present as impaired concentrating ability, decreased glomerular filtration rate (GFR), calcium deposition and diabetes insipidus. Acute tubular necrosis (ATN) is a rare manifestation of hypercalcemia. We present a case of hypercalcemia caused by excessive ingestion of calcium based antacids resulting in ATN twice over 8 years.

A 47 year old female with past medical history of hypertension, chronic pancreatitis and type 2 diabetes mellitus presented with complaints of generalized weakness, abdominal pain and confusion. Over the past several weeks, the patient ingested over 10 gm of calcium based antacids to relieve epigastric discomfort. On admission, the patient's physical examination was unremarkable. Blood pressure was 145/76, heart rate 80 with no orthostatic hypotension, temperature 96.7 F. Initial laboratory workup revealed blood urea nitrogen 30mg/dl, creatinine 3.95mg/dl, calcium 16.3mg/dl, parathyroid hormone 6 pg/ml. Urine studies revealed random sodium Na71mg/dl, creatinine 16.2mg/dl, osmolality 168 mosm, urine to plasma creatinine ratio was 8, fractional excretion of sodium 13%. Despite intravenous hydration, the patient required calcitonin for persistent hypercalcemia, which later resolved with improvement in renal function. A similar episode eight years ago required hemodialysis. Renal biopsy indicated ATN on both occasions.

ATN is a rare presentation of hypercalcemia. The mechanism of ATN from high calcium is likely from direct vasoconstriction of renal arterioles, polyurea and diabetes insipidus. The direct effect of hypercalcemia on renal tubules causing ATN is not clearly understood and minimal literature is available. The complications associated with ATN are often life threatening with in-hospital survival rate of approximately 50%. The recurrent co-existence of hypercalcemia and ATN in our patient stresses the importance of investigating for ATN in cases presenting with hypercalcemia and acute renal failure for early diagnosis, timely treatment and preventing complications.

RENAL FAILURE IN CRITICALLY ILL PATIENTS WITH INFLUENZA A (H1N1) INFECTION

Rupesh Raina, Sevag Demirjian, S.Navneethan, M. Schreiber and J. A. Guzman

In April 2009, CDC reported the first two cases in the United States of human infection with a novel influenza A (H1N1) virus. As of July 6, a total of 122 countries have reported 94,512 cases of novel influenza A (H1N1) virus infection, 429 of which were fatal; in the United States, a total of 33,902 cases were reported, 170 of which were fatal. Renal failure as part of MSOF (Multiple System Organ Failures) syndrome has been frequently observed, and was associated with poor outcome in Canada and Mexico. We describe the clinical course and risk factors for acute kidney injury (AKI) in critically ill patients with influenza A (H1N1) infection.

Methods: We performed a retrospective medical chart review of 11 patients with confirmed influenza A (H1N1) according to the CDC definitions admitted to the Cleveland Clinic MICU between 10/09-11/09. A riffles criterion was used to define and stratify the severity of AKI.

Results: Mean (\pm SD) age was 46 ± 2.5 years; 5 patients (45%) were females and 3 (27%) were ≤ 30 years of age. Nine (81%) patients had rhabdomyolysis, and 4 (36%) had a hypercoagulable disorder of unknown significance. Six patients (54%) were supported with RRT (Renal Replacement Therapy). The mean onset of severe AKI requiring RRT was 3.7 days after the ICU admission. Patients with AKI requiring RRT had higher incidence of pre-existing renal disease (mean baseline creatinine 2.85 vs. 1.03 mg/dl), higher peak creatinine (7.5 vs. 2.7 mg/dl.), higher serum creatinine kinase (CK) (3342 vs. 574 u/l), and APACHE II scores (21 – 25 vs. 11 - 15.). Hospital mortality was higher in patients with AKI requiring RRT (50% vs. 0%).

Conclusion: In our cohort pre-existing renal disease, hypercoagulable state, and elevated CK levels were associated with higher incidence of AKI. The latter was associated with higher hospital mortality.

ACUTE KIDNEY INJURY IN H1N1/ INFLUENZA A VIRUS INFECTION RELATED DEATHS IN FOUR ADULTS

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H1N1 Influenza A virus infection is a pandemic. Apparently young adults with H1N1 Influenza A virus infection are at a higher risk for mortality, compared to infection with usual seasonal influenza. We report clinical course and renal manifestations of 4 cases of H1N1/ Influenza A virus infection in young adult patients in table below.

	Patient 1	Patient 2	Patient 3	Patient 4
Age (years), sex	19, male	26,female	18, male	50,female
Race	White	White	White	Black
BMI	35.2	29.4	20	32.4
Co-morbidities	None	None	Asthma	HTN
Initial symptoms	C, F, S	C, H, D	M,C,F,D	M,C,F
RD, Intubation	Yes	Yes	Yes	Yes
IS to ICU	7 days	5 days	6 days	6 days
Pressor use	Yes	Yes	Yes	No
AKI	Yes	Yes	Yes	Yes
First Dialysis, hospital day	Day 3	Day 6	Day 6	None
IC	0.8	0.7	3.1	1.9
Peak CPK	41446	1832	49111	41177
Autopsy	Not done	Myocarditis, Renal- CN, Brain-AHL	Not done	Cardio-myopathy

(HTN- hypertension, C-cough, F-fever, H-headache, S-sore throat, D-diarrhea, M-myalgia, IS- initial symptoms, RD- respiratory distress, CPK- creatinine phosphokinase, CN- cast nephropathy, AHL- acute hemorrhagic leukoencephalopathy, IC- Initial Creatinine in mg/dl)

Our case series highlights the importance of recognizing rhabdomyolysis and AKI as important complications of H1N1/ Influenza A virus infection disease spectrum.

BIOPSY PROVEN ACUTE TUBULAR NECROSIS DUE TO VANCOMYCIN TOXICITY

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Controversy exists as to whether Vancomycin (VAN) can cause Acute Tubular Necrosis (ATN) in the absence of other nephrotoxins. Recent VAN dosing guideline changes have resulted in larger cumulative VAN doses. Here we present the case of ATN due to isolated VAN toxicity.

A 23 year old male with a history of neuroendocrine tumor, was admitted with fever attributed to cellulitis at the site of a central catheter. He was hemodynamically stable. The line was removed and the patient was treated with VAN. In the first 24 hours, he received 5 gm of VAN. On Day 2, he was anuric and serum creatinine (sCr) increased from 0.97 to 3.62mg/dl. Urinalysis was bland. On Day 4, VAN trough level was 64.7mg/L, and Scr was 9.96 mg/dl. A renal biopsy was performed and hemodialysis was initiated. Biopsy was consistent with moderate ATN.

Prior to 1960, impurities present in early formulations of VAN were thought to be responsible for nephrotoxicity. With purified preparations, VAN induced ATN has not been reported in an adult. With the utilization of higher doses of VAN, VAN alone should be considered as a potential nephrotoxin.

URINARY LIPOCALINS: NEW BIOMARKERS THAT PREDICT OUTCOMES IN PATIENTS WITH ACUTE KIDNEY INJURY (AKI)

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Previous studies have shown that urinary levels of lipocalins; Neutrophil Gelatinase-Associated Lipocalin (NGAL) and renal Fatty Acid Binding Protein-1 (FABP-1) represent sensitive and predictive early biomarkers of AKI after cardiac surgery (Kid Int 2008,73(4):465-472.). The role of urinary biomarkers in the prediction of adverse clinical outcomes in AKI has not been well studied. We evaluated prospectively a small cohort of hospitalized patients diagnosed with AKI. The objective of our ongoing study is to determine if urinary levels of NGAL and FABP-1 can predict adverse clinical outcomes judged by the need of dialysis or death. Urinary levels of NGAL and FABP-1 obtained on the first day of consultation were measured, corresponding to the initial rise of serum creatinine (sCr), by ELISA methods. The performance characteristics of urinary NGAL and FABP-1 in predicting each of the primary outcomes was described using the area under a receiver operator characteristic (ROC) curve and compared with the performance of more traditional clinical severity indices of kidney injury, including sCr, urine output, and APACHE II score. The sCr at enrollment was chosen to serve as a standard for comparison to test formally whether various models are statistically more or less discriminating. NGAL was both a sensitive and specific biomarker for dialysis and death; the areas under the ROC curves for these adverse outcomes were 0.80 and 0.89 respectively. FABP-1 was not a sensitive or specific biomarker for dialysis or death; the areas under the ROC curves for these adverse outcomes were 0.47 and 0.28 respectively, while ROC curves for creatinine were 0.58 for both outcomes. Although our data is from a small number of patients, our results are significant and suggest that the presence of elevated levels of NGAL on the first day of consultation in patients with AKI can predict adverse outcomes including the need for dialysis or increased mortality.

COMPARISON OF RIFLE AND AKIN CLASSIFICATION APPLIED TO ICU PATIENTS IN NEED OF RENAL REPLACEMENT THERAPY

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In recent years, two classification methods (RIFLE and AKIN) were proposed in attempt to formalize the definition of acute kidney injury (AKI) and describe its severity. Many studies validated both methods, but there's still no consensus on use of urine output (UO) and baseline creatinine (Cr) criteria. It is also unclear if either method can predict mortality in patients requiring renal replacement therapy (RRT). The objective of this study is to analyze the effects of applying the two classifications on mortality and renal recovery of critically ill patients in need of RRT.

We retrospectively analyzed the data that was prospectively collected for 200 patients admitted to the ICU and subsequently started on continuous RRT for AKI. Patients were stratified according to RIFLE and AKIN classifications with and without inclusion of 24 hour UO criterion. Standard logistic regression methods were used to account for selection effects. Mortality and renal recovery were assessed at both ICU and hospital discharge.

Overall patients' survival was 43% at ICU discharge and 38% at hospital discharge. There were 15, 26, 53% of patients in stages R, I, F, respectively, and 5% of patients that were classified as having no AKI, based solely on Cr. Adding UO criterion redistributed the patients to 4, 6, 89% in the same groups with 1% of patients remaining in non-AKI group. According to AKIN staging based on Cr only, 23, 15, 7% of patients were classified as Stages 1, 2, 3, respectively with 55% of patients in non-AKI group. Adding UO criterion redistributed the patients to 7, 2, 83% in the same groups with 8% of patients remaining in non-AKI group. With both AKIN and RIFLE classification methods, APACHE and Cr at RRT initiation were statistically significant mortality predictors at ICU and hospital discharge in our logistic regression model. In the same model, stages of AKIN and RIFLE were not found to predict patients' mortality or renal recovery.

UO dramatically changes distribution of patients within both AKIN and RIFLE staging schemes. AKIN staging seems to be less sensitive misclassifying 8% of patients as having no AKI even with the use of UO criterion. Neither scheme was shown to predict mortality or renal recovery in this group of patients.

RENAL INJURY IN CRITICALLY ILL PATIENTS INFECTED WITH PANDEMIC H1N1 INFLUENZA A

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Introduction: Pandemic pH1N1 Influenza A (pH1N1) has led to a global increase in severe respiratory illness. Little is known about renal outcomes in patients with pH1N1, and how these outcomes compare to non H1N1 respiratory virus infections.

Methods: 82 pH1N1 patients admitted to one of 7 intensive care units in Manitoba, Canada were prospectively followed. Outcomes were renal injury and renal failure as defined by RIFLE criteria or need for dialysis therapy and these were compared to a retrospective cohort of 24 non-pH1N1 respiratory viral infections.

Results: The pH1N1 group was composed of 82 critically ill pH1N1 patients with severe respiratory syndrome (n=41 confirmed cases, 4 probable, 37 suspected). Mortality was 13.3%. Renal injury, renal failure and the need for dialysis occurred in 52.9, 38.6 and 23.2% of pH1N1 patients respectively. Renal failure was associated with increased death (OR 7.818, CI 1.528-40.0), while renal injury, renal failure and dialysis were all associated with an increase in length of stay. In contrast, the 24 non-pH1N1 respiratory viral (RV) patients had higher average APACHE 2 scores (24.4 ± 7.5 vs. 19.0 ± 6.3 , $p=0.002$), and statistically higher rates of cancer, solid organ transplant, immunosuppressive medications and steroids use and chemotherapy. The RV group experienced much higher mortality (41.7 vs. 13.3%, $p=0.007$), but similar rates of renal failure. *Conclusion:* In critically ill s with pH1N1, renal injury and renal failure are common and associated with an increase in mortality and length of ICU stay.

SPONTANEOUS TUMOR LYSIS SYNDROME DUE TO A NEUROENDOCRINE TUMOR

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Aim: To describe a case of spontaneous tumor lysis syndrome due to a non-hematologic malignancy

Background: A 32-year-old Caucasian female who was 27 weeks pregnant presented to a tertiary care center after an ultrasound and laboratory tests revealed hepatomegaly with liver dysfunction. On admission, the patient had distressed fetal heart tones and emergent C-section was performed. Massive hepatomegaly was noted, which was biopsied intra-operatively. Patient was transferred to the ICU postoperatively. Patient developed Hyperkalemia and renal failure. Her potassium was 6.8 mEq/L and creatinine of 2.44 mg/dl from a baseline value of 1 mg/dl. Arterial blood gas revealed a pH of 7.05, PCO₂ of 49, PO₂ 65, and bicarbonate of 13. She was emergently dialyzed for Hyperkalemia. Her phosphorus was 8.1 mg/dl and her ionized calcium was 0.96 mg/dl. On post operative day one she became anuric and hypotensive. She was started on CVVH for renal support. Work-up for etiology of renal failure revealed serum uric acid of 24 mg/dl. The liver biopsy revealed metastatic neuroendocrine tumor. It was concluded her metabolic derangements were consistent with tumor lysis syndrome (TLS). Her overall condition worsened in the next few days, required ionotropes for blood pressure support, and developed multi-organ failure. She was not a candidate for any resection or chemotherapy. A family meeting was called and care was withdrawn.

Discussion: The majority of the TLS cases are reported to occur after aggressive chemotherapy for hematologic malignancies associated with large tumor burdens, such as Burkitt's lymphoma or acute leukemias. It is a rare complication of non-hematologic malignancy. Occasionally, patients develop spontaneous acute TLS, the mechanism of which is poorly understood. To the best of our knowledge, there is no prior report of spontaneous TLS due to a neuroendocrine tumor. The clinical syndrome is characterized by hyperuricemia, hyperphosphatemia, hypocalcemia, Hyperkalemia, and ensuing acute renal failure. Treatment of established tumor lysis syndrome consists of vigorous hydration and early hemodialysis in severe cases.

ROLE OF PREOPERATIVE ANGIOTENSIN CONVERTING ENZYME INHIBITORS AND ANGIOTENSIN RECEPTOR BLOCKERS IN ACUTE KIDNEY INJURY AFTER SURGERY

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Acute kidney injury (AKI) is a known post operative complication. Only a few studies have looked at the role of ACE inhibitors or angiotensin receptor blockers (ACE-I/ARB) in AKI after surgery. These studies were limited to cardiac surgeries. Most importantly they demonstrated conflicting results. No prior study has ever been done in all-type surgery patients. The purpose of this research was to examine the role of preoperative ACE-I/ARB in AKI after cardiac and non-cardiac surgery.

This was an observational study in which the medical records of 333 patients were reviewed retrospectively. We defined AKI as an increase in serum creatinin (Cr) of greater than 0.3 mg/dl or need for hemodialysis after surgery. We included any surgical patient who was given general anesthesia and stayed at the hospital for at least 2 days. ESRD and pediatric cases were excluded.

Mean preoperative and postoperative creatinin were 0.93 mg/dl and 1.17 mg/dl respectively. This shows a significant increase in serum creatinin after surgery ($p<0.000$).

Comparison of mean increase of creatinin based on prior treatment with ACE-I/ARB did not show any significant difference (0.24 mg/dl vs. 0.25 mg/dl, $p=0.91$). However mean increase in creatinin was significantly higher in

cardiac surgeries than in all other types of surgery (0.51mg/dl vs. 0.18 mg/dl, $p=0.007$).

Postoperative AKI happened in 69 patients (21.5%). Of all AKI patients, 27 (39.1%) were receiving ACE-I/ARB before surgery. The correlation between taking ACE-I and post-operative AKI was not significant (Chi-square= 1.329, $P=0.249$). Incidence of postoperative AKI was significantly higher in cardiac surgery patients (40.7% vs. 17.2%, $p<0.000$).

This study shows that over all there is a significant increase of creatinin after surgery. This increase is more remarkable in cardiac surgery patients. Cardiac surgery patients also had a significantly higher rate of AKI. Our data did not show a significant relationship between ACE-I/ARB before surgery and postoperative AKI. However, since patients on ACE-I/ARB had a higher rate of AKI (25.2% vs. 19.6%) we suggest to change the current standard of taking ACE-I/ARB before surgery. We plan to extend this study to include a larger sample size to obtain more conclusive results.

**A SIMPLIFIED CASE-MIX ADJUSTER MODEL FOR THE
PROPOSED END STAGE RENAL DISEASE (ESRD)
PROSPECTIVE PAYMENT SYSTEM (PPS)**

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In September 2009, the Centers for Medicare & Medicaid Services (CMS) released a proposed rule expanding the PPS for ESRD outpatient dialysis services, bundling payments for the current PPS items, separately billable medications and select lab services beginning 1/1/2011. This system would apply adjusters for wage index, patient-specific demographic and comorbidity factors to a base rate to calculate a per-patient, per-treatment payment. A potential issue with the currently selected case mix adjusters in the CMS proposed regression equation is that providers may not consistently have the ability to collect or accurately report all of these variables. Therefore we assessed the ability of an alternative case mix model with fewer more easily captured variables to predict ESRD resource utilization.

Data analysis was limited to separately billables. Utilizing USRDS data for CY 2006, models with a reduced number of case mix adjusters were designed. Overall, R^2 for separately billables with these simple models ranged from 4.3% to 6.9% without hospitalization and 10% to 10.8% with hospitalization. Data construction and analysis were performed by the Lewin Group.

	Model 1	Model 2	Model 3	Model 4
Age, race, gender, BSA, BMI	X	X	X	X
Dialysis duration < 4 months	X	X	X	X
Septicemia/shock Bacterial pneumonia, opportunistic infections		X		X
Hospital admission in last month			X	X
R^2	4.3%	6.9%	10.0%	10.8%

*PPS R^2 for separately billables = 8.7%

A simpler case-mix model, which would impose less of a burden on providers, has similar ability to predict resource utilization as compared to CMS' proposed model.

ALTERNATIVE HIGHER PREDICTIVE MODEL FOR THE END STAGE RENAL DISEASE (ESRD) PROSPECTIVE PAYMENT SYSTEM (PPS)

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The Centers for Medicare & Medicaid Services (CMS) has proposed an expanded outpatient dialysis PPS. Payment will be modified by patient-specific case-mix adjustment, which is based on a multiple regression analysis with 18 factors, including age, BMI, and specified co-morbidities. The ability of these factors to predict variation in resource utilization for separately billables is limited: they predict 8.7% of the variance of Medicare Allowable Charges (MAC). We evaluated additional variables to build a model with higher predictive power of cost in the care of outpatient dialysis patients.

USRDS 2006 data were used to construct patient-month observations for dialysis patients aged ≥ 18 . Variables included information from the medical evidence form, claims-based indicators for select co-morbidities and dialysis duration. Comorbidities were constructed with look-back periods and code definitions per the CMS proposed rule. Analyses were performed with log-linear models in SAS V8. The Lewin Group performed data analysis.

Alternative model-PPS variables included:	Age, Gender, BSA, BMI, Duration of RRT < 4m, Shock/Septicemia, GI tract bleed, Cancer, MDS
PPS variables excluded:	Alcohol/drug dependence, Cardiac arrest, Pericarditis, HIV/AIDS, Hepatitis B, Bacterial pneumonia & other opportunistic infections, Hereditary anemias, Monoclonal gammopathy
Variables included in alternative model proposed here:	Race/Ethnicity, Duration of RRT ≥ 4 -12m, Other Cardiac Illness, Vascular Access Procedure, Hospital Admission in Last Month

By selectively including some of the variables identified by CMS and incorporating additional factors, we built an alternative model with an R^2 for separately billables of 13%. This model has higher R^2 while using fewer case mix adjusters, thereby attempting to manage the balance between higher explanatory power and data collection burden imposed upon facilities. However, the feasibility of fully capturing patient data for the new variables proposed is undetermined.

**IMPACT OF REDUCTIONS IN INFECTIOUS COMPLICATIONS,
WITH THE USE OF THE HERO VASCULAR ACCESS DEVICE,
ON DIALYSIS PROVIDER REVENUE IN AN ERA OF BUNDLING**

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The purpose of this abstract is to report projected revenue savings by the dialysis provider when hospitalization secondary to catheter infections is decreased. It is widely recognized that the use of chronic hemodialysis catheters for vascular access is associated with increased morbidity, mortality, and costs. Hospitalization for catheter-related bacteremias is a major contributor to this increased cost of care. Missed dialysis sessions resulting from hospitalization for catheter-related bacteremias can also negatively impact dialysis provider revenue. The HeRO vascular access device is an FDA approved 6 mm ePTFE graft, which attaches via a titanium connector to a 5 mm outflow component comprised of silicone with nitinol reinforcement designed to traverse central venous stenosis by routing blood flow to the right atrium via a major central vein. The clinical trial demonstrated a reduction in infectious complications with the HeRO device vs. HD catheters in patients who are not candidates for an AV fistula or graft.

The method used to project the economic impact associated with the reduction of infectious complications with the HeRO device for the dialysis provider with the anticipated introduction of bundling in 2011, was the development of a model which allows for the input of variable clinical and economic data which projects the effect of missed dialysis sessions on dialysis provider revenue.

The results modeled were based on data available from CMS, USRDS, ESRD CPM, and published information. These projections indicate that the use of the hemodialysis catheters would result in:

Infections	Hospital Days	Missed Dialysis Treatments
Catheter-related	200,193.8	86,750.5
HeRO-related	60,928.5	26,402.4

In conclusion, economic modeling projects that the dialysis provider would generate, in addition to the savings realized through the reduction of facility antibiotic and thrombolytic use, total incremental revenue of **\$11,987,578.82 (\$637.82 per patient)** when using HeRO vs. catheters in patients who have exhausted all fistula and graft sites.

FAILURE TO REPLICATE CMS CASE MIX ADJUSTERS AND THE POTENTIAL IMPACT ON DIALYSIS REIMBURSEMENT

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The proposed Medicare prospective payment system for dialysis includes 18 case mix adjusters (CMA). These adjusters were based on regression analyses in which dialysis reimbursement was the outcome variable and patient-level medical characteristics and co-morbid conditions from the 2728 Medical Evidence forms and paid Medicare claims were the predictors. CMS reduced the dialysis base payment by 22% to offset these CMAs, and published the mean CMA for all U.S. dialysis facilities. The purpose of this study was to replicate the CMS case mix adjuster analysis in DaVita facilities. We randomly selected 4 facilities in 27 regions, resulting in a sample of 108 geographically diverse facilities. We conducted chart reviews at each of these facilities for patients with Medicare as primary payer receiving dialysis between October 1, 2008 and September 30, 2009. Fixed CMAs (age, gender, initiation of dialysis) were coded once at baseline; all others were coded monthly. Data sources included electronic medical records, hospital discharge summaries, paper charts, healthcare professional notes, and discussions with on-site healthcare professionals. After excluding centers with incomplete data, final sample included 100 clinics and 7,340 patients. DaVita facilities were lower on 8 out of the 12 co-morbidities: Hepatitis B, Septicemia, Cancer, HIV/AIDS, Hemolytic or sickle cell anemia, Monoclonal Gammopathy, Myelodysplastic Syndrome, and Pericarditis. The magnitude of the absolute differences were large in many cases: -7.0% for Hepatitis B; -6.7% for Septicemia; -4.4% for HIV/AIDS. DaVita reported higher prevalence of four co-morbidities (Cardiac Arrest, Pneumonia/Other OI, Alcohol-Drug Dependence, GI Bleeds), but in each case the difference was <2.0%. The average CMA for the 100 facilities was 1.21 versus 1.28 reported by CMS for these same facilities. The inability to replicate CMS CMAs would result in a 7% decrease in payment. In conclusion, the currently proposed system may inadvertently reduce dialysis payments as a result of the inability of clinics to replicate the case mix adjusters.

POTENTIAL FOR RACIAL DISPARITIES IN THE PROPOSED MEDICARE DIALYSIS PROSPECTIVE PAYMENT SYSTEM

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Numerous studies have identified racial disparities in health care resource use and clinical outcomes in U.S. dialysis patients. In the United States, blacks compared to whites require higher doses of epoetin alfa to achieve equivalent hemoglobin outcomes and higher vitamin D to achieve comparable PTH, and have lower rates of AV fistulas, yet paradoxically have lower mortality following initiation of dialysis. CMS considered but rejected the inclusion of a racial case mix adjuster (CMA) in the proposed prospective payment system for dialysis. We used geomapping and spatial analytics to explore the impact of the current system on dialysis facilities serving disproportionate numbers of blacks. Race was determined in two ways: we used U.S. census data to code the proportion of blacks at a zip code and county level, and also used DaVita data to code percentage of blacks served within individual dialysis facilities (only among DaVita facilities). We then used the CMS public access file to determine facilities whose payments would be reduced by more than 10% under the new payment system by subtracting the CMS-projected payments under the current system from the projected payment should the entire unit opt into the new payment system in 2011. Mapping of these facilities produced clear patterns, in which facilities either in areas with high percentages of blacks, or serving a high percentage of black patients, are more likely to experience significant decreases in payment under the proposed prospective payment system. Areas in the southeast, and urban areas throughout the country, seem to be most affected. We explore the impact of including a racial case mix adjuster on geographic disparities, in an attempt to create an adjuster that minimizes geographic biases in reimbursement. The data demonstrate the importance of race in the provision of dialysis services in the U.S., and the need to include race as a CMA to adjust for inequities with both biologic and social underpinnings.

DEPRESSION PREDICTS OUTCOMES IN DIABETICS BUT NOT NON-DIABETICS WITH CKD

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Depression is more prevalent in diabetic patients than the general population and its relief is associated with improved glycemic control. Depression is also associated with poor outcomes in CKD patients. We investigated the association of a major depressive episode assessed at enrollment with a composite outcome of ESRD, hospitalization or death at 1 year in diabetic vs. non-diabetic CKD subjects at a VA Medical Center. Subjects had stage 2-5 CKD but were not yet on dialysis.

Of 266 subjects, 55.6% were diabetic and 44.4% were not. Depression was more prevalent in diabetics than non-diabetics, 26.4% vs. 14.4%, p 0.02. In diabetics, composite outcomes were more likely if depression was present than if absent (68.4% vs. 48.2%, p 0.03), but this association was not significantly different in non-diabetics with or without depression (50.0% vs. 41.8%, p 0.54). Depression was associated with a 2-fold increase in the odds of a composite outcome in diabetics in multivariable logistic regression models (**table**), but not in non-diabetics. Depression may contribute to factors such as poor medical adherence and lead to worse

Adjusted odds of depression for the composite event		
Variable	OR (95% CI)	C-statistic
Depression only	2.33 (1.07, 5.10)	0.578
+ Age and race	2.33 (1.07, 5.10)	0.578
+ CKD stage	2.86 (1.22, 6.70)	0.712
+ CV disease	2.93 (1.25, 6.86)	0.718
+ Comorbidities	2.88 (1.20, 6.91)	0.749
+ Serum albumin	2.80 (1.08, 7.28)	0.823
+ Hemoglobin	2.92 (1.08, 7.89)	0.835
+ Ca x Pi product	2.84 (1.09, 7.44)	0.830

outcomes in CKD diabetic patients. Future randomized studies are needed to explore whether treatment of depression will improve outcomes of diabetics with CKD.

ACCESS TO HEALTH CARE IN ADULTS EVALUATED FOR CHRONIC KIDNEY DISEASE: FINDINGS FROM THE KIDNEY EARLY EVALUATION PROGRAM

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Difficulty in access to health care can contribute to suboptimal blood pressure and poor health outcomes; however, little is known about such barriers in chronic kidney disease (CKD) care. We assessed access to health care by CKD status among participants of the Kidney Early Evaluation Program, a nationwide health screening program of adults at high risk for CKD from 2000-2007 (n=76,942). Participants responded to a questionnaire survey and had laboratory testing, with CKD defined as estimated glomerular filtration rate $<60\text{ml/min/1.73m}^2$ or random spot urine albumin:creatinine ratio $>30\text{mg/g}$. There were 20,094 (26.1%) subjects with CKD. CKD subjects were more likely to be older, female, Caucasian and had lower educational status (less than high school) and greater burden of cardiovascular disease as compared to subjects without CKD. Significant differences ($p<0.0001$) by CKD status existed in health care utilization i.e. last physician visit within 1 year (91.9% vs 84.9% in CKD and non-CKD subjects respectively), difficulty in getting medical care (19.0% vs. 21.5%), presence of health insurance (85.3% vs. 80.1%), having a generalist source of care (81.9% vs 78.8%), and having insurance benefit to pay for medications (78.4% vs 75.5%). Among subjects with health insurance (n=60,437), the insurance type was public in 38.3% and private in 47.4%. Multivariable analysis with adjustment for age, race, and cardiovascular disease revealed that presence of CKD was associated only with low educational status (odds ratio, OR=1.19, $p=0.0181$) and presence of public health insurance as compared to private insurance (OR=1.50, $p<0.0001$). Despite CKD subjects reporting no greater perceived difficulty in health care access as compared to subjects without CKD, low educational status and public health insurance were associated with CKD. In adults at risk for CKD, patient factors (socioeconomic status, awareness, adherence to treatment), insurance payer status and quality of physician services (availability, appropriate knowledge and attitudes) may be more important contributors to the quality of CKD care than utilization of health care delivery system.

SILENT MYOCARDIAL ISCHEMIA AND CHRONIC KIDNEY DISEASE: THE FATAL DANCE

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The objective of this study was to determine the relationship between silent myocardial ischemia, glomerular filtration rate (GFR) and mortality in patients with coronary artery disease (CAD) patients.

This study enrolled 346 patients who had undergone percutaneous coronary intervention (PCI). Silent myocardial ischemia (SMI) was defined as the absence of chest pain in response to balloon dilatation of the affected vessel during PCI. GFR was estimated using the MDRD formula. Patients were categorized as having normal renal function ($\text{GFR} \geq 90 \text{ ml/min}$), mild renal dysfunction ($\text{GFR} 60 \text{ to } 89 \text{ ml/min}$), moderate dysfunction ($\text{GFR} 30 \text{ to } 59 \text{ ml/min}$), and severe dysfunction ($\text{GFR} \leq 29 \text{ ml/min}$).

This study included 64.2 % men, 31.2 % current smokers, and 35.3 % diabetics, with mean age $64.5 \pm 12 \text{ yr}$ (mean \pm SD), LDL cholesterol $101 \pm 34 \text{ mg/dL}$, blood pressure $141 \pm 26 / 76 \pm 14 \text{ mmHg}$, and serum creatinine $1.14 \pm 0.85 \text{ mg/dL}$. Cumulative 10-yr survival rates decreased in a graded fashion from 80% for those with normal renal function to 25% for those with $\text{GFR} \leq 29 \text{ ml/min}$. Compared with patients with normal renal function, the multivariable adjusted hazard ratios for all-cause mortality among patients with mild, moderate, and severe renal impairment were 1.11 (95% confidence interval [CI], 0.60–2.07), 1.75 (95% CI, 0.94–3.28), and 2.79 (95% CI, 1.16–6.77), respectively. Furthermore, patients with both $\text{GFR} \leq 60 \text{ ml/min}$ and SMI had a worse survival compared to those with $\text{GFR} > 60$ and had no SMI.

Renal function is a graded and independent predictor of long-term mortality in patients with CAD. SMI is associated with poor survival in patients with compromised GFR. These findings may underlie the increased sudden cardiac death in patients with renal dysfunction.

PROGRESSIVE SECONDARY POLYCYTHEMIA IN A PATIENT ON CHRONIC HEMODIALYSIS

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It is extremely rare for patients with end stage renal disease (ESRD) to develop secondary polycythemia in absence of polycystic kidney disease or renal cell carcinoma.

We are reporting a rare case of a patient with ESRD who developed acquired polycythemia with high erythropoietin and small atrophied kidneys.

A 50 year old non-smoker male, resident of Cleveland Ohio (569 feet above sea level) with ESRD on hemodialysis for the last 7 years was evaluated for polycythemia. The patient was started on recombinant human erythropoietin (Epo) at the beginning of hemodialysis due to anemia. After 4 years of initiating hemodialysis and Epo treatment, the medication was discontinued due to progressive increase in patient's hemoglobin level reaching 19gm/dl. After 3 years of discontinuation of Epo treatment, the patient continued to have high Hb of 19g/dl +/- 1gm/dl. Initial assessment included: history, physical examination, blood work, and imaging studies. Review of patient's history revealed multiple access failure with AV fistula thrombosis in the last 2 years. The physical exam was not suggestive of lung or heart diseases and showed normal neurological exam. The blood work showed normal Oxygen saturation and erythropoietin serum level of 230mIU/mL (normal level 4-24mIU/mL). The imaging studies included CT chest, abdomen and pelvis which revealed small atrophied kidneys, normal liver and otherwise normal findings. At this point, the treatment plan includes serial phlebotomies; aspirin therapy and close follow up.

Erythropoietin is a glycoprotein that is normally produced by peritubular fibroblasts of the renal cortex in response to tissue hypoxia. Cellular sites of extrarenal erythropoietin production include hepatocytes, which have a minor contribution to serum level of erythropoietin. Recently it was found that brain cells have the ability to produce small amount of erythropoietin in response to local hypoxia.

The case we are reporting raises the question of possible other extrarenal sources of erythropoietin that is yet to be identified.

**DOSING TRENDS OVER TIME OF EPOETIN ALFA
UTILIZATION IN CHRONIC KIDNEY DISEASE PATIENTS
NOT ON DIALYSIS: A PHARMACY BENEFIT PERSPECTIVE**

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This study evaluated epoetin alfa (EPO) dosing trends over time in patients with CKD not on dialysis receiving EPO through the pharmacy benefit.

An analysis of pharmacy claims from the Ingenix IMPACT (07/2002-03/2009) and the Pharmetrics (01/2003-03/2009) databases was conducted. Patients aged ≥ 18 years old newly initiated on EPO with ≥ 1 EPO pharmacy claim and ≥ 1 diagnosis of CKD within 90 days prior to their first EPO prescription were included. Patients receiving EPO in the medical setting, treated with darbepoetin alfa, diagnosed with cancer, receiving chemotherapy or dialysis were excluded. Average weekly EPO dose, weighted by the duration of treatment, was calculated and evaluated using 6-month intervals (S1: January to June; S2: July to December) according to the date of treatment initiation to assess EPO dosing trends over time. Adjusted analyses were also conducted.

A total of 7,903 EPO-treated patients were identified from both databases (IMPACT: 4,471; Pharmetrics: 3,432). The semi-annual mean weekly EPO dose decreased in both database analyses. In the IMPACT analysis evaluating EPO dosing for treatment initiated from 2002 to 2008, the weighted mean weekly EPO dose declined from 17,053 (2002 S2) to 14,237 Units (2008 S2). In the Pharmetrics analysis evaluating EPO dosing for treatment initiated from 2003 to 2008, the weighted mean weekly EPO dose declined from 16,554 (2003 S1) to 15,342 Units (2008 S2). Similar results were obtained after adjusting for potential confounders.

Based on 2 large commercial healthcare claims databases, this analysis of claims data from 7,903 CKD patients not on dialysis receiving EPO through pharmacy benefits reported a decline over time in the mean weekly dose of EPO.

DRUG UTILIZATION PATTERNS AND COSTS FOR ERYTHROPOIESIS-STIMULATING AGENTS IN ADULT PATIENTS WITH CHRONIC KIDNEY DISEASE

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This study evaluated recent utilization patterns and costs for epoetin alfa (EPO) and darbepoetin alfa (DARB), two erythropoiesis-stimulating agents (ESAs), in adult patients with chronic kidney disease (CKD) not on dialysis.

Medical claims from the Ingenix Impact database (01/2006-03/2009) were analyzed. Patients included in the study were ≥ 18 years, had ≥ 1 claim for CKD, were newly initiated on EPO or DARB, and received ≥ 2 doses. Patients diagnosed with cancer, receiving chemotherapy or dialysis, or treated with both agents were excluded. Drug cost (based on October 2009 wholesale acquisition cost: EPO \$14.44/1,000 Units; DARB \$4.94/mcg) and dose ratio (Units EPO: mcg DARB) were calculated using mean cumulative ESA dose. Weighted mean weekly dose was also reported. Stratified analyses by payer type (HMO and PPO) were conducted as well as adjusted cost analysis.

A total of 1,680 patients (EPO: 991; DARB: 689) formed the study population. The EPO and DARB groups were similar with regard to age (years, EPO 63.9, DARB 63.2; $P=.309$), gender (women: EPO 49.2%, DARB 53.6%, $P=.082$), and treatment duration (days, EPO: 100 days; DARB: 99; $P=.775$). The mean weighted weekly dose (SD) was 11,486 (11,020) Units for EPO and 49 (40) mcg for DARB. The mean cumulative dose (SD) was 164,786 (175,453) Units for EPO and 694 (690) mcg for DARB, resulting in a dose ratio of 237:1. Based on the observed cumulative ESA doses, drug cost was 44% higher for DARB than for EPO (EPO \$2,380, DARB \$3,427; $P<.001$). Stratified analysis by payer type revealed similar results (HMO: \$2,451 for EPO vs. \$3,980 for DARB, $P<.001$; PPO: \$2,676 for EPO vs. \$2,983 for DARB, $P=.2623$). Adjusted cost results remained similar.

This observational study of 1,680 CKD patients not on dialysis reported 44% higher drug cost for DARB compared to EPO and a dose ratio of 237:1.

EPOETIN ALFA DOSING TREND OVER TIME IN ADULT PATIENTS WITH CHRONIC KIDNEY DISEASE: A MEDICAL BENEFIT PERSPECTIVE

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This study evaluated recent epoetin alfa (EPO) dosing trend over time in adult patients with chronic kidney disease (CKD) not on dialysis.

Medical claims from the Ingenix Impact database (01/2006-03/2009) were analyzed. Patients included in the study were ≥ 18 years, had ≥ 1 claim for CKD, received ≥ 2 EPO doses, and were newly initiated on EPO. Patients diagnosed with cancer, receiving chemotherapy or dialysis, or receiving another erythropoiesis-stimulating agent were excluded. Patients treated under the pharmacy benefit were also excluded. Mean weekly EPO dose was calculated for the overall observation period. To evaluate EPO dosing trends over time, the mean weekly EPO dose was calculated in the prevalent EPO treated CKD population by quarter of treatment.

A total of 991 EPO treated patients with CKD formed the study population. The mean (SD) weekly EPO dose over the study period was 11,486 (11,020) Units. The mean weekly dose by quarter in the prevalent EPO population declined from 12,740 units in 1Q2006 to 8,790 Units in 1Q2009.

This observational study of EPO treated patients with CKD not receiving dialysis reported a decreasing trend in the mean weekly EPO dose from 1Q2006 to 1Q2009.

EPOETIN ALFA (EPO) UTILIZATION TRENDS IN MEDICARE PATIENTS WITH CHRONIC KIDNEY DISEASE (CKD) NOT ON DIALYSIS

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The objective of this analysis was to evaluate EPO drug utilization from 2005-2007 in Medicare patients with CKD treated in the outpatient institutional setting.

An analysis of longitudinal medical claims using the Medicare 100% Institutional Database was performed to evaluate EPO dosing trends from 2005-2007. Patients included had ≥ 1 CKD diagnosis, were EPO treatment naive, and received ≥ 2 EPO doses during a treatment episode. Patients diagnosed with cancer, myelodysplastic syndrome, receiving chemotherapy, or treated with darbepoetin alfa were excluded. If a patient received dialysis, data were censored for the quarter prior to the first dialysis. A treatment episode was defined as the time from the first EPO dose to the last EPO dose. Patients with 2 consecutive EPO doses that were ≥ 1 quarter apart were excluded from the analysis. Treatment episodes were classified according the quarter of treatment initiation. Treatment episode characteristics evaluated were: treatment duration, cumulative EPO dose, and mean weekly EPO dose.

A total of 18,767 treatment episodes were identified between 2005Q2 and 2007Q4. Age distribution was 11.6% < 65 and 88.4 % ≥ 65 . Females composed 58.0% of the study population. A diabetes diagnosis was present in 59.0% and a hypertension diagnosis was present in 68.5%. Mean (SD) treatment duration was 3.3(2.4) quarters. Mean (SD) cumulative EPO dose per treatment episode was 312,000 (450,300). Mean (SD) weekly EPO dose was 6,600 (6,100) units and was stable during the observation period.

This study of Medicare patients with CKD not on dialysis treated with EPO observed a mean (SD) weekly EPO dose of 6,600 (6,100) units. The mean weekly dose remained stable over the observation period of 2005Q2 to 2007Q4.

RECENT ERYTHROPOIESIS STIMULATING AGENT (ESA) UTILIZATION AND COSTS IN MEDICARE PATIENTS WITH CHRONIC KIDNEY DISEASE (CKD)

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The objective of this analysis was to evaluate recent epoetin alfa (EPO) and darbepoetin alfa (DARB) drug utilization in Medicare patients with CKD not on dialysis treated in the outpatient setting.

An analysis of medical claims using the Medicare 100% Institutional Database was performed to evaluate EPO and DARB use from 2005-2007. Patients included had ≥ 1 CKD diagnosis, were ESA treatment naïve, and received ≥ 2 EPO or DARB doses during a treatment episode. Patients diagnosed with cancer, myelodysplastic syndrome, receiving chemotherapy, or treated with both agents were excluded. If a patient received dialysis, data were censored for the quarter prior to the 1st dialysis. A treatment episode was defined as the time from the 1st ESA dose to the last ESA dose. Patients with 2 consecutive ESA doses that were ≥ 1 quarter apart were excluded from the analysis. Mean cumulative ESA dose was used to calculate drug costs using April 2009 wholesale acquisition unit costs (EPO \$14.44/1,000 units; DARB \$5.064/mcg).

The study population consisted of 18,767 EPO and 16,574 DARB treatment episodes between 2005Q2 and 2007Q4. Age group distributions and proportion of females were similar between groups. The EPO group had a lower mean (SD) Charlson Comorbidity Score [3.5(1.6) EPO vs. 3.6 (1.6) DARB, $P<.001$]. Mean (SD) treatment duration was 3.3 (2.4) quarters for EPO and 3.4 (2.4) quarters for DARB ($P=.017$). The mean (SD) cumulative dose was 312,000 (450,300) units for EPO and 1,170.5 (1,493.3) for DARB, resulting in a dose ratio of 267:1 (EPO units: DARB mcg). Based on cumulative ESA doses, drug cost was \$1422 (32%) higher ($P<.001$) for DARB than for EPO.

This study of Medicare CKD patients not on dialysis observed 32% higher drug costs for DARB vs. EPO and a dose ratio of 267:1.

HEMOGLOBIN DECLINE FOLLOWING HEMATIDE™ DOSE INTERRUPTION

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CKD patients have a shortened RBC life span of ~60-90 d (Nhan, 2007). In published studies, the rate of Hb decline after ESA dose interruption (I) appears to be independent of the ESA used (Locatelli, 2001; Barany, 2007) in both hemodialysis (HD) and CKD-nondialysis (ND) patients. The rates of Hb decline in these patients following ESA interruption were similar across various ESA groups (Locatelli, 2001; Barany, 2007), despite differences in ESA half-lives and pharmacokinetics. The median time to reinitiation (RI) was 7 wk for darbepoetin and 9 wk for epoetin (Locatelli, 2001). Hematide™ is a synthetic, peptidic ESA linked to polyethylene glycol that is in phase 3 development for the treatment of anemia associated with CKD. The purpose of current analysis is to characterize Hb decline over time following Hematide dose interruption.

In this unplanned analysis of 2 ongoing, open-label, long-term phase 2 extension studies in 51 CKD-ND and 100 HD pts, dose interruptions for Hb levels above 13.5 g/dL were evaluated. Median Hb at dose interruption and median time from dose interruption (I) to dose reinitiation (RI) were determined.

Results are shown below with median values for Hb. Median time to reinitiate dose in both CKD-ND and HD patients was 4.0 wk (ranges, 1.1-15.1 wk and 1.1-16.7 wk, respectively).

CKD Stage	Patient Months	I, number (n)	Median Hb at I, g/dL	Median Hb at RI, g/dL	Hb Decline, g/dL/wk
3-5	1213	30 (17)	13.7	12.1	0.40
HD	2245	53 (38)	13.8	12.3	0.38

The rate of Hb decline following Hematide dose interruption appears to be similar to that found for other ESAs, and the rate of decline did not differ between CKD-ND and HD patients.

A SINGLE DOSE STUDY OF DENOSUMAB IN PATIENTS WITH VARIOUS DEGREES OF RENAL IMPAIRMENT

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Denosumab, a fully human monoclonal antibody, binds to and neutralizes the activity of human RANKL, a key mediator of osteoclast formation, function, and survival. The objective of this phase 1 study was to evaluate the pharmacokinetics (PK), pharmacodynamics (PD), and safety of a single 60 mg SC dose of denosumab in subjects with various degrees of renal function.

Subjects were assigned to a renal function group based on CrCL as calculated by the Cockcroft-Gault equation. Blood samples were collected for PK, PD, and safety analyses at specified time points from pre-dose through day 113 after receiving denosumab.

Fifty-five subjects were enrolled (12 normal renal function, 13 mild CKD, 13 moderate CKD, 9 severe CKD, and 8 ESRD). Parametric and nonparametric analyses indicated no significant relationship between renal function and denosumab PK parameters. Denosumab treatment resulted in rapid decreases in serum type I C-telopeptide (CTXI) in all groups, which was sustained from the first observation at day 2 through the end of study. Median percent decreases in CTXI were generally similar across all groups. The incidence of adverse events reported in this study were generally similar to those reported in other denosumab studies. However, consistent with the antiresorptive mechanism, transient decreases in serum calcium were observed in subjects with severe CKD and ESRD. As a result, it is important to ensure that patients with impaired renal function, particularly those with severe CKD or ESRD, are adequately supplemented with calcium and vitamin D.

Results of this study indicate that renal impairment does not affect the PK or PD of denosumab and therefore no dose adjustment is required when denosumab is administered to patients with impaired renal function.

RACIAL DIFFERENCES IN KIDNEY FUNCTION AMONG INDIVIDUALS WITH OBESITY AND METABOLIC SYNDROME: RESULTS FROM THE KIDNEY EARLY EVALUATION PROGRAM (KEEP)

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Obesity and metabolic syndrome may differ by race. We examined whether African American (AA) and white NKF-KEEP participants with obesity and metabolic syndrome differ regarding albuminuria, eGFR, and anemia and bone/mineral metabolism derangements in CKD. Univariate and multivariate logistic regression analyses were performed to evaluate associations of race with kidney outcomes.

Of 37,107 obese participants, 48% were AA and 52% white. Whites were more likely to have metabolic syndrome components – HTN (87.1% vs. 84.8%), dyslipidemia (81.6% vs. 66.7%), DM (42.7% vs. 34.9%) – and more profoundly reduced eGFR than AAs (CKD stages 3-5 prevalence 23.6% vs. 13.0%). AAs were more likely to have microalbuminuria (OR 1.60, 95% CI 1.45-1.76), macroalbuminuria (1.61, 1.23-2.12) and CKD stages 1-2 (1.54, 1.38-1.72). Among participants with CKD stages 3-5, anemia prevalence was 32.4% in African Americans and 14.1% in whites; corresponding values for secondary hyperparathyroidism were 66.2% and 46.6%, respectively.

Obesity and metabolic syndrome may be heterogeneous disease states among AAs and whites, possibly explaining differences in long-term kidney and cardiovascular outcomes.

A QUALITY IMPROVEMENT PROJECT TO IMPLEMENT K/DOQI GUIDELINES AT THE INTERNAL MEDICINE CLINIC

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McLaren Regional Medical Center, Flint, MI. Background: Chronic kidney

disease (CKD) is a worldwide public health problem affecting more than 26 million Americans, with millions more at increased risk. It is the 9th leading cause of death in the United States. In February 2002, The National Kidney Foundation set up a Kidney Disease Outcome Quality Initiative (K/DOQI) guideline, which was updated in 2006. Studies show that

primary care physicians are unaware of these guidelines and there are limited studies documenting physician adherence to the guidelines in outpatient practices. Aim :Within 3 months, we hope to achieve 60% adherence to K/DOQI guidelines by our physicians at the internal medicine clinic (IMC).PDSA: Weekly meetings of the quality improvement (QI)

group were scheduled. After IRB approval we assessed physicians' awareness to K/DOQI guidelines using CKD protocol sheets .We reviewed 209 charts of patients seen during the 30-day study period.Results: The

etiologies for chronic kidney disease were hypertension (74%) and diabetes (28%). For all the stages of CKD, the best-controlled factor in descending order was; maintenance of normal low density lipoprotein (LDL -55.7%), blood pressure control (54%) and cigarette smoking cessation (40%). For stages 3 and 4, the best-controlled factor in descending order was maintenance of hemoglobin ≥ 11 g/dl (45%), maintenance of phosphorus levels (35%). For referrals, 28% were appropriately referred to a nephrologist, 3% to a dietician but none was referred for vascular access.

Only 15% of the patients received Influenza, pneumococcal and TdaP vaccination.Conclusion: The implementation and achievement of K/DOQI guidelines by the primary care physicians was sub-optimal. The QI group identified this problem, created awareness by physician education, and designed a protocol sheet to enhance physician implementation and achievement of the guidelines. We have subsequently created personalized formative feedback to the individual physicians to achieve better results.

Further surveys will assess the compliance to the feedback.

ASSOCIATIONS OF SERUM ALKALINE PHOSPHATASE WITH ELEVATED CRP IN CHRONIC KIDNEY DISEASE ARE INDEPENDENT OF SERUM 25 OH VITAMIN D LEVELS

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High serum alkaline phosphatase levels are associated with elevated CRP levels in the population. This association might be confounded by low serum 25 OH vitamin D levels which are associated with both elevated C reactive protein (CRP) and high serum alkaline phosphatase levels. Hence, we examined whether higher serum alkaline phosphatase levels are associated with elevated CRP levels and whether this association is independent of serum 25 OH vitamin D levels in chronic kidney disease (CKD) population.

National Health and Nutrition Examination Survey (NHANES) III data were used. CKD was defined as $eGFR < 60 \text{ ml/min/1.73 m}^2$. Elevated CRP was defined as $CRP > 3 \text{ mg/L}$. The association of SAP with elevated CRP in CKD sub-population was examined in multivariable logistic regression models.

Of the 16,864 adults in NHANES III with valid data for GFR estimation, 5.4% who had CKD and non-missing data were included in the analyses. The mean (\pm SEM) age was 69.6 ± 0.7 years, 37% were men and 7% were African-American. Higher serum alkaline phosphatase levels were associated with lower serum 25 OH vitamin D levels. Compared to the lowest quartile, the highest quartile of serum alkaline phosphatase was associated with 2.54 (95% CI 1.28 - 5.04) fold higher odds of elevated CRP adjusted for demographics, comorbidity, GFR, liver function tests and serum calcium, phosphorus and 25 OH vitamin D levels.

The associations of serum alkaline phosphatase with elevated CRP are independent of serum 25 OH vitamin D in CKD. Therefore, serum alkaline phosphatase might be a marker of inflammatory milieu in the CKD population.

SAFETY AND IMMUNOGENICITY OF A NOVEL HEPATITIS B VACCINE ADJUVANTED WITH IMMUNOSTIMULATORY SEQUENCE (ISS) IN RENAL PREDIALYSIS AND DIALYSIS PATIENTS

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Purpose: Achieving rapid protection against hepatitis B (HBV) is critical for patients with chronic renal failure (CRF). HEPLISAV™ (H) is an investigational vaccine containing Hepatitis B surface antigen (HBsAg) and 1018 ISS, a Toll-like Receptor 9 agonist adjuvant.

Methods: This was a single-blind, randomized study of 41 subjects, 40–70 years of age, with progressive loss of renal function (glomerular filtration rate [GFR] ≤ 45 mL/min). Subjects were randomized 1:1 to receive either: H single dose or H double dose, at 0, 4 and 24 weeks. Anti-HBsAg concentrations were measured at weeks 4, 12, 24, 28 and 50. Immunogenicity endpoints included seroprotection rate (SPR) and serum geometric mean concentrations (GMC). Safety of H, including local and systemic reactogenicity and adverse events was assessed.

Results: A total of 21 subjects received the single dose and 20 subjects received the double dose. Fifteen subjects were on dialysis. Forty of the 41 randomized subjects (97.6%) received the first 2 injections of study drug. Only 2 subjects (1 at each dose level) received the third injection due to premature discontinuation of the study due to an FDA imposed clinical hold that was subsequently removed. The SPR for the combined H groups was 10% (4/40) after one dose at week 4, 58 % (23/40) after 2 doses at week 12 and 100% (11/11) after 2 doses at week 24. GMC was 3.2, 20.4 and 258.7 mIU/mL at week 4, 12 and 24, respectively. No significant differences were observed in subjects pre-dialysis or on dialysis. The most frequently reported local and systemic reactogenicity events were pain at the injection site and fatigue.

...Conclusion: H was immunogenic and well-tolerated and preliminary results suggest the potential of rapid protection against HBV in patients with chronic renal failure.

**CORRECTED SERUM CALCIUM BUT NOT
PHOSPHATE CORRELATES WITH CHANGE IN
SERUM PTH IN TREATMENT OF STAGES 3-5 CKD**

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Purpose: Serum PTH is thought to gauge phosphate (P) retention as signaled via FGF-23 and perhaps 1,25 vitamin (vit) D; for this reason, high PTH entrains phosphate binders, low phosphate diet, and both 25 vit D and active vit D (AVD) treatment. Serum calcium (Ca), the prime regulator of PTH, is often neglected, but may ultimately determine PTH. If so, PTH in treated CKD should follow Ca not P.

Methods: Among 100 patients (37 stage 3, 50 stage 4, 13 stage 5) treated in the Litholink CKD program, we measured baseline and treatment PTH, P, Ca, corrected Ca (cCa), and eGFR; correlation of PTH with these values was tested using simple and multivariate regression. Median follow-up interval was 74 days.

Results: Mean initial PTH, cCa, and P were 120 ± 146 pg/ml, 9.5 ± 0.5 mg/dl, and 4.0 ± 0.8 mg/dl, respectively. Change in PTH correlated with change in cCa ($p < 10^{-7}$, $r = 0.52$, $\text{adj } r^2 = 0.26$) but not P ($p = 0.23$). In a general linear model with treatment PTH as dependent, and pre-treatment PTH, treatment and pre-treatment P, Ca, cCa, and 25 vit D, eGFR and use of AVD, as independent variables, only 3 contributed independently: initial PTH, and final and initial cCa (partial r values were 0.96, -0.25, and 0.16, respectively). P, eGFR, Ca, 25 vit D, and use of AVD did not enter. In a separate analysis, change in cCa and change in P did not correlate.

Conclusion: Howsoever tentative at this early stage, we find no relationships between PTH and P. It is cCa, an established regulator of PTH secretion that has a great effect in our patients thus far. Possibly, PTH may gauge Ca homeostasis, as usual, and therefore, in relation to P be an indirect and even misleading index.

PHYSICIANS USING A COMPUTER GUIDANCE SYSTEM BASED ON KDOQI GUIDELINES APPEAR TO REGULATE SYSTOLIC BLOOD PRESSURE AROUND THE RECOMMENDED TARGET OF 130 mmHg

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Purpose: KDOQI guidelines recommend physicians treat systolic blood pressures (SBP) to below 130 mmHg in CKD patients. Whether or not physicians follow this guideline is presently untested. If they did, we should find that a plot of change in SBP (Δ SBP) vs. initial SBP (iSBP) passes through 0 at 130 mmHg. Here we report results for physicians using a KDOQI – based computer guidance program.

Methods: Among 84 stage 3-5 CKD patients in the Litholink CKD program, we have obtained Δ SBP and iSBP values from 14 practices (10 nephrology, 4 primary care). Median follow-up interval was 83 days. From the intercept and coefficient of simple linear regression we calculated the value for iSBP at Δ SBP =0.

Results: The regression of Δ SBP on iSBP gave an intercept of 81.4 with a coefficient of -0.641 ($F=59$, $p<0.0001$; adjusted squared multiple $R = 0.4$). From this, the crossing point is 127 mmHg. In other words, at SBP below 127 mmHg, SBP tends to rise, whereas above this value, it is reduced.

Conclusion: The strong regression reflects active physician treatment, for one would not expect such large slopes from simple regression to the mean. The set-point at 127 mmHg may reflect that physicians have absorbed and internalized KDOQI or that our program is being followed. Either way, we have first evidence in a range of practices, outside of a formal trial, that the 130 mmHg guideline for SBP is practical and can be achieved.

EFFICACY AND TOLERABILITY OF ORAL IRON AS INITIAL TREATMENT FOR IRON DEFICIENCY IN PRE-DIALYSIS ANEMIA OF CKD

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Oral (OFe) or intravenous (IvFe) iron is required for most CKD patients receiving Erythropoiesis Stimulating Agents (ESA). We retrospectively examined the efficacy and tolerability of OFe plus oral ascorbic acid (VitC) in iron naïve CKD patients as initial treatment for iron deficiency anemia.

Iron naïve patients with iron deficiency were identified from the UAB CKD Clinic database. They received OFe + VitC in a dose escalation protocol with the target dose of iron sulfate 325 mg tid. IvFe was given if transferrin saturation (TSAT) $\geq 20\%$ and ferritin ≥ 100 mg/dl were not achieved over a 6 month period. Statistical analysis was performed using descriptive statistics, T-test, Wilcoxin test and χ^2 test as appropriate to compare OFe success vs failure groups.

27 patients were identified an 18 month period. 68% were diabetic, 62% were female, 68% were black, 15% were smokers and 38% were receiving an ESA. Median (minimum, maximum) age was 63.7 years (19.9, 88.1), ferritin 78 ng/mL (1, 812), TSAT 16% (2, 34), Hemoglobin (Hb) 9.7 gm/dL (6.6, 11.6), GFR 22.5 ml/min/1.73 m² BSA (8, 60). 18% did not tolerate OFe (constipation 3, nonspecific 2). 38% required IvFe. There were no differences in baseline TSAT, ferritin, Hb, GFR, age, race or ESA status between those who achieved adequate iron stores on OFe vs those who did not.

OFe + VitC was successful in adequately replacing iron stores for 62% of iron naïve and iron deficient pre-dialysis CKD patients and is a reasonable first line therapy.

CHRONIC KIDNEY DISEASE IN HISPANICS: BASELINE CHARACTERISTICS OF THE HISPANIC CHRONIC RENAL INSUFFICIENCY COHORT (HCRIC) STUDY

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In addition to being the fastest growing minority group in the United States, Hispanics constitute a rapidly increasing proportion of the end-stage renal disease population. However, little is known regarding earlier stages of chronic kidney disease (CKD) in Hispanics.

We conducted a cross-sectional analysis of participants at enrollment into the HCRIC Study, an ancillary study of the CRIC Study. HCRIC participants were recruited at the University of Illinois from 2005 to 2008 and included Hispanics aged 21-74 years with CKD using age-based estimated glomerular filtration rate (eGFR). Baseline demographic and clinical characteristics of HCRIC Hispanics were compared with CRIC non-Hispanics.

Among HCRIC Hispanics, 75% were Mexican-American, 15% Puerto Rican, and the remainder had other Latin American ancestry. Significant comparisons are presented in the table below (p<0.05).

	Hispanics (n=327)	Whites (n=1638)	Blacks (n=1651)
Mean age (yrs)	57	59	58
Income < \$20,000	71%	16%	39%
Education < high school	68%	5%	26%
Hypertension	86%	79%	93%
Diabetes	68%	40%	51%
Coronary artery disease	16%	23%	22%
Blood pressure > 130/80	66%	35%	57%
eGFR (ml/min/m ²)	36.4	43.7	43.5
Median urine protein/d (g)	0.89	0.12	0.24

HCRIC is one of the largest prospective cohort studies of Hispanics with CKD. These study participants are disproportionately burdened with lower socioeconomic status, greater comorbid illnesses, worse blood pressure control, and more severe CKD than their non-Hispanic counterparts. Longitudinal analyses will demonstrate the impact of these health disparities upon clinical outcomes.

EFFECTS OF VITAMINE D2 REPLETION IN PATIENTS WITH CHRONIC KIDNEY DISEASE STAGES 3 AND 4

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Vitamin D deficiency is very common upon patients with chronic kidney disease (CKD). This problem is associated with hyperparathyroidism, which, in turn, is related to renal osteodystrophy, increased cardiovascular risk and arterial calcification. Since 2003, the Kidney Disease Outcomes Quality Initiative (KDOQI) recommends to treat patients with CKD stages 3 and 4 with vitamin D₂ supplements. However, few clinical evidences support this practice.

The main objective of this study is to determine the effects of ergocalciferol (vitamin D₂) administration on calcidiol (25 OH vitamin D) and parathyroid hormone (PTH) levels of patients with stages 3 and 4 CKD. Secondary objectives aim to evaluate the importance of calcidiol deficiency and to estimate ergocalciferol effect on average doses of active vitamin D therapy.

The retrospective study was conducted in a predialysis clinic at IUCPQ. To evaluate the first objective, patients with low calcidiol and high PTH levels who had received ergocalciferol supplements, as recommended by the KDOQI, were included. Serum calcidiol and PTH were assessed at baseline and at the endpoint. Calcium and phosphorus levels were measured every 3 months. Changes in active vitamin D dosing were also assessed.

Among the 29 patients treated with ergocalciferol for an average of 6.5 months, serum calcidiol increased by at least 12 nmol/L in 21 patients, while the target value (≥ 75 nmol/L) was reached by 17 of them. A 25% or higher PTH decrease was observed in 6 patients. Serum calcidiol was below target in 85 of the 124 patients evaluated for this objective, and their baseline value was 65.3 ± 29.4 nmol/L. The necessary dosing of active vitamin D decreased in only 1 of the 9 patients receiving this treatment. None of the results were statistically significant.

Over 2/3 of patients with stages 3 and 4 CKD included in this study had calcidiol deficiency, which is a higher prevalence than usually met in general population. Although ergocalciferol supplementation was associated with an improvement in serum calcidiol in most cases, neither did it result in a significant variation in PTH levels from baseline, nor did it allow a worthy decrease in active vitamin D needs. Indeed, if low calcidiol levels are observed in hyperparathyroid patients, ergocalciferol supplementation should not delay the use of active vitamin D therapy, which has already proven his effectiveness.

IS THERE A DIFFERENCE BETWEEN THE GLOMERULAR FILTRATION RATE (GFR) OF PATIENTS WITH HBV AND HCV CHRONIC HEPATITIS AND PATIENTS WITH CIRRHOSIS?

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Renal function in patients with cirrhosis is important prognostically, both before and following liver transplantation, as reflected by the inclusion of serum creatinine in the model for end-stage liver disease (MELD) score. MELD has shifted allocation of livers to patients with renal dysfunction.

We performed a cross-sectional analysis of patients with HBV chronic hepatitis, HCV chronic hepatitis and cirrhosis caused by these viruses hospitalized during 2002-2006 in our Hepatology department. Mean GFR was evaluated by the MDRD4 method.

The results are shown in Table 1.

Table 1.

Diagnosis	HBV	HCV	CH HBV	CH HCV
Number of patients	203	591	76	170
Age	42.08±12.9	50.48±11.17	52.56±10.52	61.14±10.93
Gender	129M, 74F	220M, 371F	42M, 34F	47M, 123F
GFR (ml/min)	83.39±17.1	77.5±16.07	79.5±27.26	71.57±22.81

The mean GFR was higher in patients (pts) with HBV chronic hepatitis than in pts with HCV chronic hepatitis ($p<0.001$). Patients with cirrhosis secondary to HBV infection had a higher GFR than patients with cirrhosis secondary to HCV ($p=0.01$). The GFR of pts with HCV chronic hepatitis was higher than the GFR of pts with cirrhosis due to this virus ($p<0.001$).

HBV chronic hepatitis, HCV chronic hepatitis and cirrhosis secondary to these viruses are associated with a reduction of the GFR. Functional renal impairment in diseases caused by HCV is more important than in diseases caused by HBV. The GFR is statistically lower in cirrhosis secondary to HCV than in HCV chronic hepatitis, which could signify that renal impairment as assessed by the eGFR might parallel the severity of liver disease.

WITHIN THE BROAD RANGE OF RENAL FUNCTION AS ASSESSED BY SERUM CREATININE, THE HIGHER THE CREATININE, HIGHER THE HOMOCYSTEINE AND THE LOWER IS HDL CHOLESTEROL, WITH RESULTANT HIGHER RISK FOR CARDIOVASCULAR DISEASE

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In 3239 patients referred for the diagnosis and therapy of hyperlipidemia, our first specific aim was to determine whether there were significant relationships between renal function as assessed by serum creatinine, and cardiovascular risk factors homocysteine, fasting serum insulin, and HDL cholesterol. Of the 3239 patients, 3012 (92.99%) were Caucasian, 159 (4.91%) Black, and 58 (1.79%) other, with 1576 (49%) women and 1663 (51%) men. Creatinine levels encompassed the usual broad population range with median values for the 5 quintiles being 0.7, 0.8, 0.9, 1.0, and 1.3mg/dl respectively. Creatinine was correlated with lipids, homocysteine and insulin with Spearman r values as follows, (all $p < .0001$): homocysteine 0.43, HDL-C 0.21, insulin 0.07, triglyceride 0.09. By stepwise multiple regression with homocysteine being the dependent variable and triglyceride (TG), creatinine, methylmalonic acid (MMA), insulin, age, race and gender as explanatory variables, major significant determinants of homocysteine were as follows:

Significant explanatory variable	Sign, p	Partial R ²
Methylmalonic acid	+, < .0001	6.2%
Creatinine	+, < .0001	4.1%
Age	+, < .0001	1.8%
Male sex	+, < .0001	1.5%
TG	+, < .0075	0.2%
Black race	+, < .025	0.1%

With HDL cholesterol as the dependent variable, and explanatory variables TG, creatinine, MMA, insulin, age, race and sex, major significant determinants of HDL-C were as follows:

Significant explanatory variable	Sign, p	Partial R ²
Female sex	+, < .0001	9.4%
Insulin	-, < .0001	3.81%
TG	-, < .0001	1.9%
Age	+, .012	0.1%
Creatinine	-, .016	0.2%

Optimization of renal function, even within the normal creatinine range, may be important in prevention of cardiovascular disease as mediated by homocysteine and HDL.

PITFALLS OF VITAMIN D REPLACEMENT THERAPY

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Vitamin D plays a vital role in the metabolic functions of the body. Since it requires renal activation, many patients with chronic kidney disease (CKD) have low levels. Recent epidemiologic studies have suggested that adequate levels of vitamin D improve morbidity and mortality in patients with CKD, hence KDOQI guidelines have recommended replacement therapy.

We present the case of a 47-year-old African American male nursing home resident who presented to clinic complaining of progressively worsening nausea, vomiting and sleep disturbance for an uncertain amount of time. Past history included CKD secondary to type II diabetes mellitus. He also had vitamin D deficiency, for which he was getting oral ergocalciferol 50,000 international units (IU) monthly. He was later started on IV paricalcitol for secondary hyperparathyroidism upon initiating hemodialysis, with doses titrated per his parathyroid hormone (iPTH) levels. Physical examination was within normal limits. Labs drawn that day included iPTH and both 25 and 1,25 hydroxy Vitamin D levels.

Levels returned one week later, showing his 25[OH]D level was >155 ng/mL (increased from 30.9 ng/mL) and his 1,25[OH]D level was 23.9 pg/mL (decreased from 27.4 pg/mL). iPTH levels were at goal. Review of his medication administration record from the nursing home revealed that he had erroneously received oral ergocalciferol 50,000 IU daily for the previous 69 days. The patient was called back and evaluated for signs and symptoms of vitamin D toxicity.

During attempted replacement with both ergocalciferol and paricalcitol, our patient developed toxic levels of 25[OH]D, but 1,25[OH]D levels remained within normal limits - in fact, decreased slightly – in spite of supratherapeutic ergocalciferol doses.

This case demonstrates that there may not be much benefit in supplementing CKD patients with compounds that require renal conversion for activation and may, in fact, expose them to the risk of toxicity. This case report gives an example of the pitfalls faced in replacing vitamin D in patients with CKD.

PREGABALIN THERAPY IN REFRACTORY UREMIC PRURITUS

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Pruritus in hemodialysis patients is a vexing clinical problem for which many medical therapies have been proposed. Gabapentin has been used successfully in some studies but has the disadvantage of sedation and other neurological adverse drug reactions (ADR). Pregabalin a derivative of gabapentin associated with less neurological ADRs and could potentially be used for symptoms relief in uremic pruritis. Twenty patients on thrice a week maintenance hemodialysis with severe pruritis refractory to traditional measures including emollients, antihistamines, xylocaine cream and tacrolimus ointment were enrolled in a non-randomized single arm study to evaluate the efficacy of pregabalin at 75 mg every alternate day for four weeks. Pruritis was measured with visual analogue scale (VAS) on a 10 cm line at 0, 1, 2, 3 and 4 weeks after starting therapy. Patients with pre-existing liver and skin diseases were excluded. No changes were done dialysis Rx. ADRs associated with pregabalin, especially neurological, were noted. Baseline characteristic of patients included mean age 57.05 ± 7.52 yrs, sex (M/F) 12/8, mean time on dialysis 291.65 ± 156.61 days, mean kt/v per session 1.27 ± 0.07 , mean iPTH 169.35 ± 64.21 pg/ml, mean Ca 9.48 ± 0.43 mg/dl, mean iP 4.48 ± 0.83 mg/dl, Ca x P 42.467 ± 80 mg²/dl², mean Hb 10.21 ± 0.65 g/dl and mean S.albumin 3.44 ± 0.46 g/dl. VAS score showed significant improvement at the end of 4 weeks ($7.720 \pm .93$ and $5.941 \pm .36$, $p < 0.0001$). ADRs included drowsiness (n=1, stopped Rx after 2 weeks), vertigo (n=1), sedation (n=4) and ataxia (n=1). Presence of diabetes mellitus correlated with response to therapy ($p = 0.029$). Pregabalin is a promising agent for uremic pruritis although the incidence of ADRs appears to be high in the ESRD population primarily as the drug is renally excreted. Large trials are necessary to establish the efficacy and safe dose of this agent in the chronic kidney disease patients.

HEMATOLOGIC OUTCOMES AND DOSING PATTERNS IN ANEMIC PATIENTS WITH PRE-DIALYSIS CHRONIC KIDNEY DISEASE (CKD) SWITCHING FROM DARBEPOETIN ALFA (DARB) TO EPOETIN ALFA (EPO)

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The purpose of this study was to compare dosing patterns, hemoglobin (Hb) levels, and erythropoiesis-stimulating agent (ESA) cost outcomes in pre-dialysis CKD patients switched from DARB to epoetin alfa (EPO).

This was a retrospective observational chart review of pre-dialysis CKD patients from a nephrology clinic in the southwestern U.S. Patients were initially treated with DARB and converted to EPO between January 2005 and December 2007. Patients included were age ≥ 18 years, had ≥ 1 medical claim for CKD, switched from DARB to EPO, received ≥ 2 doses of both DARB and EPO, and received ≥ 180 days treatment on either side of the switch. Exclusion criteria included treatment gaps >90 days, dialysis initiation, or active cancer/chemotherapy during study period.

A total of 85 patients met inclusion criteria. Mean (SD) age was 71.4 years (12.7), and 60.0% were female. Common comorbidities included hypertension (94.1%) and diabetes (67.1%). Mean ESA dosing interval was similar pre- and post-switching (pre-switch DARB 34.5 days, post-switch EPO 33.7 days, $P=.684$) as were mean Hb levels (pre-switch 11.6 g/dL, post-switch 11.5 g/dL, $P=.509$). The mean weekly DARB dose was 44.1 mcg and EPO dose was 10,218 Units associated with a dose ratio of 232:1 (Units EPO: mcg DARB). Mean weekly ESA costs were \$217 for DARB and \$133 for EPO reflecting a 63% DARB price premium.

In this analysis of claims data of pre-dialysis CKD patients who switched from DARB to EPO, similar ESA dosing intervals and Hb levels pre- and post-switching were observed. Weekly ESA costs were lower following the switch. Further research is warranted in other centers.

SHORT-TERM EFFECT OF DIETARY PHOSPHORUS RESTRICTION AND LANTHANUM CARBONATE ON FGF23 IN CHRONIC KIDNEY DISEASE PATIENTS

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Dietary phosphorus stimulates FGF23 secretion, but the effects of dietary phosphorus restriction and/or phosphorus binders on FGF23 levels in pre-dialysis CKD have not been studied in detail. We investigated the hypothesis that dietary phosphorus restriction alone or in combination with lanthanum carbonate would lead to decreased FGF23 levels in normophosphatemic stage 3-4 CKD patients.

Sixteen subjects (mean eGFR 40 ± 12 ml/min/m²) were randomly assigned to 1 of 4 groups (N=4 per group) for 2 weeks: 1) 800 mg of daily dietary phosphorus and lanthanum carbonate 1000 mg three times a day; 2) 1500 mg of daily dietary phosphorus and lanthanum carbonate 1000 mg three times a day; 3) 800 mg of daily dietary phosphorus and placebo; or 4) 1500 mg of daily dietary phosphorus and placebo.

Dietary phosphorus restriction was accomplished with the use of tightly controlled diets prepared by a metabolic kitchen. Serial measurements of 24-hr urine phosphate, serum phosphate, calcium and cFGF23 were used in the repeated-measures analyses.

The post-intervention response in 24-hr urine phosphate and cFGF23 was significantly different between group 4 and groups 1-3 ($p < 0.05$ for both). The 24-hr urine phosphate decreased significantly in all groups, except for group 4. The greatest lowering in 24-hr urine phosphate was detected in group 1 (from 659 ± 270 mg/day at baseline to 214 ± 163 mg/day at day 3, $p < 0.0001$). While there was no significant change in serum phosphate or calcium in any group, cFGF23 levels rose early and significantly in group 4 (from 121.8 ± 60 RU/ml at baseline to 178.5 ± 61.7 RU/ml at day 3, $p = 0.02$). Despite a significant drop in 24-hr urine phosphate, there was no change in cFGF23 levels in groups 1-3.

Dietary phosphorus restriction alone or in combination with lanthanum carbonate effectively lowers 24-hour urine phosphate without accompanying changes in serum phosphate or calcium. Reductions in FGF23 levels may require interventions with longer duration.

KIDNEY-RELATED EFFECTS OF EMERGING MEDICATIONS FOR HEART FAILURE

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Diuretics are thought to contribute to deterioration of renal function in patients with heart failure (HF). Renal dysfunction and hyponatremia are both known to be associated with increased morbidity and mortality in these patients. Vasopressin receptor antagonists (VRA) and Adenosine-A1 receptor antagonists (A1RA) represent emerging pharmacological therapies that potentially lack the renal adverse effects of diuretics. This study explores the currently available data on the impact of these agents on kidney-related parameters. Articles cited in PubMed database from 1980 to 2009 using key words: “vasopressin receptor antagonist”, “ADH-receptor antagonist”, Adenosine-A1 receptor Antagonists”, and “heart failure” were searched. Those clinical randomized controlled trials that exclusively included HF population were identified, and relevant articles were selected. The results of these studies were then reviewed and compared with regards to kidney-related parameters. A total of 27 and 22 relevant articles were identified for VRA and A1RA respectively. Nine randomized, placebo-controlled trials were selected to be included in this study for VRA, and 6 for A1RA. For VRA, 4 studies (44.5%) could not find any significant change in serum sodium levels, and the rest showed only a modest rise (e.g. ≤ 4 meq/l). Seven studies (77.8%) did not find any significant change in renal function. For A1RA, 3 studies (50%) did not show any significant change in renal function, and only two studies (33%) found increase in urine volume. While there is a promising theoretical basis and possible cardiac benefits for use of VRA and A1RA as adjunct therapy for patients with HF, currently available data show only modest beneficial impact on kidney-related parameters for both therapies. Future large-sized trials are clearly needed to further evaluate these effects and their potential impact on morbidity and mortality of patients with HF.

A COST-EFFECTIVENESS MODEL OF PHOSPHATE BINDERS FOR THE TREATMENT OF HYPERPHOSPHATEMIA IN CHRONIC KIDNEY DISEASE (CKD)

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Hyperphosphatemia in CKD patients is associated with comorbidities and an increased risk of mortality. The objective of the analysis was to evaluate the cost-effectiveness of Fosrenol® (lanthanum carbonate), relative to Renagel® (sevelamer hydrochloride), for the treatment of hyperphosphatemia in CKD.

A cost-effectiveness model was constructed from a U.S. managed care perspective. Model inputs were wholesale acquisition cost (WAC), Daily Average Drug Consumption (DACON), and phosphate binding capacity (mg of phosphorus bound per 1 gram tablet). Comparators included lanthanum carbonate (LC) 1,000 mg and sevelamer hydrochloride (SH) 800 mg. The model was structured to allow for variation in parameter estimates for each product, such as phosphate binding capacity. The primary model outcome was the cost per mg of phosphate bound daily. A probabilistic sensitivity analysis was conducted to evaluate the robustness of the estimates from the model.

LC has a lower DAACON (3.3) compared with SH (8.2). The mean estimates for the amount of phosphate bound daily indicate, based on urinary P, that LC has a higher daily phosphate binding capacity per gram (109 mg) versus SH (36 mg). Multiplying the phosphate binding capacity by the dose per tablet and the DAACON translates into 359.7 mg of phosphorus bound daily with LC compared to 236.2 mg daily with SH. The cost per mg of phosphorus bound daily was \$0.04 for LC as compared to \$0.07 for SH based on a cost per pill of \$4.31 for LC and \$2.13 for SH. The model was robust to changes in the phosphate binding capacity. Using 1,000 simulations for the probabilistic sensitivity analysis revealed that LC has greater than 95% probability of having the lowest cost per mg of phosphate bound daily versus SH.

These results suggest LC is a more cost-effective phosphate binder in the treatment of hyperphosphatemia among CKD patients than SH. Sensitivity analyses also suggest that the cost-effectiveness of LC was robust to variations in the primary model outcome.

ROLE OF ACIDOSIS IN THE MANAGEMENT OF ANEMIA OF CHRONIC KIDNEY DISEASE

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Metabolic acidosis can developed as kidney function decreases. It has also been associated with altered protein binding and enzymatic functions. The purpose of this study is to investigate if there is an association between metabolic acidosis and impaired erythropoiesis as manifest by increased doses of Erythropoietin stimulating agents (ESA) in adult outpatients with CKD not requiring dialysis.

This is a retrospective cohort study of patients who were treated at the Cleveland Clinic. The clinical charts and electronic records of adult outpatients with CKD who initiated ESA therapy before March 2005 were reviewed. One hundred and seventeen patients naïve to erythropoietin therapy were sequentially selected and were followed up for more than 12 months. For each eligible patient ESA dose, Hb, plasma bicarbonate levels were recorded at 0, 3, 6 and 12 month period following initiation of ESA therapy. Multivariate analysis was then performed to determine correlation between any of these parameters.

The analysis included data from 113 outpatients (mean [SD] age, 65.9 [14.4] years; 53.2 % male; 66.7% white, 29.7% black, 3.6% other). Twenty-six patients received Epoeitin (EPO) only and 87 received Darbapoeitin (DARB) only. For each patient monthly dose was calculated. ESA dose per month was standardized by converting EPO to DARB using dose conversion algorithm. Data was analyzed using repeated measures mixed models to develop the reduced regression equation. Of all the variables there was a significant positive correlation ($p < 0.05$) between race (black vs. other), time in months, frequency, and type of ESA(Epoeitin) and dose per month. There was no significant correlation between plasma bicarbonate and ESA dose.

Variations in plasma bicarbonate levels were not associated with differences of plasma bicarbonate and increased ESA dosing requirements among adult CKD outpatients not requiring dialysis.

EPO AND HEMATIDE™ REQUIREMENTS DIFFER IN ESA HYPORESPONSIVE HD PATIENTS

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About 5% to 10% of HD pts are hyporesponsive to ESAs (Johnson, 2007), which has been associated with increased CV mortality (Regidor, 2006). Hematide™ is a synthetic, peptidic ESA linked to polyethylene glycol that is in phase 3 development for the treatment of anemia associated with CKD. The purpose of the current analyses was to compare Hematide and baseline epoetin alfa (EPO) dose requirements in patients who were hyporesponsive to EPO treatment.

Unplanned analyses of two phase 2 studies were performed. HD pts were switched from TIW EPO to QM (Q4W) Hematide. Doses were titrated over 5 to 6 mo to maintain target Hb (11-13 g/dL). 145 pts who received ≥6 doses of Hematide were divided into 3 groups on the basis of their baseline EPO dose: highest (top 10%; hyporesponsive), lowest (bottom 10%; sensitive), and middle (80%).

The ratio of top 10% to bottom 10% was 5.6 for EPO and 2.2 for Hematide to maintain similar Hb levels (Table). Although mean TSAT increased from 42% to 50% from baseline to end of study ($p<0.001$), this was not considered clinically important. 4 pts (3%) had treatment-related AEs; each occurred in 1 pt. The safety profile of Hematide appears consistent with that of other ESAs (Macdougall, 2008).

Group	EPO		Hematide	
	Mean Baseline Dose (U/wk)	Hb (g/dL)	Mean End of Study Dose (mg/mo)	Hb (g/dL)
Top 10%	22950	11.9	10.7	11.5
Middle 80%	9350	11.5	6.5	11.2
Bottom 10%	4080	11.5	5.0	11.3
Top 10% to Bottom 10%	5.6	1.03	2.2	1.01

Compared with EPO dose requirements, Hematide dose requirements appear to be lower in pts who were hyporesponsive to EPO treatment.

CARDIOVASCULAR DISEASE PREVALENCE IN THE HISPANIC CHRONIC RENAL INSUFFICIENCY COHORT (HCRIC) STUDY

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Cardiovascular disease (CVD) is the leading cause of death in patients with chronic kidney disease (CKD). Hispanics are the fastest growing minority group in the U.S. and are known to have a higher burden of CVD risk factors, but data about the prevalence of CVD among Hispanics with CKD are limited.

The Hispanic Chronic Renal Insufficiency Cohort (HCRIC) Study, an ancillary study of the CRIC Study, recruited Hispanics who were aged 21-74 yr and had CKD using age-based eGFR inclusion criteria. We conducted a cross-sectional analysis to compare the prevalence of self-reported clinical CVD and subclinical measures of CVD between Hispanic and non-Hispanic participants of HCRIC and CRIC, measured during the first year following study entry.

The results are summarized in the table below. Statistically significant pairwise comparisons ($p < 0.025$) are bolded. Hispanics were the referent category.

	Hispanic n=496	White n=1638	Black n=1651
MI or prior revascularization	18%	23%	22%
Congestive heart failure	7.5%	7.1%	13%
Peripheral vascular disease	7.1%	6.4%	7.1%
Coronary artery calcium (CAC) score >100	33%	41%	34%
Left ventricular hypertrophy	70.5%	40%	63%
Ankle-brachial index (ABI) <0.9	14%	11.5%	18%

In these cohorts, Hispanics have a lower prevalence of self-reported MI and CAC score >100 and a higher prevalence of left ventricular hypertrophy (LVH) as compared with non-Hispanic whites. Compared with non-Hispanic blacks, Hispanics have a lower prevalence of self-reported heart failure and abnormal ABI, and a higher prevalence of LVH. Factors that contribute to these racial/ethnic differences in CKD as well as their clinical significance will be evaluated in subsequent longitudinal analyses.

KIDNEYMOBILE: ON THE ROAD TO HEALTHIER LIVING

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In 2005, the National Kidney Foundation of Illinois introduced the KidneyMobile® to identify unknown cases of kidney disease and diabetes. The program provides education and facilitates access to healthcare among underserved and high risk populations. The objective of this presentation is to describe the screening results related to prevention and control of diabetes.

Screenings are centralized in geographic areas with greater numbers of individuals from high risk and/or underserved populations, especially African American and Latino populations. Each participant completes a short health-related questionnaire, provides urine and blood samples. Screening measures obtained include: BMI, waist circumference, blood pressure, glucose, hemoglobin, albuminuria, hematuria, and pyuria. Screenings are administered by trained medical/health education professionals. Participants are informed of results. Those with abnormal results are advised to contact their health care providers for follow-up; those without a provider are referred to a local Federally Qualified Health Center; and NKFI staff contact these participants to support follow-up with a provider or with NKFI's partner Access Community Health Network.

The KidneyMobile has screened greater than 10,000 individuals: average age of 53.59 years; 66% female; 18.7% African American, 41.5% Non-Hispanic White, 34% Hispanic, 4.6% Asian, and 1.2% "Other"; and 65.3% without health insurance. Of the total, 20% had a known diagnosis of diabetes. Of those without known diabetes, screening results suggested that 1.9% had possible diabetes, 9.3% had possible pre-diabetes, and 88.8% had normal glucose values. Chi square comparisons across these 3 categories by gender, ethnicity, and insurance status indicated significant differences only for gender with women having a higher proportion of normal results. Additional analyses will be presented.

The KidneyMobile is a successful community based approach to reaching underserved populations for prevention efforts.

WARFARIN DOSING IN PATIENTS WITH IMPAIRED RENAL FUNCTION

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Warfarin pharmacokinetics are influenced by many clinical and genetic factors, posing significant challenges for optimal anticoagulation management in the general medical population. Patients with chronic kidney disease (CKD) may require lower warfarin doses. We assessed the degree of warfarin dose reduction associated with moderate or severe CKD in a derivation cohort, and replicated the CKD-dose association in an independent cohort.

Linear-regression analysis was conducted to assess the influence of CKD, *CYP2C9* and *VKORC1* genotype, age, race, gender, body mass, comorbid conditions and drug interactions on warfarin doses. The derivation cohort was comprised of 708 participants enrolled in a warfarin pharmacogenetics study at UAB. The replication cohort was comprised of 272 participants from the University of Illinois in Chicago (UIC cohort).

Patients were categorized into 3 groups based on their eGFR (eGFR ≥ 60 no/mild CKD; eGFR=30-59 moderate CKD; eGFR<30 ml/min/1.73 m² severe CKD). Among UAB and UIC participants the majority (59.3% and 65.8%) had no/mild CKD, 31.8% and 27.6% had moderate CKD, and 8.9% and 6.6% had severe CKD, respectively.

Renal impairment influences warfarin dose requirements. As compared to patients with no/mild CKD, patients with moderate CKD required 10.3% lower warfarin doses (95% CI: 4.9%-15.4%; p=0.0005) and patients with severe CKD required 19.2% lower doses (95% CI: 11.3%-26.5%; p<0.0001). Age, weight, amiodarone therapy and possession of *CYP2C9* and *VKORC1* variants were also associated with significantly lower dose requirements (all p-values <0.0005).

In conclusion, initial warfarin doses in CKD patients should be lower than those used for the general population.

**A RETROSPECTIVE COHORT STUDY OF TRENDS IN
HEMOGLOBIN (HB) LEVELS AMONG ERYTHROPOIESIS-
STIMULATING AGENT (ESA)-TREATED CHRONIC KIDNEY
DISEASE (CKD) NON-DIALYSIS (NOD) PATIENTS IN THE
FRESENIUS MEDICAL CARE NORTH AMERICA (FMC-NA)
CKD DATA REGISTRY.**

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We evaluated Hb trends among CKD-NOD patients receiving ESA treatment during a 36-month period in which several factors may have impacted clinical practice, including the CREATE and CHOIR studies (11/2006), and ESA label changes (03/2007 and 11/2007).

Monthly cross-sectional data was obtained from the FMC-NA CKD Data Registry comprised of observational information from outpatient nephrology practices from 1/2006 to 12/2008. Patients identified with CKD (ICD-9 585.x), or two serum creatinine levels >1.8 mg/dL separated by ≥60 days were enrolled in the Registry. The study group was documented to be on an ESA treatment protocol throughout the period of study. All individual Hb levels from ESA order start date until the first occurrence of progression to chronic renal replacement therapy, renal transplant, loss to follow-up, death or discontinuation of ESA treatment were included in the analysis. For each patient, Hb measurements within each month were averaged. Monthly population mean and standard deviation (SD) Hb levels were estimated. Overall, the mean (SD) Hb decreased, from 11.7 (1.4) g/dL in Jan 2006 (n=122) to 11.2 (1.3) g/dL in Dec 2008 (n=258) (p=0.003). This decrease was consistent when stratified by gender, age and CKD stage. Comparing Jan 2006 to Dec 2008, the frequency of patients with Hb >12 g/dL declined from 37.7% to 22.9%, there was an increase in the proportion of patients with Hb 10-12 g/dL, 51.6% vs. 62.8% and an increase in patients with Hb <10g/dL, 10.7% vs. 14.3%. These results were not adjusted for differences in case-mix and, given the small sample size, may not be generalizable. These data suggest that nephrologists anemia management practice patterns may have changed and been impacted by several factors, including reports in the literature and label changes.

COMPARATIVE EFFECTIVENESS OF PARICALCITOL VERSUS CALCITRIOL TREATMENT IN CHRONIC KIDNEY DISEASE [CKD] PATIENTS WITH SECONDARY HYPERPARATHYROIDISM [SHPT]

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To assess the comparative effectiveness of medication use, outpatient services, hospitalizations and costs in pre-dialysis CKD patients with SHPT receiving paricalcitol or calcitriol.

A retrospective matched analysis of 1,266 pre-dialysis patients per cohort was performed to compare health outcomes and costs of paricalcitol with calcitriol treatment for SHPT. Patients were matched using propensity scoring for age, gender, Charlson co-morbidity index, and pre-index total costs. Multivariate models were adjusted for age, gender, insurance, physician specialty, region, pre-index co-morbidity, and pre-index costs to evaluate the impact of pre-dialysis paricalcitol treatment versus calcitriol on medications, outpatient services, hospitalizations and total costs.

Multivariate analyses demonstrated fewer hospitalizations and outpatient services, and lower total costs:

Paricalcitol vs Calcitriol	Parameter Estimate	Utilization/ Cost Ratio	95% Confidence Interval	
Cardiovascular-Related Medication use	-0.005	0.995	0.987	1.003
Cardiovascular-Related Outpatient visits	-0.026	0.975	0.966	0.984
Cardiovascular-Related Hospitalizations	-0.107	0.899	0.846	0.955
Cardiovascular-Related Total Costs	-0.111	0.895	0.847	0.946
All-Cause Medication use	-0.022	0.979	0.973	0.983
All-Cause Outpatient visits	-0.054	0.948	0.942	0.954
All-Cause Hospitalizations	-0.141	0.868	0.821	0.918
All-Cause Total Costs	-0.102	0.903	0.866	0.941

Paricalcitol use was associated with fewer cardiovascular-related and all-cause medication use, out-patient services and hospitalizations, as well as lower costs compared to calcitriol. Further comparative studies may be needed to confirm these real-world findings.

ELEVATED HUMAN CHORIONIC GONADOTROPHIN (HCG) LEVELS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Females in different age groups are often subjected to serum HCG testing prior to most diagnostic and therapeutic interventions. A positive test result leads to further testing to rule out pregnancy and avoid possible fetal teratogenicity. The impact of chronic kidney disease (CKD) on HCG testing has not been studied to a large extent.

Study: A retrospective chart review was done for patients that had been screened for possible renal transplantation at North Shore University Hospital. Of the total 171 charts, case charts of all female patients were reviewed and all with positive HCG >5IU/L were studied.

Results: 5 patients were identified with positive HCG test on routine pre transplant screening. Two patients were dialysis dependent while the remaining had CKD Stage V. Mean GFR was 12.43 cc/min, creatinine 4.96mg/dl and mean duration of CKD was 9.2 years. Four patients were post menopausal. The mean value of HCG was 8.4IU/L (range 6-10). EBV serologies were positive in all patients; 2 were CMV positive. No other environmental exposures which could have led to the production of HCG were identified. Despite aggressive investigations of their elevated levels, the elevations were without cause. This positive test contributed to delays in transplantation and increased overall cost of treatment in our patients. This study might suggest that CKD may affect HCG levels by impairing metabolism. Why it is positive in some CKD women and not all is unclear at this point. Correcting HCG levels for age and co morbidities might be prudent to avoid procedural delays and treatment costs in such women.

SYMPTOMS AND IMPACTS REPORTED BY PATIENTS WITH TYPE 2 DIABETES AND NONDIALYSIS CHRONIC KIDNEY DISEASE RELATED ANEMIA

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In a qualitative research study with a group of patients having type 2 diabetes (T2D) and anemia related to their nondialysis Chronic Kidney Disease (CKD), we aimed to describe patient experiences with anemia-related symptoms and related impacts.

Forty patients with a clinical history of T2D and nondialysis CKD related anemia were identified and recruited to participate in interviews (32 individual and 2 focus groups of 4 each). Patients were asked open-ended questions about the symptoms and symptom-related impacts that they attributed to their anemia. Interviews were audio recorded and transcribed. Concepts expressed by patients were coded using Atlas.ti software, grouped by like responses and summarized in tables.

The study population was 68% female, 43% Caucasian, with a mean age 66.3 ± 12.4 years, mean eGFR 32.4 ± 12.6 mL/min/1.73m², mean hemoglobin 10.3 ± 0.6 , and mean HbA1C $7.2 \pm 1.2\%$. Low energy was reported by 85% of patients. Patients' expressions included: "I wear out quickly," "I'm so tired my body aches," and "I feel drained."

Limitations in physical functioning were reported by 56% to 82% of patients, including: difficulty with housework, work activities, self-care, walking long or even very short distances, and climbing stairs. Patient expressions included: "I don't have the energy to sweep," "when making the bed I have to stop and rest," "I use to have more energy to do my job," "It wears me out to even get in the bathtub," "I walk a little and then stop," and "I go up [stairs] very slowly."

Patients with T2D and anemia related to nondialysis CKD reported feeling low in energy and very limited in physical functioning.

ASSOCIATION OF THE FRIESINGER SCORE AND ESTIMATED GLOMERULAR FILTRATION RATE IN AN URBAN SOUTH ASIAN PATIENT POPULATION

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Background The correlation between kidney function and the Coronary Artery Disease (CAD) severity assessed by the angiographic Friesinger score has not been studied in the South Asian population.

Methods: We performed a single-center, cross-sectional study. Estimated glomerular filtration rate (eGFR) calculated by the Modification of Diet in Renal Disease (MDRD) equation and Friesinger score to quantify the severity of CAD were the primary endpoints.

Results: The mean eGFR was significantly lower in participants with a Friesinger score of ≥ 5 compared to those with a score of < 5 (68 vs. 90 ml/min/1.73m² by MDRD). In univariate and multivariate analysis, an eGFR of < 60 ml/min/1.73m² was associated with a 9.4 fold increased odds of a higher Friesinger score compared to an eGFR ≥ 60 ml/min/1.73m² (p=0.043). Also, a 10 ml/min/1.73m² decrease in eGFR was associated with a 1.63 fold increased odds of higher score (95% CI 1.10–2.37, p = 0.042).

Conclusion: Our study demonstrates that kidney function as assessed by eGFR is a significant independent predictor of severity of CAD as determined by Friesinger Score.

PREVALENCE OF IRON DEFICIENCY IN CHRONIC KIDNEY DISEASE-. ANALYSIS IN A SINGLE OUTPATIENT NEPHROLOGY SETTING

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The frequency of iron deficiency associated with anemia in the non-dialysis CKD population has been evaluated in a number of recent epidemiologic studies. We explored the prevalence of iron deficiency in CKD patients with or without anemia in a single nephrology practice, in an outpatient setting.

We analyzed the laboratory results of all new consults referred to our nephrology office from 2006 to 2008. 308 patients of a total of 625 were included in the analysis. These patients had sufficient data available to assess CKD stage, anemia and iron status. Iron deficiency was defined as a serum ferritin \leq 100mg/dl and/or an iron saturation of \leq 20(%). Anemia was defined as Hgb less than 12gm/dl.

In this CKD population, the overall prevalence of iron deficiency was 40%. The incidence was similar in both women and men.

Anemia was present in 66% of the population, 58% in men and 69% in women ($p=0.06$). Iron deficiency anemia was found in 23% of the population. In patients with anemia, iron deficiency was found in 40% of men and 30% of women. There was no significant difference between genders. In advanced CKD (stage 3-5), the prevalence of iron deficiency associated with anemia was 48%, again similar in men (61%) and women (52%).

The incidence of anemia in the non-dialysis CKD population, associated with the presence of iron deficiency, requires awareness and testing for these conditions. Iron replacement makes the therapy of anemia in CKD easier and more rational.

CHRONIC KIDNEY DISEASE PROGRESSION TO ESRD: SMOOTH AND PROGRESSIVE VS UNEVEN AND STACCATO PATTERNS? – A MAYO CLINIC PBRN-BASED 82-MONTH ANALYSIS OF 100 HIGH-RISK CKD PATIENTS – IMPLICATIONS FOR A PARADIGM CHANGE IN RENO-PROTECTION.

Macaulay Onuigbo¹, Nnonyelum Onuigbo², Mayo Clinic, Rochester, MN, USA & Midelfort Clinic, Eau Claire, WI, USA¹; NTEC Solutions, LLC. Eau Claire, WI².

We continue to experience an ESRD epidemic in the USA and worldwide. Current literature on ESRD outcomes in CKD is contradictory and unclear. The prevailing consensus is the continuous progressive loss of eGFR leading to ESRD. All contemporary theories of reno-protection derive wholly from this premise. We hypothesized from anecdotal evidence based on our clinical experience that ESRD in CKD is an unpredictable event, often triggered by ARF.

Since September 2002, we have prospectively followed a cohort of 100 high-risk CKD patients who had presented with AKI at enrollment. We report an 82-month patient-level PBRN-based analysis.

52 M/48 F, age 71.5 (25-92) years. Mean eGFR at enrollment - 22.1 +/- 8.8. CKD stages at enrollment - III (24), IV (58) and V (16). Final CKD stages - I (3), II (10), III (41), IV (22) and V (20). eGFR was stable/improved in most. 17 progressed to ESRD – 2 from stage III, 11 from stage IV, and 4 from stage V. ESRD progression was abrupt, unpredictable, and preceded by acute medical/surgical events in 15/17 (88%) patients – hypotension/shock (7), sepsis (2), cardiac surgery (2), lymphoma (1), contrast nephropathy (1), bladder outlet obstruction (1), and dementia/failure to thrive (1).

The theory of a continuous progressive loss of eGFR in CKD leading to ESRD was debunked by our study. ESRD was often precipitated unpredictably by AKI. We submit that in 2009, Nephrologists still do not understand the natural history of CKD. A better understanding will allow effective prevention to decelerate the ESRD epidemic. Larger studies are warranted. If confirmed, our findings would require a major paradigm shift in our current understanding and perception of the entire concept of reno-protection.

**TO STENT OR NOT TO STENT: RENAL ARTERY STENOSIS
– A MAYO HEALTH SYSTEM HYPERTENSION CLINIC 82-
MONTH PBRN-BASED PATIENT-LEVEL PROSPECTIVE
DATA ANALYSIS OF 26 HIGH- RISK CKD PATIENTS WITH
RENAL ARTERY STENOSIS PRESENTING WITH ACUTE
KIDNEY INJURY**

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The appropriate role of percutaneous transluminal renal angioplasty and stenting (PTRAS) in renal artery stenosis (RAS) is controversial especially with reference to preservation of renal function. We hypothesized that PTRAS is only useful in the setting of ARF/AKI.

Since September 2002, we have continued to prospectively follow a cohort of 26 high-risk CKD patients with RAS presenting with AKI. Patients who continued to demonstrate uncontrolled hypertension, flash pulmonary edema and/or persistent renal failure underwent PTRAS. We completed an 82-month prospective analysis in June 2009.

26 Caucasians, M:F = 10:16, age 75.3 ± 6.4 (63–87) years. Follow-up 40.3 ± 23.2 (1–74) months. RAS was bilateral in 6, unilateral in 19, and single kidney in 1. 9 (35%) underwent PTRAS, 8 unilaterally and one bilaterally. 6/26 patients (23%) progressed to ESRD - 2/6 following PTRAS, 4/6 without. 10 (38%) died - 2/10 following PTRAS, 8/10 without. In 7 patients following PTRAS, eGFR increased from 27.4 ± 12.7 (11–47) to 50.3 ± 21.7 (23–68) mL/min/1.73 m² BSA ($p = 0.018$) after 46.9 ± 22.6 (15–65) months. In 13 without PTRAS, eGFR increased from 27.4 ± 9.18 (13–45) to 36.2 ± 20.3 (13–87) mL/min/1.73 m² BSA ($p = 0.098$) after 52.7 ± 12.1 (29–71) months.

Our 82-month prospective patient-level data supports the paradigm that PTRAS produces better renal salvage in RAS patients experiencing ARF, compared to medical management alone. These findings from our study call for larger prospective RCT where patients with RAS and ARF are randomized to PTRAS plus medical therapy vs medical therapy alone. It must however be acknowledged that PTRAS only opens up the main renal arteries, but without effect on renal arteriolar and/or capillary narrowing, which syndrome we had earlier termed microvascular RAS in a previous report. The debate rages on.

PHASE 2b/3 TRIAL DESIGN AND BASELINE PATIENT CHARACTERISTICS OF A STUDY TO DETERMINE EFFECTS OF BARDOXOLONE METHYL (BARD) IN PATIENTS WITH TYPE 2 DIABETES (T2DM) AND CHRONIC KIDNEY DISEASE (CKD)

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BARD is a novel agent that activates Nrf2, inducing the transcription of >250 genes that decrease ROS and down-regulate NF- κ B, angiotensin II, and other inflammatory mediators. BARD treatment improved eGFR (4-component MDRD) and other uremic solutes in patients (pts) with Stages 3/4 CKD and T2DM in randomized, open-label studies for 1 and 2 mos in 60 and 18 pts, respectively. The current study is the first blinded, placebo (pbo)-controlled trial to test the hypothesis that BARD improvements in renal function are sustained at 6 and 12 mos. Objectives are: 1^o- to assess the effect on eGFR in T2DM with CKD of 3 BARD doses relative to pbo at 24 wks and the safety and tolerability at 52 wks; 2^o- to assess BARD effect on eGFR at 52 wks. Design is multi-center, double-blind, randomized, pbo-controlled, parallel-group trial in pts with T2DM and CKD (eGFR 20 - 45 mL/min/1.73m²). Pts randomized to 4 groups: pbo, 25, 75 or 150 mg orally daily, and stratified on CKD (Stages 3 or 4), albuminuria (macro +/-) and HbA1c (< or \geq 7%). Pts undergo titration (\leq 20 wks), maintenance (to 52 wks) and then off treatment (28 days). Key inclusion criteria: >18 yrs old, known T2DM, ACE and/or ARB for at least 3 mos and stable dose for \geq 8 wks. Key exclusion criteria: non-T2DM CKD, HbA1c>10%, active CVD, BP>160/90, and hepatic or biliary disease. The study power is 90% with α =0.019 for N=220. 43 sites randomized 227 pts. Baseline characteristics are as follows:

Age (yr)	66 \pm 9	eGFR	32.4 \pm 6.9	ACE/ARB	98%
Male	56%	SCr	2.0 \pm 0.5	SBP	130 \pm 13
Af Amer, Hisp/Lat	19% 27%	Stage 4 CKD	41%	DBP	69 \pm 9
T2DM (yr)	18 \pm 10	ACR	680 \pm 1197	BMI	35.4 \pm 7.6

This rigorous trial is underway to assess the effect of BARD treatment on eGFR for 6 and 12 mos in pts with T2DM and CKD with baseline characteristics representative of the affected population.

FACTORS ASSOCIATED WITH QUALITY OF LIFE IN AFRICAN AMERICANS WITH CKD

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Health-related quality of life (HRQOL) has been evaluated in patients with ESRD but much less is known about HRQOL in patients with CKD, particularly among African Americans. We examined the relationship between HRQOL and demographic, clinical, and psychosocial factors in African Americans with hypertensive CKD with a cross sectional analysis of data drawn from the African American Study of Kidney Disease and Hypertension (AASK) Cohort Study. Quality of life was assessed using the Medical Outcomes Study Short Form (SF-36). Psychosocial factors were assessed utilizing Coping Strategies Inventory Short Form, Interpersonal Support Evaluation List-16, Satisfaction with Life Scale, and Beck Depression Inventory-II. GFR was estimated using the AASK Study equation.

Among 691 cohort participants, lower income and unemployment were associated with lower HRQOL scores ($p<0.01$, $p<0.05$, respectively). Medical co-morbidities were associated with lower physical component summary (PCS) scores ($p<0.01$) but not mental component (MCS) scores. While SF-36 MCS scores were similar across the spectrum of eGFR ($p=0.23$), SF-36 PCS scores were significantly lower in participants with an eGFR < 30 ml/min/1.73m² ($p<0.01$). Subjects with higher HRQOL scores, particularly MCS scores, reported higher social support, coping strategies, and satisfaction with life and lower depressive symptoms ($p < 0.005$).

In conclusion, in African Americans with hypertensive CKD, lower income and unemployment were associated with lower HRQOL. Comorbidities and eGFR <30 ml/min/1.73m² were associated with lower PCS scores. Higher HRQOL was associated with higher social support, coping strategies, and satisfaction with life and lower depressive symptoms.

**EFFECT OF CALCIUM ACETATE (CaAc) ON SERUM
PHOSPHORUS (P) LEVELS IN NONDIALYZED PATIENTS WITH
ADVANCED STAGES OF CHRONIC KIDNEY DISEASE (ND-CKD)**

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Hyperphosphatemia is associated with increased
mortality risk in ND-CKD. Purpose of this study
was to compare effects of CaAc vs. placebo on
control of serum P, iPTH, Ca x P and calcium in
hyperphosphatemic ND-CKD patients with stages 4
and 5.

This study was a prospective, multicenter,
randomized, double-blind, placebo-controlled
trial. Subjects enrolled if serum P >4.5mg/dL.
Following 6 week washout, 110 subjects randomly
allocated to CaAc or placebo for 3 months. Study
drug dose titrated to achieve P 2.7-4.5mg/dL.
Serum P, Ca, and iPTH measured at baseline and
biweekly. Primary efficacy measure is serum P at
12 weeks.

Baseline P did not differ between the groups.
By ITT analysis at 12 weeks, serum P was
significantly lower in CaAc compared to placebo
(4.4 ± 1.2 mg/dL vs. 5.1 ± 1.4 mg/dL; $p=0.04$), iPTH was
also lower in CaAc (150 ± 157 vs. 351 ± 292 pg/mL;
 $p<0.001$), Ca x P trend was lower in the CaAc but
adjusted Ca was higher in CaAc group (9.5 ± 0.8 vs.
 8.8 ± 0.8 mg/dL; $p<0.001$). Adverse events were
comparable.

CaAc was significantly more effective and as
safe in reducing serum P and iPTH levels compared
to placebo in CKD stage 4 and 5 nondialysis
patients.

PREVALENCE AND SIGNIFICANCE OF UNRECOGNIZED MYOCARDIAL INFARCTIONS IN CHRONIC KIDNEY DISEASE

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In the general population, unrecognized myocardial infarctions (UMI) are common and associated with an increased mortality risk. However, few data are available on the prevalence and clinical implications of UMI among adults with chronic kidney disease (CKD).

Participants (n=27,299) in the community-based REasons for Geographic and Racial Differences in Stroke study (REGARDS) completed a baseline examination that included questionnaires, labs, physical examination and an ECG. They have had a median follow-up of 4 years. UMI was defined as the presence of ECG findings suggestive of MI based on Minnesota Code criteria in the absence of self-reported MI (RMI). CKD stages were defined based on estimated glomerular filtration rate (using CKD-EPI equation) and albuminuria following Kidney Dialysis Outcome Quality Initiative guidelines.

The prevalence of UMI in the REGARDS population was 9% for those without CKD, and 9%, 11%, 10%, 11% for individuals with stage 1, 2, 3, and 4-5 CKD, respectively. Among those with any MI, the multivariable-adjusted odds ratios for UMI versus RMI decreased progressively with worsening CKD stages and were 0.85 (95% CI: 0.64 – 1.14), 0.82 (95% CI: 0.65 – 1.03), 0.70 (95% CI: 0.58 – 0.84), 0.55 (95% CI: 0.35 – 0.86) for stage 1, 2, 3, and 4-5 CKD versus no CKD respectively (p-trend < 0.001). Among all participants with CKD, the hazard ratios for mortality associated with UMI and RMI were 1.27 (95% CI: 1.01 - 1.60) and 1.58 (95% CI: 1.32 - 1.90), respectively.

Among individuals with an MI, those with CKD were less likely to have an UMI. However, in those with CKD, having an UMI was associated with a significantly increased risk for mortality.

CALCIPHYLAXIS CASE STUDY WITH RESPONSE TO SODIUM THIOSULFATE TREATMENT

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Calciophylaxis is a disorder characterized by medial arteriolar vascular calcification which results in subcutaneous necrosis with ischemic skin ulceration. Despite current treatment options, mortality can be as high as 87%. There are a few case reports, including this one, in which sodium thiosulfate (STS) was used in conjunction with more conventional treatments resulting in the resolution of necrotic skin lesions associated with calciophylaxis. Sodium thiosulfate is thought to help mobilize calcium from tissue in the form of calcium thiosulfate, which is 250-100,000 times more soluble than other calcium salts.

Our case report focuses on a 58 year old white female with a history of ESRD due to diabetes mellitus. She had received hemodialysis (HD) three times a week for the past two years. She presented with a single, painful, erythematous skin lesion and was initially treated with antibiotics and wound care for a presumed cellulitis. Her skin lesion became ulcerated with an eschar and she was soon diagnosed with calciophylaxis. While undergoing conventional treatment therapies her condition continued to worsen and she developed a second necrotic lesion on her right thigh. Conventional therapies included increased frequency of HD to five days a week with low calcium dialysate, hyperbaric oxygen therapy, pentoxifylline 300mg po daily and non-calcium phosphate binders. Treatment was then begun with STS 25 grams IV after HD five times a week and significant improvement was seen in both necrotic lesions within several weeks.

This case report demonstrates the improved outcome that may be seen in calciophylaxis when STS is included in the treatment regimen. Most current treatment strategies involve infection prevention and improved oxygenation of necrotic tissue but do little to correct the underlying pathology. More study is needed to determine the mechanism of action of sodium thiosulfate. Additionally, further investigation is needed to establish optimal dosing regimens, as dosing is currently based on treatment of other metastatic calcium disorders and not specifically calciophylaxis. With increased use and investigation, treatment of calciophylaxis with STS will hopefully improve morbidity and mortality in this deadly disease.

THE ASSOCIATION OF SERUM PHOSPHORUS AND PULSE PRESSURE IN MEN AND WOMEN WITH CHRONIC KIDNEY DISEASE: DATA FROM THE KIDNEY EARLY EVALUATION PROGRAM

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Background: Higher serum phosphorus is associated with CVD and the association may be stronger in men than women. **Methods:** A total of 6335 participants in the Kidney Early Evaluation Program (KEEP) with CKD (eGFR < 60 ml/min/1.73m²) at screening were included in the analysis. **Results:** Mean Pulse Pressure (PP) across increasing phosphorus quartiles for men was 60.8 ± 17.1, 60.5 ± 16.9, 58.5 ± 16.0, 60.9 ± 17.4 mmHg (p for trend = .09). Mean PP across increasing phosphorus quartiles for women was 60.2 ± 17.9, 60.1 ± 18.3, 60.4 ± 18.4, and 58.2 ± 17.6 (p for trend = .03) respectively. After multivariate adjustment, the gender difference persisted (p for gender interaction = .02). Among women with CKD, PP was 1.6 mmHg lower in the highest phosphorus quartile than in the other quartiles (95% CI: 0.3-2.8, p = .01) but similar to the other quartiles in men. **Conclusions:** Higher serum phosphorus is associated with lower pulse pressure in women but not men with CKD. Whether pulse pressure mediates gender differences of phosphorus and CVD requires further study.

COMPARISON OF AWARENESS OF CHRONIC KIDNEY DISEASE GUIDELINES AMONG PRIMARY CARE PHYSICIANS & RESIDENT PHYSICIANS: A NATIONAL SURVEY

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Since many patients with chronic kidney disease (CKD) are seen by primary care physicians (PCPs) initially, we performed survey to assess current PCPs & current resident physicians (RPs) or future PCPs awareness & knowledge of NKF KDOQI guidelines regarding CKD.

We performed both online & paper cross-sectional national survey. 354 RPs & 116 PCPs completed questionnaire. 57% RPs & 75% PCPs knew CKD definition and 3/4th RPs & PCPs knew CKD stages. Most of the RPs (89%) & PCPs (91%) would consider nephrology referral based on estimated glomerular filtration rate (eGFR) but half of RPs & PCPs would also look at serum creatinine level for referral. 61% RPs & half of PCPs would refer at eGFR 30-59 & 19% RPs & 1/3rd PCPs at eGFR 15-29. 2/3rd RPs & PCPs would use MDRD & Cockcroft-Gault equation while half of RPs & PCPs would also use serum creatinine to estimate kidney function. Almost all RPs & PCPs were aware of diabetes mellitus (DM) and hypertension as risk factors for CKD but only 38% RPs & half of PCPs knew smoking is a risk factor too. Majority of RPs & PCPs were aware that anemia, cardiovascular & bone diseases are complications of CKD but were less aware about other complications like stroke & malnutrition. 40% RPs & 46% PCPs would not assess for proteinuria in CKD patient with spot urine sample for protein to creatinine ratio but 60% RPs & 64% PCPs would assess with 24 hour urine collection for protein. 95% RPs & 97% PCPs chose angiotensin converting enzyme inhibitors as drug of choice for patients with Micro & Macroalbuminuria. 1/3rd RPs & PCPs were not aware of target HbA_{1c} is < 7% in patients with DM & CKD. 40% RPs & half of PCPs didn't know target blood pressure in patients with DM & CKD is < 130/80. 36% RPs & 17 % PCPs knew target Hb level is 11-12 g/dl in CKD patients. 14% RPs & 28% PCPs don't check vitamin D levels in CKD patients. 34% RPs & 24% PCPs check 1,25(OH)₂ vitamin D only.

Educational efforts are needed to raise awareness of NKF KDOQI guidelines among both current PCPs & current RPs or future PCPs.

OUTCOMES IN PATIENTS WITH AND WITHOUT PROTEINURIA IN A SINGLE CENTER HIV COHORT

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We sought to examine prevalence and incidence of proteinuria and outcomes of patients with and without proteinuria in a single center HIV cohort.

In a retrospective chart review analysis, data were collected from all patients who were referred to HIV clinic in the year 2005. Urinalyses on the initial HIV visit were used to determine the prevalence of proteinuria. Patients with trace proteinuria were not included as having proteinuria. Other data gathered included age, initial CD 4 count, and serum creatinine. In November 2009, charts of these patients were again reviewed. Study outcomes included development of ESRD and death.

Total number of patients was 203. 50 patients had proteinuria on initial visit. 22 patients developed proteinuria subsequently who did not have proteinuria on the initial visit. 6 patients had doubling of creatinine. Four developed ESRD. There were total of 27 deaths.

Proteinuria was found to be associated with higher mortality and development of ESRD in an unadjusted model. After adjustment with age, serum creatinine, and CD 4 count, association of proteinuria with mortality was found not to be significant although there was a trend towards higher mortality. Low CD 4 count was independently associated with higher mortality after adjustment with age, serum creatinine, and proteinuria.

Adjusted Odds Ratios for mortality:

Variable	Odds Ratios	Confidence Limits
Proteinuria on initial visit	1.446	0.569-3.677
Creatinine	1.127	0.895-1.420
Age	1.026	0.987-1.067
CD 4 count	0.997	0.995-0.999

In summary, proteinuria is highly prevalent in HIV population, and it may be associated with worse outcomes including development of ESRD and Death. Further study is warranted in examining outcomes in patients with HIV with proteinuria to determine best interventions to prevent or slow progression of renal damage.

ASPECTS OF IV FERUMOXYTOL ADMINISTRATION AND ACUTE ADVERSE EVENTS IN CKD PATIENTS

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Feraheme™ (ferumoxytol) Injection, a novel IV iron for the treatment of iron deficiency anemia in patients with CKD, is composed of an iron oxide core with a unique carbohydrate coating, is isotonic, and has a neutral pH, with evidence of lower free iron than other IV irons. One of the advantages of ferumoxytol is that it can be administered as a 510 mg rapid (<1 min) injection with a favorable tolerability profile. To investigate aspects of administering a 1.02 g course, we hypothesized that the rate of acute AEs (within 24 hours post dose) would not differ following the first and second injection, or differ by number of days between injections. We pooled data from 3 pivotal studies in 590 adult dialysis and non-dialysis CKD patients who received 2 x 510 mg ferumoxytol within 2-8 days. There was no meaningful difference in AEs following one (7.8%) vs two (4.8%) injections. There was no trend in AEs associated with how soon the second injection was given (2-4 days: range, 4.6% to 16.0%; 5-8 days: 10.5% to 22.2%); in fact, the shortest interval between doses (2 days) was associated with one of the lowest AE rates (6.5%). These results suggest that 510 mg ferumoxytol is well tolerated, irrespective of the number of injection or interval between injections.

UNDER-MEASUREMENT OF QUANTITATIVE URINARY PROTEIN IN STAGE 3 KIDNEY DISEASE

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Increased urinary protein has been shown to be an independent risk factor for progressive kidney and cardiovascular disease. NKF guidelines recommend quantitative urine protein (QUP) measurement for patients with chronic kidney disease (CKD) and/or diabetes. Studies evaluating provider compliance with urinary protein measurement, however, remain sparse. The purpose of the present study was to evaluate whether measurement of QUP was being performed by medical providers in stage 3 CKD patients in an academic medical center. Demographic information, serum creatinine and urinary protein measurements were extracted from the electronic clinical data warehouse for patients registered at the ambulatory clinic at Columbia University Medical Center (CUMC) from 2004 to 2007. Patients were designated as having Stage 3 CKD if their maximum MDRD-eGFR over a 4 year period was $< 60 \text{ ml/min/1.73m}^2$ and average eGFR between 30 and $60 \text{ ml/min/1.73m}^2$. Diabetic status was assigned from ICD9 coding. Patients were considered as having QUP measurement if either urine protein or albumin over creatinine ratios were present or if 24 hour urine protein or albumin measurements were present once over the 4 year period. 896 adults with chronic kidney disease were identified, of which 64% were male, 53% were diabetic and 29% were Black or Hispanic. 74% of diabetic CKD patients had QUP measurement compared to only 35% of non-diabetic CKD patients. eGFR was significantly higher in CKD patients without QUP than those with (46.7 vs 42.2 , $p=0.01$). Other factors such as age and gender were not associated with QUP measurement. In conclusion, a significant number of non-diabetic stage 3 CKD patients do not have quantitative urine protein measurement possibly secondary to higher eGFRs and under-recognition of disease. An electronic clinical decision support system may become an important tool in prompting recognition of these patients.

PARICALCITOL TREATMENT IN CKD PATIENTS WITH SECONDARY HYPERPARATHYROIDISM IS ASSOCIATED WITH BETTER HEALTH OUTCOMES WHEN COMPARED WITH NO VITAMIN D RECEPTOR [VDR] ACTIVATOR TREATMENT

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We aimed to assess the medication use, outpatient services, hospitalizations and costs in pre-dialysis CKD patients with SHPT receiving paricalcitol compared with no VDR activation treatment.

MedStat, I3 and IMS databases containing 2,524 patients with CKD and SHPT were analyzed to compare matched cohorts of patients treated with paricalcitol versus no VDR activator treatment. 1,262 patients per cohort were matched using propensity scoring of age, gender, Charlson Co-morbidity Index, and pre-index total cost. Multivariate analyses adjusting for baseline age, gender, CKD severity, insurance, physician specialty, region, pre-index co-morbidities, and pre-index costs were used to explore differences.

Multivariate analyses demonstrated lower all-cause and CV-related hospitalizations and total costs in paricalcitol cohort:

Paricalcitol vs No VDR Activator	Parameter Estimate	Utilization/ Cost Ratio	95% Confidence Interval	
Cardiovascular-Related Medication use	0.201	1.222	1.210	1.223
Cardiovascular-Related Outpatient visits	-0.001	0.999	0.989	1.009
Cardiovascular-Related Hospitalizations	-0.158	0.854	0.804	0.906
Cardiovascular-Related Total Costs	-0.095	0.909	0.856	0.966
All-Cause Medication use	0.174	1.191	1.184	1.197
All-Cause Outpatient visits	-0.055	0.947	0.940	0.953
All-Cause Hospitalizations	-0.221	0.802	0.760	0.846
All-Cause Total Costs	-0.097	0.907	0.867	0.950

Although medication use was higher in those receiving paricalcitol, this was offset by significantly fewer outpatient services and hospitalizations, as well as lower total costs compared with no VDR activator treatment. Further studies may be needed to confirm these real-world findings.

EFFECT OF NIACIN ON PHOSPHATE CONTROL IN CHRONIC KIDNEY DISEASE

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Hyperphosphatemia is a major cardiovascular risk factor in CKD. Niacin can control phosphate levels in dialysis patients; however its phosphate lowering effects on CKD patients are unknown. We conducted this retrospective study to determine if niacin is effective in lowering phosphorus (P) in CKD patients.

Charts of 332 CKD patients were reviewed. Data collected included age, gender, race, GFR (MDRD) niacin dose and duration of treatment, levels of P, cholesterol, HDL, LDL and triglycerides before and while on niacin, and concomitant phosphate binder usage. Paired t-test was used to compare values before and during niacin therapy. A p-value <0.05 was considered significant.

Thirty patients were on niacin; 4 patients were on phosphate-binders and were excluded; 18 patients had CKD3 and 8 had CKD4. Niacin doses ranged from 100-2000 mg/d with the majority on 500 (n=11) or 1000 (n=11). Phosphorus values before and during niacin treatment were available for 15 patients (9 CKD3 and 6 CKD4). In CKD4 mean P pre-niacin was 3.88mg/dL and while on niacin was 3.4 mg/dL (p=0.56). Corresponding values in CKD3 were 3.55 and 3.48 mg/dL respectively. Changes in P among all patients receiving 500 mg/day of niacin showed a mean increase of 0.05 mg/dL after starting niacin (n=8, p=0.61). Patients receiving 1000 mg/day showed a mean P decrease of 0.5 mg/dL (n=7, p=0.42).

Niacin caused a 0.48 mg/dL decrease in phosphorus in CKD4 despite having half of the patients on suboptimal doses of 500 mg/day. This did not reach statistical significance as the study sample was small. Prospective studies using niacin dose of at least 1000 mg/day are needed.

CKD-EPI EQUATION IS MORE ACCURATE THAN MDRD-EQUATION IN A MULTI-ETHNIC ASIAN POPULATION

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The Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation is reportedly more accurate than the MDRD GFR-estimating equation for a wider GFR range. We compared the performance of the CKD-EPI and MDRD equations in a multi-ethnic Asian population.

We prospectively recruited 232 chronic kidney disease patients (52% male, Chinese 40.5%, Malay 32%, Indian and others 27.5%). We measured standardized serum creatinine by an enzymatic method. GFR was estimated (eGFR) using “white” or “other” ethnicity with CKD-EPI (Levey AS et al. Ann Intern Med 2009:150,604) or re-expressed MDRD: $175 \times \text{Cr}^{-1.154} \times \text{Age}^{-0.203} \times 0.742$ (if female). We measured glomerular filtration rate (mGFR) using 3-sample plasma clearance of $^{99\text{m}}\text{Tc-DTPA}$, calculated by the slope-intercept method, with body surface area normalization (du Bois) and Brochner-Mortenson correction. eGFR was compared overall and >60 & <60 mL/min/1.73m² of mGFR, and accuracy by percentage of estimates to within 30% of mGFR and by root mean square error (RMSE). We defined bias as the median difference between eGFR and mGFR, and precision as the inter-quartile range for the differences.

Population means: age 58.4 ± 12.8 years, creatinine 1.73 ± 1.04 mg/dL, measured GFR 51.7 ± 27.5 mL/min/1.73m².

Group (n)		All (232)	GFR<60(160)	GFR>60(72)
Bias	CKD.EPI	-1.23	-1.46	0.88
	MDRD	-3.0	-2.4	-5.3
Precision	CKD.EPI	12.1	9.3	22.0
	MDRD	12.2	9.2	18.3
Accuracy (%)	CKD.EPI	82.8	78.8	91.7
	MDRD	79.7	78.8	81.9
RMSE	CKD.EPI	13.9	12.8	15.3
	MDRD	15.2	12.6	19.8

The CKD-EPI-equation is more accurate than the MDRD equation. The bias and accuracy are improved especially at higher GFR.

ETHNIC COEFFICIENTS ARE NOT REQUIRED FOR THE MDRD-EQUATION IN A MULTI-ETHNIC ASIAN POPULATION

Boon Wee Teo, Hui Xu, Borys Shuter, Danhua Wang, Jialiang Li, Arvind Kumar Sinha, Sunil Sethi, Evan Lee; National University Health System, Singapore

Practice guidelines recommend the MDRD GFR-estimating equation (eGFR) to classify chronic kidney disease (CKD). We evaluated the performance of the MDRD-equation in a multi-ethnic Asian population and estimated ethnic coefficients.

We prospectively recruited 232 CKD patients (52% male, Chinese 40.5%, Malay 32%, Indian and others 27.5%) by gender, ethnicity, and eGFR. We measured standardized serum creatinine by an enzymatic method and calculated eGFR with re-expressed MDRD: $175 \times \text{Cr}^{-1.154} \times \text{Age}^{-0.203} \times 0.742$ (if female). We measured glomerular filtration rate (mGFR) using 3-sample plasma clearance of $^{99\text{m}}\text{Tc-DTPA}$, calculated by the slope-intercept method, with body surface area normalization (du Bois) and Brochner-Mortenson correction. eGFR was compared overall, by ethnicity, and >60 & <60 mL/min/1.73m² of mGFR, and accuracy to within 15%, 30%, and 50% of mGFR, and mean bias \pm SD (mL/min/1.73m²). We forced all MDRD coefficients to our mGFR to obtain the fitted ethnic coefficients.

Population means: age 58.4 ± 12.8 years, creatinine 1.73 ± 1.04 mg/dL, measured GFR 51.7 ± 27.5 mL/min/1.73m².

Group (n)	Estimated ethnic coeff. (CI)	Accuracy (%)	Mean bias \pm SD
All (232)	1.09 (1.05-1.12)	50.5, 79.7, 95.3	0.95 \pm 15.2
>60 (72)	NA	52.9, 81.9, 98.6	-2.26 \pm 19.9
<60 (160)	NA	48.8, 78.8, 93.6	-0.37 \pm 12.6
Chinese (94)	1.14 (1.08-1.19)	53.2, 77.7, 97.9	-3.87 \pm 13.3
Malay (74)	1.07 (1.01-1.13)	52.7, 83.8, 94.6	-1.43 \pm 11.7
Indian+ (64)	1.03 (0.96-1.10)	42.2, 78.1, 92.2	-3.88 \pm 19.8

The MDRD-equation performed with similar accuracy in a multi-ethnic Asian population. We suggest that for this population, ethnic coefficients modifying the MDRD-equation are not required since the differences are small and less than differences attributable to methodology, derivation population, and natural variability.

EARLY AND LATE NATIVE KIDNEY BIOPSIES IN ORTHOTOPIC LIVER-ALONE TRANSPLANT (OLT) RECIPIENTS

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CKD is a recognized complication of OLT. Kidney biopsy (KB) not only establishes the morphological diagnosis but is able to reveal underlying mechanisms of the disease and provide guidance for further management.

The study population included patients who underwent native KB in 1996-2006 after the OLT. A total of 1058 OLT were done during this time period. 33 patients (3.12%) were identified for the study: 3 of those underwent an early (EB) (< 3 months) and 30 - a late (LB) (> 3 months) post-OLT KB. We analyzed the timing, indications, morphological findings and complications of the renal biopsies. We also analyzed kidney function before OLT and at the time of KB and then followed this cohort of patients for 1 year after the biopsy to identify kidney function and patient outcomes.

KB in EB group was done in 3 cases for AKI with hematuria and/or proteinuria and revealed IgA nephropathy in 2 cases and acute interstitial nephritis in 1 case. In LB group indications for KB were: progressive CKD in 9 (30%), proteinuria in 6 (20%), AKI in 7 (23.3%) and renal mass in 8 (26.7%) patients. Morphologic findings in LB group included FSGS in 11 (36.7%), chronic tubulo-interstitial changes in 10 (33.3%), calcineurin inhibitor toxicity in 9 (30%), malignancy in 7 (23.3%), vascular sclerosis in 7 (23.3%), focal global glomerulosclerosis in 4 (13.3%), IgA nephropathy in 4 (13.3%) and diabetic nephropathy in 3 (10%) patients. Membranoproliferative glomerulonephritis and ATN were found in 2 cases each and 1 more case showed chronic interstitial nephritis. 20 of 33 patients had normal renal function at the time of OLT. In LB average time to KB was 8.3 (range 0.4-17.4) years. All patients in EB demonstrated improvement in kidney function that extended beyond 1 year following KB. In LB group improvement was observed in 15 patients at 6 month and extended beyond 1 year in 11 of them. 10 patients progressed to ESRD within 1.4 (range 0-3.5) years after KB. No significant complications of the KB were observed.

KB is valuable and safe tool in management OLT recipients who developed kidney disease.

AUTOMATED REPORTING OF ESTIMATED GFR ALTERS REFERRAL PATTERNS TO A NEPHROLOGY CLINIC

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An estimated 11-25% of the United States adult population has chronic kidney disease (CKD). Historically, elevated serum creatinine concentration (S-cr) has been the primary laboratory indicator of CKD, but it has been shown to be insensitive. In 2002, the National Kidney Foundation published a 5-stage classification of CKD based on estimated glomerular filtration rate (eGFR), calculated from a four-variable formula adapted from the Modification of Diet in Renal Disease (MDRD) trial. Subsequently, laboratories have increasingly reported eGFR. We hypothesized that, in populations with modestly elevated S-cr, eGFR reporting has improved detection of CKD and increased referrals to the outpatient nephrology clinic.

We performed a retrospective audit of S-cr and eGFR of new referrals to a university-based outpatient nephrology clinic 12 months before and 36 months after the institution of eGFR reporting. The patient population is 40% Caucasian and 60% African American in an urban setting with a mix of private insurance and public funding.

A total of 1780 patients were referred to the nephrology clinic from January 1 2005 through December 31 2008. The proportion of referrals identifying earlier stage CKD increased significantly for 2 years following the institution of eGFR reporting but was not sustained ($p = 0.018$). Referrals with eGFR greater than 45 ml/min increased from 48% of all referrals in 2005 to 57% in 2006 and 56% in 2007, returning to 49% in 2008. The proportion of referrals in African-American women with earlier stage disease increased significantly in the first year only with 69% in 2005 and 80% in 2006 ($p = 0.043$). No significant trend was seen with respect to age.

Institution of automated eGFR reporting resulted in an increased but non-sustained proportion of patients referred with earlier stages of CKD. Additionally, the proportion of African-American women referred at earlier stages increased initially. This may be explained by increased diagnosis of patients with early stage disease with no change in disease prevalence, suggesting automated eGFR reporting will not cause sustained or overwhelming increase in nephrology referrals.

CKD AND OLDER ADULTS: A REVIEW AND IMPLICATIONS FOR SOCIAL WORK PRACTICE AND RESEARCH

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Purpose: The elderly are the fastest growing segment of the ESKD population. By the year 2030, older adults will constitute well over half of persons living with CKD. In light of the increased prevalence of older persons in all stages of CKD, social work interventions must apply to older adults. The purpose of this review is to address two important questions: (1) to what extent do social work journals contain research about older adults with CKD?; and (2) does the research offer implications for social work practice and research?

Methods: Articles published between the years 1998 and 2008 were reviewed. Scholarly articles were selected from health-related social work journals: Health & Social Work, Journal of Nephrology Social Work, Social Work in Health Care; and The Journal of Gerontological Social Work. Table of contents and abstracts were reviewed for aging and kidney content using the search terms, “renal”, “nephrology”, “kidney”, “aging”, “elderly”, “older”, “gerontology”, and “geriatrics”. An article met the criteria if the content was research based and addressed implications for social work practice with older adults in dialysis and transplant patients or implications for research on older adults with CKD. .

Results: Close to 1,000 articles were reviewed. Of those, 4 articles met the review criteria. These articles addressed practice implications such as the need for psychosocial evaluation, assessment, and education. Implications for research were provided in 1 abstract.

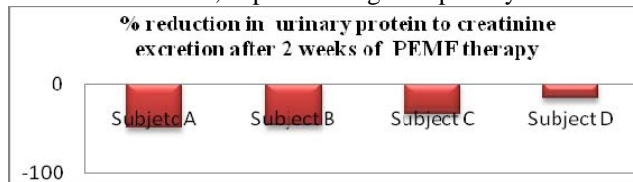
Conclusion: There is a remarkable gap in research with older adults with kidney disease. The majority of health and aging social work research focuses on persons with cancer, diabetes, and HIV/AIDS. Geriatric kidney patients experience visual and hearing impairment, malnutrition, cognitive impairments, urinary incontinence, and limited functional status and psychosocial issues including lack of social support, economic hardships, and isolation. Social work interventions on self-care, functioning, and quality of life are critical to the well-being of a burgeoning high risk and vulnerable population.

EFFECTS OF PULSED ELECTROMAGNETIC FIELD THERAPY (PEMF) ON REDUCING PROTEINURIA (P) IN CKD

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Our laboratory demonstrated that the use of supramaximal doses of angiotensin receptor blockers (ARBs) reduced P independent of BP. We investigated the effectiveness of PEMF during a 2 week trial on reducing (P) in subjects with CKD, evaluating for synergy between PEMF and ARBs. PEMF has inherent anti-inflammatory and anti-fibrotic properties that modulate the calcium calmodulin-dependent nitric oxide and cGMP signaling pathways.

Four volunteers with progressive proteinuric nephropathies applied PEMF to their lower-thoracic spine, allowing electromagnetic energy to pulse over both kidneys for 30 min, 3 times a day for 2 weeks. All medications were continued without change, including previously prescribed ARB's. Urinary spot collections were analyzed for protein to creatinine ratio's, expressed in grams per day.



During a two week observational trial the application of PEMF demonstrated reductions in protein to creatinine ratio's expressed on urinary spot collections. Students paired t- test demonstrated in the four subjects, $p = 0.06$. There were no significant changes in the glomerular filtration rate (MDRD) or mean arterial pressures. No adverse events were reported. The reduction in proteinuria over 2 weeks was arithmetically, but not statistically significant due to small population size. This reduction in proteinuria warrants further study to determine long term effectiveness and possible synergy with RAS blockade.

BARDOXOLONE METHYL (BARD) INHIBITS INFLAMMATORY SIGNALING IN CULTURED MESANGIAL CELLS

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Diabetic nephropathy is the major cause of end-stage renal disease. Pro-inflammatory stimuli including hyperglycemia and chronic activation of the renin-angiotensin system (RAS) increase oxidative stress in the kidney. Activation of the Keap1-Nrf2 pathway suppresses oxidative stress and inhibits inflammatory signaling. BARD, the lead molecule from the Antioxidant Inflammation Modulator (AIM) drug class, is among the most potent inducers of Nrf2 activity and improves renal function in type 2 diabetics with chronic kidney disease (CKD). The objective of this study was to investigate the effects of BARD on inflammatory signaling in cultured mesangial cells. To assess Keap1-Nrf2 activation, several mesangial cell lines were treated with a range of BARD concentrations and the mRNA levels of various Nrf2 target genes were measured using quantitative RT-PCR. The effect of BARD on NF- κ B signaling was assessed in mesangial cells treated with TNF α or bovine serum albumin (BSA) by monitoring phosphorylation of I κ B by western blot. Mesangial cells were also stimulated with angiotensin II to assess the effect of BARD on mesangial cell contraction. In addition, the dose- and time-dependence of BARD effects on mesangial cell gene expression are being profiled using cDNA microarrays. We found that BARD induced expression of Nrf2 target genes in all cell lines at concentrations as low as 10nM. Also, NF- κ B signaling induced by TNF α or BSA was suppressed by BARD. A trend toward less mesangial cell contraction induced by angiotensin II was observed in cultures treated with BARD at concentrations as low as 50nM. The inhibitory effect reached statistical significance at 250 and 500nM BARD. The results from this study demonstrate that BARD potently induces Nrf2 activation and suppresses inflammatory signaling in mesangial cells and support the evaluation of this molecule in renal indications where RAS activation, oxidative stress, and inflammation contribute to the pathology. A 12-month randomized, blinded, placebo-controlled Phase 2b/3 study in CKD patients is currently underway.

CHRONIC KIDNEY DISEASE AWARENESS IS LIMITED IN PATIENTS SEEN BY NEPHROLOGISTS

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Purpose: Patient awareness of chronic kidney disease (CKD) is an important factor in the execution of successful disease self-care. Studies have shown low awareness of CKD in the general population, even in those who have seen a primary care physician in the past year. The aim of this study is to describe awareness of CKD diagnosis in patients seen by nephrologists, and characterize associations of level of awareness.

Methods: Four-hundred established adult patients with CKD (Stages 1-5), seen at least once previously in the Nephrology clinic, were enrolled from May 2009 to October 2009. They were asked about awareness of their diagnosis ("Do you have chronic kidney disease? Yes/No") and rated their perceived disease specific knowledge in nine areas on a scale of 1-4 (1=No knowledge, 2=A little knowledge, 3=A good amount of knowledge, and 4=A lot of knowledge). Measurements included demographics, visit information, and laboratory values. Health literacy was assessed with the Rapid Estimate of Adult Literacy in Medicine (REALM) survey.

Results: The mean age of this cohort was 56.7 years (SD 15.8), 83% were White, and 76% had CKD stage 3-5. Low literacy (<9th grade level) occurred in 18%. 28% were unaware they had CKD. 45% of stage 1-2 patients were unaware vs. 22% of those stage 3-5 ($p=0.01$). Low awareness was associated with increased eGFR [$\rho=-0.32$; $p=0.01$], lack of attendance in a kidney education class [no attendance 36% unaware vs. attendance 19% unaware; $p=0.01$], less provider visits within the past year [≤ 2 visits 36% unaware, ≥ 3 visits 22% unaware; $p=0.01$], and lower overall perceived knowledge [2.36 (0.51) vs. 2.71(0.61); $p=0.01$]. Sex, race, age, and REALM scores were not associated with awareness of CKD in this cohort.

Conclusion: Awareness of CKD is limited even in patients seen by a nephrologist. Early and repeated communication regarding CKD may improve patient awareness of their diagnosis. Further study is needed to determine the impact of CKD awareness on self-care behaviors and clinical outcomes.

AN ETHNIC CHINESE COEFFICIENT FOR THE RE-EXPRESSED MDRD EQUATION USING STANDARDIZED CREATININE

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The MDRD glomerular filtration rate-estimating equation (eGFR) was modified with very different ethnic coefficients for ethnic Chinese (1.23) (Ma et al. JASN 2006:17,2937) and ethnic Japanese (0.81)(AJKD 2009:53,982) . We evaluated the validity of the Chinese coefficient using the same GFR measurement method as Ma et al.

To show a 20% difference between measured GFR (mGFR) and eGFR, we need >89 patients (Dupont et al; Controlled Clinical Trials 19: 589). We prospectively recruited 94 Chinese chronic kidney disease patients (51% male), and measured GFR using 3-sample plasma clearance of ^{99m}Tc -DTPA at 2, 3.5, and 5 hours, calculated by the slope-intercept method, with body surface area normalization (du Bois) and Brochner-Mortenson correction. We measured standardized serum creatinine by an enzymatic method and calculated eGFR with the re-expressed MDRD: $175 \times \text{Cr}^{-1.154} \times \text{Age}^{-0.203} \times 0.742$ (if female). To determine if methodology was a cause of the ethnic coefficient, we also calculated measured GFR as described by Ma et al with 2-samples. To simulate the MDRD population, we confined our sample (n=53) to mGFR <70 and age <70 years, bootstrap 50 random samples (n=60) to estimate the Chinese coefficient. We forced all MDRD coefficients to our mGFR to obtain the fitted Chinese coefficients.

Population means: age 58.1 ± 13.5 years, creatinine 1.70 ± 0.92 mg/dL measured GFR 54.2 ± 28.7 mL/min/1.73m².

Group (n)	mGFR method	B-M correction	Estimated ethnic coeff. (CI)
All (94)	3-samples	Yes	1.14 (1.08-1.19)
All (94)	2-samples	No	1.23 (1.17-1.29)
Reduced (53)	3-samples	Yes	1.15 (1.08-1.22)

Using standardized creatinine and Brochner-Mortenson correction, the Chinese coefficient for the MDRD equation is 1.14, and is significantly different from the previously reported Chinese coefficient of 1.23. A significant portion of the coefficient was contributed by the method of calculating and correcting measured GFR.

STANDARDIZED SERUM CREATININE WITH CYSTATIN C IMPROVES ACCURACY OF GFR ESTIMATES IN ASIANS

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We evaluated the performance of glomerular filtration rate estimating-equations (eGFR) with standardized serum creatinine (SCr) alone, serum cystatin C (cysC) alone, and in combination with demographic variables in a multi-ethnic Asian population.

We prospectively recruited 232 CKD patients (52% male, Chinese 40.5%, Malay 32%, Indian and others 27.5%) by gender, ethnicity, and eGFR. We measured SCr by enzymatic method and cystatin C by nephelometry. eGFR was calculated for SCr alone with revised MDRD: $175 \times \text{SCr}^{-1.154} \times \text{Age}^{-0.203} \times 0.742$ (if female); cystatin C alone and in combination from Stevens et al (AJKD 2008:51,395). No adjustment for ethnicity was made. We measured glomerular filtration rate (mGFR) using 3-sample plasma clearance of ^{99m}Tc -DTPA, calculated by the slope-intercept method, with body surface area normalization (du Bois) and Brochner-Mortenson correction. eGFR was compared overall and by ethnicity, and accuracy to within 15%, 30%, and 50% of mGFR, and mean bias \pm SD (mL/min/1.73m²).

Population means: age 58.4 ± 12.8 years, SCr 1.73 ± 1.04 mg/dL, cysC 1.48 ± 0.69 , mGFR 51.7 ± 27.5 mL/min/1.73m².

Group (n)	Equation type	Accuracy (%)	Mean bias \pm SD
All (232)	MDRD	50.5, 79.7, 95.3	0.95 \pm 15.2
All (232)	cysC	40.5, 64.2, 84.9	11.1 \pm 16.2
All (232)	cysC+demo	48.3, 74.1, 90.5	7.83 \pm 14.9
All (232)	cysC+SCr+demo	59.1, 84.5, 95.7	4.51 \pm 14.2
Chinese	cysC+SCr+demo	64.9, 88.3, 95.7	3.43 \pm 14.2
Malay	cysC+SCr+demo	54.1, 87.8, 94.6	2.67 \pm 13.3
Indian+	cysC+SCr+demo	56.3, 75.0, 96.9	8.23 \pm 15.0

Cystatin C alone for estimating GFR was no better than standardized creatinine alone. Performance was improved with both in combination with demographic variables but bias was noted. More research in ethnic variation of cystatin C is required.

THREE YEARS EXPERIENCES OF JAPANESE VERSION OF KIDNEY EARLY EVALUATION PROGRAM (KEEP JAPAN)

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The International Kidney Evaluation Association Japan (IKEAJ) started the Japanese version of Kidney Early Evaluation Program (KEEP JAPAN) following the US National Kidney Foundation since 2006.

The 1334 participants with diabetes or hypertension, or family history of diabetes, hypertension, or kidney disease (KEEP group) and 453 participants without above risk factors (non-KEEP group) were included. Overall, mean age was 55.5 ± 17.3 years; 831 were men and 956 were women. Of them, 811 and 508 participants were examined in the second and third year, respectively.

Of KEEP group, CKD prevalence was 24.3%, defined by positive albumin-creatinine ratio (\geq or > 30 mg/gCr) and estimated glomerular filtration rate. In contrast, of non-KEEP group, the prevalence was 11.7%. In KEEP group, 24.3% and 54.5% self-reported diabetes and hypertension, respectively; 77.3% had a family history of diabetes, hypertension, or kidney disease. In non-KEEP group, although none had self-reported hypertension, 299 participants (48.5%) had high blood pressure and 15.1% of them had CKD, 3.1 odds ratio vs. normal blood pressure.

In KEEP group, CKD prevalence in older participants (> 65 years) was 38.2%; odds ratio vs. younger participants, 2.6. Also in non-KEEP group, CKD prevalence was higher in older participants (30.6%, odds ratio 3.5). It is to note that the prevalence of CKD among smoking participants in KEEP group (16.6%) was lower than non-smoking participants. The prevalence of CKD among obese participants in non-KEEP group (8.5%) was significantly higher than non-obese participants. From annual follow-up, the incidence of CKD in KEEP group was 8.6-13.1%, besides that in non-KEEP group was 2.4-2.9%. However, until third year follow-up examination, only 20.8% of participants firstly enrolled to CKD were enrolled to CKD in the other second and third examinations in KEEP group. Furthermore, in non-KEEP group, this rate was much lower (6.6%).

In conclusion, the prevalence and yearly incidence of was high in KEEP group compared with general Japanese population. Age, smoking and obesity should be other risk factors for the CKD.

ROLE OF SUBCLINICAL INFLAMMATION IN EARLY DIABETIC NEPHROPATHY

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The study is designed to investigate the role of subclinical inflammation in pathogenesis of early diabetic nephropathy. We hypothesized that urinary albumin excretion in patients with type 2 Diabetes Mellitus (Type 2 DM) might be related to markers of chronic inflammation such as high-sensitivity C-reactive protein (HS-CRP). Fifty Type 2 DM patients were enrolled in the study and categorized as normoalbuminuric or microalbuminuric. Urinary albumin excretion (UAE) was estimated as spot urine microalbumin/creatinine ratio. Fifteen patients with no Type 2 were taken as control. We measured HS-CRP in all of the above patients and its correlation with proteinuria was studied.

We observed that those with microalbuminuria had greater concentrations of inflammatory parameters than normoalbuminuric patients with DM. The mean levels of HS-CRP were 1.30 ± 0.32 mg/L, $2.60 \pm .73$ mg/L and 4.90 ± 1.80 mg/L, in the control, normoalbuminuric and microalbuminuric groups respectively ($P < .001$).

Taking the 50 diabetic patients as one group, a correlation coefficient of 0.813 ($P < .001$) was found between UAE and levels of HS-CRP. The level of HbA1c at baseline showed a significant positive correlation with UAE ($r = 0.592$, $P < .001$) and HS-CRP ($r = 0.732$, $P < .001$) in the diabetic population. After adjusting for the effect of other variables like duration of diabetes, Blood pressure, HbA1c and Serum creatinine, the previous association between UAE and the levels of HS-CRP remained significant ($P < .001$).

The significant association between inflammatory parameters and UAE indicates that inflammation may be a pathogenetic mechanism of diabetic nephropathy. Further analyses are necessary to confirm the intrarenal production and implication of inflammation in the pathogenesis of diabetic nephropathy.

FACTORS IN THE PROGRESSION OF DIABETIC NEPHROPATHY AND ITS COMPLICATIONS: A SINGLE CENTER EXPERIENCE IN SAUDI ARABIA

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To investigate the confounding factors related to the progression of diabetic nephropathy (DN) to end-stage renal disease (ESRD) and its associated complications.

A single hospital-based retrospective analysis of type 2 diabetics seen between January 1989 and January 2004 at Security Forces Hospital, Riyadh, Saudi Arabia was conducted. DN was diagnosed by presence of persistent proteinuria on 3 consecutive readings/year and/or serum creatinine $>130 \mu\text{mol/L}$ and/or GFR $<60 \text{ mL/min/1.73m}^2$.

Of 1952 type 2 diabetic files reviewed, 621 (31.8%) had DN, 294 (47%) were males. Mean age was 66.9 ± 11.4 years and mean duration of diabetes was 15.4 ± 7.5 years. Glomerular Filtration Rate deteriorated from mean baseline GFR of $78.3 \pm 30.3 \text{ mL/min/1.73m}^2$ to $45.1 \pm 24.1 \text{ mL/min/1.73m}^2$ at last visit with mean yearly GFR drop of 3.3 mL/year . Progression of DN was seen in 455 (73.3%) patients, 250 patients (40.3%) had doubling of serum creatinine in 9.98 ± 6.04 follow-up years. At the end of study, 75 (16.5%) patients reached ESRD and were eventually dialyzed. High rates of cataract, retinopathy, stroke and cardiovascular complications were seen with progression of DN. High baseline GFR $> 90 \text{ mL/min/1.73m}^2$, duration of diabetes > 10 years, persistent proteinuria, systolic BP $>130 \text{ mmHg}$ and presence of retinopathy were significantly associated with progression of DN.

Progression of DN to ESRD among Saudis is progressive with doubling of serum creatinine and high GFR decline rate of 3.3 mL/year . High baseline GFR, diabetes of >10 years, persistent proteinuria, SBP $>130 \text{ mmHg}$ and presence of retinopathy were significant predictors associated with progression of nephropathy.

NOVEL ANTIOXIDANTS BLOCK HIGH GLUCOSE-INDUCED TUBULAR HYPERTROPHY IN PROXIMAL TUBULE EPITHELIAL CELLS

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Diabetic nephropathy (DN) is the leading cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) worldwide. DN is characterized by glomerular remodeling and tubulointerstitial fibrosis (TIF), which best predicts progression to ESRD. Although current therapies have significantly improved renal function, the purpose of this study is to identify novel treatment options that may contribute to better outcomes in DN progression, and to elucidate their mechanisms of action. Using MTT cell-based viability assays, time- and dose-dependent studies, and immunoblotting analysis, we have shown that two novel antioxidants, F1—a glutathione precursor and F2-N-acetyl cysteine, are potent inhibitors of high glucose-induced tubular hypertrophy in human proximal tubule epithelial HKC-8 cells. Antioxidants F1 and F2 increased cell viability and blocked high glucose inhibitory effects on HKC-8 cell viability. F1 and F2 abolished high glucose activation of the mitogen-activated protein kinase (MAPK) p44/42 and JAK/STAT pathways. To investigate F1 and F2 effects on the oxidative stress content of HKC-8 cells cultured in high glucose, we showed that F1 and F2 reversed high glucose activation of subunits of the NADPH oxidase complex, namely p47^{phox}, p67^{phox}, and Nox-4 in a time-dependent manner, suggesting that these antioxidants also block oxidative stress-sensitive pathways. F1 and F2 also blocked TGF- β protein expression, a key hypertrophic and fibrotic factor, in HKC-8 cells. Together, our data reveal that these two novel antioxidants are potential therapeutic targets for DN, that exert their function by altering key stress-sensitive and TGF- β pathways associated with the development of tubular hypertrophy, and subsequent TIF in DN progression. Future studies will investigate the effectiveness of these novel antioxidants to abrogate or reverse TIF in experimental DN animal models, and to improve CKD outcomes in clinical trials.

IN-VIVO ANALYSIS OF GLOMERULAR AND PROXIMAL TUBULE HANDLING OF AGE-MODIFIED ALBUMIN USING TWO-PHOTON MICROSCOPY

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Advanced glycation end product (AGE) modification of tissue and circulating proteins has been shown as a strong marker of complication in Diabetes. However, AGE compounds derived from the Dicarbonyl α -oxaldehyde Methylglyoxal (MGO) are closely associated with hyperglycemia, poor control of diabetes and increased susceptibility to diabetic nephropathy.

Previous studies indicate that proximal tubule cells may be the site of catabolism of AGE proteins. However, little is known about the glomerular and proximal tubule handling of these glycated albumins in live kidney tissue. Therefore, this study explores the hypothesis that there is no difference in glomerular handling of AGE-modified albumin, but proximal tubule cells process AGE-modified albumin differently compared to un-glycated albumin. To directly quantify the *in vivo* processing of albumin filtration and proximal tubule handling of the AGE-modified albumin, two-photon microscopy of Munich Wistar rat kidney following infusion of Texas red albumins (modified and un-modified) was investigated.

Similar glomerular permeability was found between AGE-modified albumin and un-glycated albumin. However, AGE-modified albumin showed a significant reduction in plasma fluorescence at 2 and 24 hrs compared to un-glycated albumin. These differences indicate that it is not the glomerular handling, but protein handling and trafficking by the renal proximal tubule of the AGE-modified proteins that is affected. This may confirm a link between the pathogenic role of AGEs and early proximal tubule functional changes seen leading to hypertrophy and nephropathy in diabetes.

DECREASED PODOCYTE-SPECIFIC PROTEIN EXPRESSION CORRELATES WITH SEVERITY OF DIABETIC NEPHROPATHY

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Podocytes are injured and lost in diabetic nephropathy (**DN**), whether by detachment or apoptosis. Our goal is to correlate the degree of podocyte loss with severity of renal disease, and to show whether the mechanism of loss is via apoptosis.

Podocyte-specific proteins (**PSPs**) synaptopodin (**S**), podocin (**P**) and nephrin (**N**) expression were evaluated by immunohistochemistry (**IHC**) on **DN** (study) and non-diabetic (control) human renal biopsies. Expression was quantified as a % of positive staining over entire glomerular area (Image J, NIH). TUNEL staining was used for detection of apoptosis. Statistical analysis was performed using Mann-Whitney test and Spearman rank correlations.

Seventeen **DN** and 5 controls were analyzed. Controls for **S** and **P** included IgA nephropathy (1), minimal change disease (2), membranous nephropathy (1), and normal kidney (**NK**) (1); **NK** also served as control for **N**. There was a statistically significant decrease in the expression of **S** (p .004) and **P** (p .014) in **DN** compared to controls. There was, however, complete loss of **N** expression in **DN** as compared to control, an unexpected finding. **S** and **P** expression negatively correlated with degree of proteinuria (rho -0.32, -0.05), serum creatinine (rho -0.32, -0.28), systolic blood pressure (rho -0.23, -0.38) and serum BUN (rho -0.35,-0.03) respectively. TUNEL staining was negative for all **DN** biopsies.

In conclusion, we have demonstrated that decreased **S** and **P** expressions reflect the severity of renal disease, while **N** expression is completely lost irrespective of disease activity. These findings support the use of selective **PSPs** as markers for renal disease in **DN**, and **N** as the earliest marker. The negative TUNEL staining suggests that the dominant mechanism of podocyte loss is likely detachment rather than apoptosis, especially since urinary podocytes have been found in **DN** patients. We will verify the utility of these markers in fresh urine from **DN** patients.

URINARY EXCRETION OF PODOCYTES AS AN EARLY MARKER OF DIABETIC NEPHROPATHY

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Pathogenesis of diabetic nephropathy (DN) is related to podocyte injury and loss. Podocytes, which are located on the outer surface of the glomerular basement membrane, appear in the urine as a result of detachment. Microalbuminuria has been traditionally used to detect the onset of DN; its appearance usually prompts aggressive treatment. The aim of this study is to determine whether the podocytes are lost in diabetic patients even before the appearance of microalbuminuria, and that it may serve as an earlier marker of DN.

Twelve diabetic patients (9 patients with normoalbuminuria, 2 patients with microalbuminuria and 1 patient with macroalbuminuria) and 9 healthy controls were studied. Urinary cell pellet was obtained via centrifugation from fresh urine. The pellet was cytospun for immunofluorescence. Urinary podocytes were identified by co-localization of the podocyte specific markers nephrin and podocin, as well as synaptopodin and podocin. The podocytes-to-creatinine ratio was calculated for semiquantification of podocyturia.

We found that urinary podocytes were absent in the healthy controls, as expected. On the other hand, all diabetic patients, even those with normoalbuminuria, demonstrated presence of urinary podocytes. The mean podocyte-to-creatinine ratio in the diabetic patients with normoalbuminuria was 1222.5 podocytes/mg creatinine, as compared with 119.3 podocytes/mg creatinine in the microalbuminuria group. The degree of podocyturia did not correlate with the amount of albuminuria.

In conclusion, we found significant podocyturia in normoalbuminuric diabetic patients, revealing podocyte injury in this presumed "unaffected" population. Thus, we believe that podocyturia is an earlier marker of DN than microalbuminuria, and that it may be a better predictor of progression, since once lost, podocytes do not regenerate. Given our small sample size, we will need to validate our results in larger group, and to follow these patients prospectively for renal outcomes.

IMPACT OF IMPAIRED eGFR ON HEMOGLOBIN A1C AND AVERAGE GLUCOSE CORRELATION IN DIABETES MELLITUS

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The optimal target for glycemic control in diabetic Chronic Kidney Disease (CKD) has not been established, partly because hemoglobin A1c (A1c) as the standard marker has been called into question, due to physiologic and pathologic factors related to CKD.

We evaluated the correlation between A1c and average glucose levels in 1600 CKD patients at a large diabetes referral center. Average A1c was positively correlated with average glucose levels with a Pearson correlation coefficient of 0.52 ($p<0.0001$). Average eGFR was negatively correlated with average serum creatinine levels, as expected (-0.76 , $p<0.0001$). We then categorized eGFR according to CKD classifications into groups of 1-15, 16-30, and 31-60 cc/min/1.73m², and derived correlations between average A1c and average glucose levels in each category.

eGFR (1 to 60) (cc/min/1.73m ²)	N=1600	Pearson Coefficient (P value)
1 to 15	54	0.43 ($p=0.001$)
16 to 30	230	0.41 ($p<0.0001$)
31 to 60	1316	0.55 ($p<0.0001$)

While the correlation remained significant in each category, the level of correlation decreased at lower eGFR. In a regression model using A1c as a dependent variable and glucose, average eGFR, and their interactions as explanatory variables, the interaction term GLU*eGFR was statistically significant. The estimate of the coefficient indicated that the correlation between average A1c and glucose values increased as the level of eGFR increased.

Hemoglobin A1c may be less valid as a glycemic marker in advanced CKD. Factors which weaken the correlation between A1c and glucose levels even in patients not yet on dialysis need further evaluation.

SERUM MAGNESIUM CONCENTRATION AFTER MAGNESIUM CITRATE BOWEL PREPARATION

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Hypermagnesemia (>2.6 mg/dl), rarely seen in clinical practice, has been associated in case reports with respiratory depression, cardiac arrhythmias and death in elderly patients with normal kidney function taking magnesium citrate as a laxative. Magnesium citrate is now more widely used as a colonoscopy bowel preparation. We undertook a systematic physiologic study to evaluate the frequency and magnitude of serum magnesium increases after magnesium citrate bowel preparation for outpatient colonoscopy. We measured serum electrolytes, urine electrolytes, PTH, blood pressure, heart rate, ECG, and deep tendon reflexes in 17 men and women 2-3 days prior to colonoscopy and on the day of colonoscopy after a standard 3-bottle magnesium citrate bowel preparation. The mean serum magnesium level was 2.11 ± 0.26 at baseline and 2.43 ± 0.22 after bowel prep ($p=0.0043$). Four patients developed mild asymptomatic hypermagnesemia (2.7-2.8 mg/dl). The median increase in PTH after magnesium citrate was 8.5 pg/mL ($p=0.016$). Five patients had abnormal increases in PTH, and one was subsequently diagnosed with a parathyroid adenoma. Total serum calcium dropped 0.19 mg/dL ($p=0.0431$). Serum phosphorus dropped 0.32 mg/dL ($p=0.044$). Urine calcium and phosphorus excretion dropped nonsignificantly. We hypothesize that the rise in serum PTH after the magnesium citrate load may be due to a transient decrease in ionized calcium from citrate binding and will measure ionized calcium in the remaining 33 subjects to be enrolled.

A CASE OF PERIPARTUM HYPERCALCEMIA: A ROLE FOR PROLACTIN?

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In most cases the differential diagnosis of hypercalcemia in pregnant individuals is the same as in the nonpregnant state. We present a rare case of hypercalcemia of late pregnancy and postpartum period which appears to be due to lactation associated prolactin release.

A 33 years-old female, gravida 2, para 1, was diagnosed with hypercalcemia (serum calcium 15.1 mg/dL) at 33 weeks of gestation when she presented with preeclampsia. Her medical history was significant for nephrolithiasis and erythema nodosum and her family history for nephrolithiasis. 7 years prior, at 30 weeks during her first pregnancy, she had hypercalcemia that did not improve with conservative measures, a steroid course or delivery. At the time she had a suppressed parathyroid hormone (PTH) and PTH related peptide (PTHrp), a normal 25 OH vitamin D and a slightly elevated 1, 25-OH vitamin D at 70 pg/mL (normal 6 to 62 pg/mL). Four weeks postpartum she received a dose of intravenous pamidronate after stopping breastfeeding with normalization of her calcium levels over the next 10 days.

During the second pregnancy, evaluation yielded hypercalciuria, a slightly elevated 1-25 vitamin D, suppressed PTH and PTHrp. Hypercalcemia did not improve with fluids or induced delivery for severe preeclampsia at 36 weeks. A liver biopsy was negative for granulomatous disease. Treatment with a bisphosphonate was contemplated due to worsening hypercalcemia, thus she was instructed to wean breastfeeding. Her serum calcium levels decreased as her prolactin levels declined. A breastfeeding challenge resulted in an increase in levels of both serum calcium and prolactin. In follow up her serum calcium normalized and she remains normocalcemic at 6 months follow up.

Although several animal studies have suggested a calciotropic role for prolactin, this is the first human case report of an association between physiologic prolactin release in pregnancy and lactation and hypercalcemia.

HYPERCALCEMIA OF MALIGNANCY FROM SQUAMOUS CELL CARCINOMA OF LUNG - A THERAPEUTIC CHALLENGE

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We present a case in which bone metastasis, elevated Parathyroid hormone related protein (PTHrP) & ectopic parathyroid hormone (PTH) secretion from squamous cell carcinoma (SCC) of the lung caused recalcitrant hypercalcemia and presented a therapeutic challenge.

47 y/o male with scc of lung was admitted for severe hypercalcemia. Had negative sestamibi scan for parathyroid adenoma. Calcium was 13.3 mg/dl, intact pth was 1336 pg/ml, and PTHrP was 8.7 pmol/l, and phosphorus was 1.8 mg/dl. 24 hr urinary calcium was 8.4 gm/l. He was treated with IV fluids, lasix, calcitonin, pamidronate & cinnacalcet. Calcium increased to 15.5 mg/dl & pth to 8677 pg/ml. Patient received carboplatin and dacetaxel and calcium decreased to 7 mg/dl and pth to 5598 pg/ml in 24hrs. Restained bronchial brushings for parathyroid tissue were negative. Sudden correction of the pth level & serum calcium after chemotherapy indicate that the scc in lung or metastatic area was responsible for the pth production

6 cases of ectopic pth secreting tumor have been reported till date. Only once case of ectopic pth production from scc of lung has been recorded. Ectopic pth secretion, with PTHrP & bone metastasis from SCC of lung has never been reported before. In recalcitrant cases of hypercalcemia looking for ectopic pth production and PTHrP with treatment of primary tumor might be the best option to treat hypercalcemia

**DIAGNOSTIC VALUE OF URINE SODIUM
CONCENTRATION IN HYPONATREMIA DUE TO
SYNDROME OF INAPPROPRIATE ANTIDIURESIS (SIAD)
VERSUS HYPOVOLEMIA**

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We are often left with the differential diagnosis of SIAD versus hypovolemic hyponatremia. It is difficult to tell who will respond to isotonic saline infusion and who will not, if the urine Na value is not completely suppressed (urine Na >10 mEq/L). The diagnostic accuracy of the urine Na value was compared to that of a complete work-up and hospital course, including a response to saline infusion in patients with a final diagnosis of SIAD or hypovolemic hyponatremia. We also examined the diagnostic value of urine Na/BUN ratio which should improve separation between SIAD and hypovolemia since the urine Na and BUN move in opposite directions in these two conditions.

Twenty cases were assigned the final diagnosis of SIAD, and 16 cases were hypovolemic hyponatremia. Receiver-operating-characteristic (ROC) curves were constructed. The urine Na value of 50 mEq/L conferred the best accuracy in separating SIAD from hypovolemia: sensitivity 0.89, specificity 0.69, and accuracy 0.82. The diagnostic accuracy, as quantified by the areas under the ROC curves (AUC), was not statistically different between urine Na alone (AUC 0.89, 95% CI 0.77-0.96) and the urine Na/BUN ratio (0.93, 95% CI 0.83-0.98) (p=0.33).

When the underlying cause is inconclusive between SIAD and hypovolemic hyponatremia, and when only basic laboratory results are available at the time of initial evaluation, the urine Na < 50 mEq/L alone will be adequate to guide initial fluid management.

BRIDGING THE GAP: THE USE OF SPACED EDUCATION TO TEACH MEDICAL STUDENTS PRINCIPLES OF ACID-BASE AND FLUID-ELECTROLYTE DISORDERS

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Purpose of the study: This project aims to employ spaced education techniques to increase student understanding of the physiological mechanisms underlying fluid electrolyte and acid base disorders, and to improve the student's ability to apply this understanding in a clinical setting. Spaced education is a novel approach to teaching complex concepts that aims to improve long term retention by providing information multiple times over prolonged intervals of time.

Methods: In this project, second year medical students are sent an email twice a week for a –week period. Each email contains a link to a series of multiple choice questions which begin with a clinical scenario, and test the pathophysiological concepts, diagnostic approaches and appropriate therapies for disorders of acid base and fluid electrolyte physiology. Once the student selects an answer, the correct answer will be revealed along with explanations for each of the answer choices. Identical questions are repeated at predetermined intervals, so that students review the same material consistently over time.

Results: The effectiveness of our pilot program will be measured using qualitative data from focus groups and quantitative data from a pretest and posttest given to all study participants. Student feedback will include the overall structure and appeal of the program, the use of email to deliver course material, time spent on questions, and the value of answer explanations for students. The pretest and posttest will quantify the learning that occurred over the course of the program.

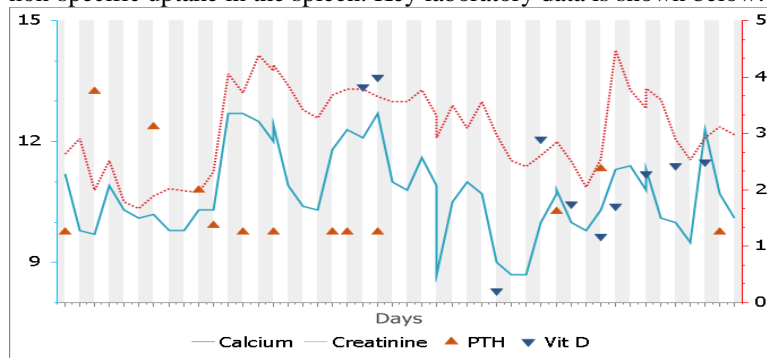
Conclusions: This study will provide data on a novel teaching approach to a complex subject area traditionally taught by nephrologists.

Results of our study will be presented at the meeting.

VITAMIN D MEDIATED HYPERCALCEMIA & RECURRENT KIDNEY INJURY

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A 81yr old white man with long standing hypertension was evaluated for hypercalcemia & elevated serum creatinine without proteinuria. Abdominal ultrasound revealed normal sized kidneys and heterogenic echogenicity of unclear etiology at the splenic hilum. Exhaustive serological evaluation was negative except for 1,25-Vitamin D (calcitriol) - 122 pg/ml (ref 15-75), 25-vitamin D - 23 ng/ml (ref 30-80) and a persistent M-Spike on serum protein electrophoresis. Bone marrow biopsies (MGUS) x 2 and kidney biopsy (moderate to severe arterial nephrosclerosis) failed to reveal the cause of hypercalcemia. CT of the chest, abdomen, pelvis was unremarkable, PET scan showed non-specific uptake in the spleen. Key laboratory data is shown below:



While the possibility of underlying granulomatous disease remains, exhaustive evaluation has yielded no explanation for excess calcitriol production. A diagnosis of idiopathic calcitriol mediated hypercalcemia was made and patient started on low dose prednisone (20 mg/d) with swift improvement in serum creatinine, calcium & calcitriol levels. Patient continues to be steroid-dependent as attempts to taper steroids were limited by worsening renal function (see figure).

To date, only 4 published case reports have described excess extra-renal calcitriol production in the absence of clear underlying etiology; all cases being responsive to steroids. Chloroquine and ketoconazole have been effective in reducing hypercalcemia in sarcoidosis and may be an alternative to steroids in calcitriol mediated hypercalcemia.

ELEVATED PTH AND PTH-rp IN A PATIENT WITH HYPERCALCEMIA OF MALIGNANCY

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Hypercalcemia is a common paraneoplastic syndrome associated with ovarian tumors. Rarely, these are associated with elevated levels of intact parathyroid hormone (iPTH) secretion. We describe the clinical, pathologic, and radiologic findings in a patient with metastatic ovarian carcinoma and hypercalcemia of malignancy associated with elevations of both iPTH and parathyroid hormone-related peptide (PTH-rp).

A 54 year-old woman presented to our hospital with weakness and confusion. She was found to be markedly hypercalcemic with a corrected calcium level of 19 mg/dL. On examination, she was found to have an enlarged liver span. A CT scan of her abdomen demonstrated a right ovarian mass and necrotic liver lesion. A biopsy of the liver showed metastatic grade 3 carcinoma, favoring adenocarcinoma with focal squamous differentiation. Serum carbohydrate antigen-125 and CEA levels were elevated. Both the iPTH (843) and PTH-rp (>2.8 pmol/L) were elevated. A dedicated 99m sestamibi parathyroid scan showed enhancement of the ovarian mass and liver lesion but no enhancement of the thyroid area. This suggested that primary tumor secretion was the source of both the iPTH and PTH-rp.

According to a review of the published literature, this is the second case reported where both the iPTH and PTH-rp were elevated as the cause of humoral hypercalcemia of malignancy.

HYPERNATREMIA IN A PATIENT TREATED WITH SODIUM POLYSTYRENE SULFONATE

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Sodium polystyrene sulfonate (SPS) is a cation exchange resin often used in the management hyperkalemia. We report a case of hypernatremia which occurred in the setting of SPS therapy. Only two other similar cases have been reported in the literature and both in neonates.

A 44 year old female resident of an extended care facility, with previously normal baseline renal function, was admitted to the hospital for confusion and lethargy. She had poor oral intake for the week prior to admission and her narcotic medications had been recently increased. Home medications included furosemide, spironolactone and lisinopril. Past history includes cirrhosis due to hepatitis C and chronic back pain. The initial evaluation revealed acute renal failure and hyperkalemia (7.1mmol/L). On physical examination she was hypotensive, afebrile and appeared volume depleted and lethargic. Laboratory values on admission are presented in Table 1. She was treated with intravenous insulin, dextrose, and saline infusion, with improvement in her urine output. Her diuretics and lisinopril were discontinued on admission. SPS, 60 grams every 6 hours (total of 240 grams), was administered resulting in watery diarrhea. Her serum sodium increased from 134mmol/L on admission to 151mmol/L within 24 hours later. The fractional sodium excretion was below 1%. The hypernatremia was likely secondary to free water losses due to the osmotic diarrhea induced by SPS. It normalized with free water hydration and renal function and potassium levels returned to normal.

The sodium avid state and physical signs of volume depletion lead us to believe that the net free water loss due to SPS induced diarrhea resulted in this patient's hypernatremia rather than a sodium exchange mechanism. This is the first adult case of hypernatremia in the setting of SPS therapy described to our knowledge. We hope to raise awareness among clinicians of this potential side effect of SPS therapy in adult patients.

UNIQUE ETIOLOGY OF HYPERCALCEMIA IN MALE TO FEMALE TRANSGENDER PATIENTS

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Male to female transgender individuals use a variety of silicone products and estrogen to produce a more feminine appearance. We postulate a unique etiology of hypercalcemia in 2 male to female transgender patients with puzzling symptomatic hypercalcemia.

Our first patient is a 38 yr old Hispanic phenotypic female referred to renal clinic for recurrent symptomatic hypercalcemia. She presented with a distant history of bilateral silicone breast implants; and multiple free silicone injections into her lips, hips and breasts, long standing hypercalcemia complicated with recurrent urolithiasis and renal failure from resulting obstructive uropathy. She was on estrogen tablets. Exam revealed multiple firm, non tender nodular swellings at sites of prior silicone injections. Labs showed hypercalcemia (14mg/dl), hyperphosphatemia (4.8mg/dl), hypercalciuria (379.6mg/24hr), elevated Cr (3.1mg/dl), low 25-OH Vit D (13ng/ml), high normal 1,25-OH Vit D (51ng/ml), low normal PTH assay (18.0pg/ml). PTH-RP, SPEP, CXR, parathyroid nuclear scan, bone scan were unremarkable.

The second patient is a 48 yr old Hispanic male to female transgender who presented with recurrent urolithiasis and worsening renal failure secondary to hypercalcemia. She had numerous free silicone injections into her hips, face and breasts 8 years prior to presentation that resulted in clinically appreciable injection granulomas. She took estrogen tablets as part of her feminizing regimen. Labs showed hypercalcemia (11.8mg/dl), hypercalciuria (637mg/24 hr), low 25-OH Vit D (9ng/ml), high normal 1,25-OH Vit D (63ng/ml), low PTH (1pg/ml). PTH-RP, SPEP and UPEP were unremarkable. Both patients had hypercalcemia secondary to elaboration of 1,25-OH Vit D by silicone induced granulomas likely exacerbated by estrogen use.

Silicone, once considered innocuous, has been reported to cause granulomas that leads to an unregulated extra-renal production of 1,25-OH Vit D resulting in hypercalcemia. Estrogen has been reported to increase calcium absorption by increasing 1,25-OH Vit D. We highlight the combined effect of silicone induced granulomas and estrogen causing hypercalcemia in this unique patient population.

AN UNUSUAL CASE OF SEVERE HYPOKALEMIA PRESENTING WITH HEMIPLEGIA

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Hypokalemia is a common clinical electrolyte abnormality.

A 68 year old male with a past medical history of hypertension and cerebrovascular accidents with residual left sided hemiparesis, presented with right sided hemiplegia. He also had a 10 week history of constipation and poor oral intake. Physical examination revealed hypoactive bowel sounds and residual left-sided hemiparesis. There was complete flaccid paralysis of all muscle groups on the right upper and lower extremities. He was on 25mg of hydrochlorothiazide daily.

Laboratory results showed a serum potassium of 1.5 mEq/L, magnesium 3.6 mEq/L, bicarbonate 19 mEq/L, urine potassium 25 mEq/L, Urine anion gap of +10, Urine PH of 7.0, non-gap metabolic acidosis and trans-tubular potassium gradient (TTKG) of 10.2. Patients total potassium body stores were significantly depleted. He required a total of about 100meq of potassium per day for the first 2 to 3 days.

The right-sided flaccid paralysis improved significantly with potassium replacement and power was fully restored by the 4th day of potassium replacement therapy. The etiology of hypokalemia in this gentleman is multifactorial and causes include 1) Type 1 (distal) renal tubular acidosis (RTA) 2) Decreased potassium intake 3) Thiazide diuretic therapy. Type 1 RTA certainly played a significant role in the severe hypokalemia in this patient. Type 1 RTA alone does not usually produce such severe hypokalemia and thus decreased oral intake and diuretic therapy may have played a role in the severity of hypokalemia. Thiazide diuretics increases urinary potassium excretion and could lead to clinically significant hypokalemia. In conclusion, in patients with type 1 RTA, diuretic exposure and poor oral intake could lead to life severe life threatening hypokalemia that could affect all muscle groups including skeletal, gastrointestinal, cardiac and respiratory muscles.

BULIMIA AS A CAUSE OF METBOLIC ALKALOSIS IN A PATIENT WITH CHRONIC KIDNEY DISEASE

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Chronic kidney disease (CKD) is associated with metabolic acidosis and hyperkalemia because of inability of the kidney to excrete hydrogen and potassium ions. We report a case of a 48 year old African American male with history of Human Immunodeficiency Virus (HIV) and Hepatitis C infection and history of hypertension who was sent for evaluation of Chronic Kidney Disease. Upon presentation he had a potassium level of 3.3 meq/L, bicarbonate (HCO₃) level of 42.1 meq/L, and creatinine (Cr) of 2 mg/dl (eGFR of 43 ml/min by the modified MDRD formula) The serum pH on arterial blood gas analysis was 7.51. Patient was on hydrochlorothiazide (HCTZ) initially but euvolemic. HCTZ was stopped and lab values obtained after 4 days, showed a potassium value of 3.9 meq/L and HCO₃ 38.8 meq/L. More history revealed daily binge eating followed by induced vomiting. Patient was started on Proton Pump Inhibitors (PPI) based on the assumption that preventing hydrogen secretion might ameliorate metabolic alkalosis. Repeat labs 2 weeks later showed resolving alkalosis.

Visit	K meq/L	HCO ₃ meq/L	Cr mg/dl
Initial	3.3	42.1	2.0
4 d after stopped HCTZ	2.9	37	2.2
Start of PPI	3.9	38	1.6
2 weeks after PPI	4.6	23	1.9

Bulimia can cause multiple electrolyte and acid-base abnormalities including hypokalemia and metabolic alkalosis. The focus of treatment is treating the underlying disorder as well as volume and potassium replacement. Therapy with proton pump inhibitors may be useful to improve hypokalemia and metabolic alkalosis by preventing hydrogen ion secretion in gastric fluid. This case illustrates the importance of history taking. It also highlights that bulimia is not always self evident.

FATAL HYPERCALCEMIA FROM TREATING HYPERKALEMIA

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Calcium is routinely administered for treatment of hyperkalemia with associated EKG changes. We describe a patient with CKD who developed fatal hypercalcemia during treatment of hyperkalemia.

A 59 year old man with a history of CKD stage 5 was found unresponsive at home. At the scene, a rhythm strip showed asystole. Cardiopulmonary resuscitation was performed including atropine and epinephrine injection by EMS. On arrival to the ER, an EKG revealed bradycardia, LVH and prominent T waves. The potassium level was 6.5 mEq/L. Calcium level was not reported. Presuming the EKG findings were due to hyperkalemia, multiple doses of calcium chloride (12 grams) were administered, as well as insulin and dextrose. Because of only transient improvement of bradycardia and hypotension, additional calcium was given for a total dose of 18 grams. After resuscitation, the serum calcium level was 24 mg/dL. A subsequent EKG showed bradycardia, a shortened QT interval, and unchanged prominent T waves with LVH. The potassium level was 5.9 mEq/L. The patient became hypotensive, was started on vasopressors and transvenous pacing. Shortly after admitted to the ICU, the family requested comfort measures and the patient expired.

A CKD patient presented with cardiac arrest. Hyperkalemia was considered the cause of bradycardia and prominent T wave, and emergently treated with calcium. Despite only a transient response, calcium continued to be given without considering the potential effects of hypercalcemia or underlying cardiac problem. A subsequent EKG showed shortening of the QT segment, persistent bradycardia, and unchanged T wave in the face of improved potassium levels. Although the exact dose of calcium for treating hyperkalemia is not well defined, only a few grams of calcium gluconate are generally recommended. This case demonstrates the unusual consequence of hypercalcemia from treatment of hyperkalemia. Intravenous calcium must be used judiciously and with close monitoring as resulting hypercalcemia can provoke fatal cardiac arrhythmias.

**HYPERCALCEMIA DUE TO GRANULOMATOUS ACTIVITY OF
PNEUMOCYSTIS JIROVECI PNEUMONIA IN A RENAL
TRANSPLANT PATIENT**

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Hypercalcemia due to macrophage production of 1-alpha hydroxylase is a known complication of granulomatous processes such as sarcoidosis and tuberculosis. It is a rare complication of Pneumocystic Jiroveci Pneumonia (PJP), which does not typically form granulomas.

We describe a 49 year old renal transplant recipient with stage 4 chronic kidney disease and secondary hyperparathyroidism who presented with shortness of breath, fever and pulmonary infiltrates. Ionized calcium on blood gas analysis was 1.36mmol/L. Further work-up revealed an elevated 1,25OH Vitamin D level of 90pg/mL. 25OH Vitamin D5 was 29.6ng/mL and PTH was 25pg/mL. Serum calcium rose to 12.6mg/dL. One year earlier, PTH was 215pg/mL and Vitamin D levels were normal. He developed respiratory failure with worsening bilateral pulmonary infiltrates. Cultures from broncho-alveolar lavage grew PJP. He was treated with intravenous bactrim and solumedrol with good clinical response. After one week of therapy, the 1,25OH Vitamin D level normalized. Serum calcium normalized shortly afterward.

Granulomatous activity leading to hypercalcemia in PJP infection is rarely described. We found 5 cases of PJP-associated hypercalcemia in the medical literature, two occurring in renal transplant patients. All cases responded to PJP therapy. Nephrologists should be aware of this entity as appropriate antimicrobial therapy should lead to resolution of the hypercalcemia.

HYPERCALCEMIA ASSOCIATED WITH GLUTEAL SILICONE INJECTIONS: A CASE REPORT

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Hypercalcemia is often the inaugural presentation of a variety of pathologic conditions including disorders of parathyroid glands, vitamin D metabolism or malignancies. However, less common etiologies should be considered in the differential diagnosis.

Case: A 49-year-old Hispanic speaking female with a history of hypertension, hysterectomy for fibroids and multiple plastic surgical procedures including breast augmentation with saline-containing implants. She was seen in medical clinic for myalgias and fatigue and was referred to the nephrology clinic for evaluation of an elevated serum creatinine (Cr) of 2.7 mg/dL with a BUN of 43 mg/dL. Subsequent testing revealed severe hypercalcemia (14.6mg/dL) and anemia (Hb 9g/dL). Serum albumin was 2 mg/dl phosphorus was 2.2 mg/dl, basic chemistry panel was normal. PTH appropriately suppressed (4 pg/ml), vitamin D (25-OH) 10ng/dL and TSH was normal. The patient was treated with IV fluids and pamidronate. A complete work up was initiated to rule out malignancies including a negative SPEP, UPEP, HIV antibody, mammogram and colonoscopy. CT scan revealed bilateral punctuate calcifications in the renal medullary regions and minimal edema was observed around both breast implants. A transvaginal ultrasound raised a concern for a pelvic mass and pelvic MRI showed enlarged bilateral inguinal and right-sided obturator lymph nodes and also inorganic material in the gluteal areas. Patient recognized having had gluteal silicone injections. A biopsy of an inguinal lymph node was consistent with silicone lymphadenopathy. In order to elucidate the nature of her renal insufficiency a renal biopsy was performed and revealed extensive nephrocalcinosis and chronic interstitial scarring with collagen infiltration (50 – 60%). Oral prednisone was added to her regimen. Cr and calcium stabilized around 2.2 mg/dL and 10.9 mg/dL respectively. She was also treated with oral iron for iron deficiency and was encouraged to maintain hydration. Extensive literature search only showed a single report of hypercalcemia associated with silicone injections.

HYPONATREMIA AND STEROID RESISTANCE IN HIV DISEASE-

Jiwan Thapa, Satinder Singh, Apurv Khanna, SUNY Upstate, Syracuse, NY, USA

We present a case report of hyponatremia in a 28 year old male with HIV/AIDS who presented with 1 month duration of weakness, dry mouth, diarrhea, abdominal pain and dizziness. He was febrile with blood pressure of 107/64 mm Hg and orthostatic hypotension. Examination revealed truncal hyperpigmentation, absence of peripheral edema and otherwise normal systemic findings. Laboratory investigations revealed serum sodium of 122mmol/L, serum potassium- 4.1 mmol/L, serum bicarbonate- 23mmol/L, serum chloride- 92mmol/L, serum BUN -12mg/dl, serum creatinine-0.6mmol/L & positive CMV antigenemia. Stool studies, sigmoidoscopy with biopsy & imaging studies were negative.

Diarrhea improved with Gancyclovir therapy. However, orthostatic hypotension persisted despite volume expansion with 14 liters of normal saline over 3 days. At this stage patient had a urine output of 4.5liters/24 hours, urine sodium-132meq/L with urinary sodium loss of 594 mEq/day. Copious urine sodium loss, clinical hypovolemia & orthostatic hypotension narrowed the differential diagnosis to cerebral salt wasting and adrenal insufficiency. The clinical presentation was not consistent with cerebral salt wasting. Random cortisol level was normal at 19.8mcg/dl with appropriate cosyntropin stimulation. Conventionally these values would exclude adrenal insufficiency as an etiology but in patients with HIV, acquired steroid resistance at the peripheral receptor level with a high normal baseline cortisol level and appropriate response to cosyntropin has been described.

We initiated treatment with Hydrocortisone 50 mg IV QID and Fludrocortisone 0.1 mg BID. The patient showed resolution of orthostatic hypotension and marked reduction in his urine output to 1.5 liters /day, urine sodium -23meq/L with reduction in sodium excretion to 34mEq/day. Serum sodium improved to 137 mmol/L.

This case report exemplifies an unusual etiology of hyponatremia and adrenal insufficiency in patients with HIV disease and the therapeutic outcome supports our clinical hypothesis. Acquired steroid resistance at the peripheral receptor level in patients with HIV disease deserves further elucidation with laboratory testing.

PREVALENCE OF METABOLIC ACIDOSIS AND THE DISTRIBUTION BETWEEN ANION GAP AND NON-ANION GAP ACIDOSES

Sandeep Tiyyagura, Thinh Nguyen, Beth Stefanchik, Andrew Bohmart, and Godfrey Burns. St. Vincent's Catholic Medical Center, New York, NY and New York Medical College, Valhalla, NY.

Metabolic acidosis is a common finding in hospitalized patients. However, its prevalence and the distribution between anion gap (AG) and non-anion gap (NAG) acidoses has not been described. The aim of this study is to document the prevalence of metabolic acidosis in hospitalized patients and to determine the distribution between AG and NAG acidoses in this population.

1,000 consecutive patients admitted to the general medical service were screened for an admission serum bicarbonate of less than 24 mmol/L, our definition for metabolic acidosis. The anion gap was calculated and adjusted for serum albumin. An anion gap greater than 12 was considered elevated. Demographics, diagnoses, serum and urine electrolytes, blood gas, lactate, and eGFR were obtained.

Of the 1,000 patients studied, 108 (11%) had a serum bicarbonate less than 24 mmol/L. Of these, 51 (47%) had a NAG acidosis and 57 (53%) an AG acidosis. Patients with an AG acidosis were more likely than those with a NAG acidosis to have a bicarbonate less than 20 (39% vs 10%), underlying CKD (40% vs 24%), ESRD (9% vs 2%), and ARF (39% vs 24%) and had a lower incidence of HIV (9% vs 18%). These patients were more likely to have had a lactate measured (19% vs 16%) and for it to be greater than 2 (12% vs 0%) and also more likely to have had a blood gas performed (18% vs 8%) and the pH to be less than 7.35 (7% vs 2%).

Metabolic acidosis was a common finding in our study population with a prevalence of 11%. The distribution between AG and NAG acidoses was near equal. An incidental finding was that patients with established renal disease were more likely to have an AG acidosis, while patients with HIV were more likely to have a NAG acidosis. Further studies are indicated in a larger population to validate our findings.

HYPONATREMIC PATIENTS DEMONSTRATE IMPROVED COGNITION AFTER AN INCREASE IN SERUM SODIUM

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Hyponatremia occurs in 15% to 30% of hospitalized patients, making it the most common electrolyte abnormality in this population. Mild hyponatremia has generally been considered asymptomatic, however, a recent study showed that patients with mild to moderate chronic hyponatremia had an increased risk of falls possibly due to gait and attention impairments. The present study was designed to assess cognition levels using the Mini-Mental Status Exam (MMSE) in patients with varying degrees of hyponatremia pre and post serum sodium (SNa) improvement.

Fourteen hospitalized patients with SNa values ≤ 130 meq/L from a single center were included. The MMSE was administered to these patients and scores recorded out of a maximum of thirty. The MMSE was repeated when the investigators felt the SNa improved appreciably, which on average was 9.2 meq/L. Initial SNa levels were approached therapeutically with 0.9% NS, fluid restriction, vasopressin receptor antagonists, withholding medications, or 3% NS. The MMSE were administered at least 72 hours apart and the individual questions were altered when the test was repeated. Pre and post SNa improvement MMSE scores were compared.

The initial SNa levels of the fourteen patients ranged from 117 to 130 meq/L with a mean of 123.8 meq/L ($SD \pm 4.0$) and post-improvement SNa ranged from 127 to 137 meq/L with a mean of 133.0 meq/L ($SD \pm 3.5$, $p=0.001$). Overall, thirteen of the fourteen patients (93%) had an increase in MMSE score after improvement in SNa ($p=0.001$). Six patients had a 4-10% increase in MMSE score, five patients had a 12-20% increase, one patient had a 21% increase, and one patient had a 35% increase in the MMSE score. Interestingly, the patient with the largest increase in the MMSE score had the least number of comorbidities.

When SNa levels are improved in hyponatremic patients, cognitive function is also be enhanced.

SEVERE ELECTROLYTE ABNORMALITIES AFTER CETUXIMAB- MANAGEMENT CHALLENGE

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Introduction: Renal Magnesium Wasting Syndrome occurs in patients treated with epidermal-growth-factor receptor (EGFR)-targeting antibodies for colorectal cancer. Magnesium is essential for bone stability, neuronal excitability, muscular relaxation and many other functions. Severe hypomagnesemia can lead to significant hypokalemia and hypocalcemia. Many patients with colorectal cancer have partial colectomies and oral supplementation can pose significant challenge.

Case Report: We present a case of a 69 year old female with history of metastatic colon cancer to the liver, diagnosed few years ago s/p partial colectomy with ileostomy, hypertension, ascites, who was admitted to hospital with shortness of breath. She was treated with cetuximab about a week prior to admission. On admission, she had no diarrhea and was not on diuretics. She was noted to have K 2.9 mmol/L, total serum calcium 6.5 mg/dl, albumin of 3.5 gm/dl and serum magnesium of 1.4 mg/dl. Urine magnesium was inappropriately high demonstrating significant renal wasting of magnesium. She was treated with oral magnesium, potassium and calcium supplements and with intravenous supplementation on an as needed basis. Over the next few days, she remained severely hypomagnesemic to as low as 0.4 mg/dl at times requiring more than 8 grams/day of magnesium supplementation and severely hypocalcemic to as low as 4.5 mg/dl with symptoms of tetany and seizures. She was noticed to have pills in her ostomy bag and thought not to be absorbing oral medications. She was treated with aggressive IV magnesium and calcium supplementation with some improvement and amiloride, which is a potassium and magnesium sparing diuretic with improvement in electrolytes.

Conclusion: Hypomagnesemia from magnesium wasting due to cetuximab therapy can be very. Oral supplementation can be a challenge in patients with ileostomy. Amiloride may be a useful adjunctive therapy due to magnesium sparing properties.

MANAGEMENT OF GITELMAN SYNDROME IN PREGNANCY

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Gitelman Syndrome (GS) is a rare autosomal recessive disorder caused by mutations in the gene for the thiazide-sensitive sodium-chloride cotransporter in the distal renal tubule. GS is characterized by hypokalemic metabolic alkalosis, hypomagnesemia with low urinary calcium excretion. Muscle weakness, tetany, cardiac arrhythmia and palpitation can occur in these patients due to severe hypokalemia and hypomagnesemia although most of the patients remain asymptomatic. Female patients with GS develop more severe symptoms during pregnancy due to worsening hypokalemia and hypomagnesemia. However, there are only scant reports discussing the management of GS in pregnant patients.

We report a 32 year old female who was diagnosed with GS at age 18 by 24 hour urine after developing symptoms of hypokalemia. She had maintained stable serum potassium and magnesium on 60 mEq KCl and 400 mg magnesium oxide supplements until she became pregnant. At 3 weeks gestation she developed generalized muscle weakness and extreme fatigue and was found to have a serum potassium of 2.9. Intravenous repletion of potassium was required and her daily supplementation was increased throughout her pregnancy. During her 3rd trimester, the daily supplementation had been increased to 160 mEq KCl and 1600 mg magnesium oxide which is a 3-4 fold increase from her pre-pregnant dose. Despite magnesium-induced diarrhea, she remained asymptomatic with a serum potassium level of 3.5- 4.1 and a magnesium level of 1.5-1.7 with normal fetal development.

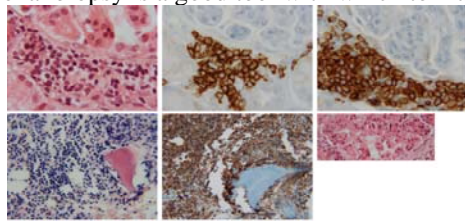
Renal wasting of potassium and magnesium in GS is dramatically increased during pregnancy. This effect is thought to be mediated by increased GFR and elevated aldosterone levels. Daily supplementation of potassium and magnesium can rise as high as 4 fold in order to maintain normal serum levels. Our case illustrates the complicated management issues that arise in pregnant patients with GS. With aggressive repletion and close monitoring, it is possible to achieve adequate serum magnesium and potassium levels as well as an optimal obstetric outcome.

SMALL B CELL LYMPHOMA MANIFESTING SOLELY AS NEPHROTIC RANGE PROTEINURIA. A CASE REPORT

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Introduction: Renal involvement in lymphomoid malignancy is a well known phenomenon; however renal manifestation of Non Hodgkin lymphomas as the only extra nodal site presentation is extremely rare.

Case Report: A 45 year Caucasian female with no significant past medical history presented with bilateral lower extremities edema for 6 months. Physical examination was unremarkable except for pitting edema. Initial work up showed hypoalbuminemia & 24 hour urinary proteinuria of around 7 grams. Further lab work was negative for hepatitis profile, Anti streptolysin O titer (ASO), Anti-nuetrophilic antibody (ANA), anti-neutrophilic cytoplasmic antibodies (ANCA). Quantitative immunoglobulin showed low levels of IgG with out M peak. Serum Uric acid and LDH were normal however complements (C3, C4) levels were low. Renal biopsy showed pattern of membranoproliferative glumerulonephritis (MPGN) type I but interestingly also showed atypical interstitial infiltration composed of B-Cells positive for CD 20. Bone Marrow Biopsy and flow cytometry revealed monoclonal lambda B cells marked positively for CD19 & CD 20, confirming the diagnosis of low grade B cell lymphoma. CT scans of neck, chest, abdomen and pelvis were unremarkable. PET scan was also negative. Patient then received chemotherapy with CHOP/Rituxan regimen for 8 cycles with complete remission of lymphoma, confirmed by repeat bone marrow biopsy and quite interestingly her proteinuria also resolved completely. Discussion: MPGN is an uncommon complication of B cell lymphoma. The possibility of lymphoma should be taken into consideration in patient with unexplained proteinuria and renal biopsy is a good tool with which to make an early diagnosis.



**MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS
(MPGN) SECONDARY TO TYPE III CRYOGLOBULINEMIA
ASSOCIATED WITH SMALL LYMPHOCYTIC LYMPHOMA
TREATED WITH RITUXIMAB**

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A 63 year old male was evaluated for anasarca and was found to have renal failure, nephrotic range proteinuria and microscopic hematuria. His ANA screen, ANCA ,hepatitis B & C and HIV serologies were negative. Serum C3 and C4 were low. Serum cryoglobulins were positive and immunofixation showed type III cryoglobulinemia. Serum electrophoresis was negative for monoclonal proteins but free light chain assay revealed Kappa and Lambda chains. Renal biopsy showed MPGN, immune complex mediated. Further workup included CT scan, which revealed periaortic and pelvic lymphadenopathy and a bone marrow biopsy, which showed lymphoid aggregates, positive for CD5 and CD20. A diagnosis of small lymphocytic lymphoma was made. The patient underwent plasmapheresis twice and completed three of the four scheduled courses of rituximab, each consisting of 4 weekly treatments of human/mouse chimeric monoclonal antibody. He has responded well to the treatment with normalization of renal function and complete resolution of anasarca. His creatinine level has improved from 2.34 mg/dl to 0.8 mg/dl and urine protein to creatinine ratio from 7.8 to 0.2. Test for cryoglobulin is now negative.

This case illustrates that secondary form of MPGN may sometimes respond to treatment of the primary disorder. It also highlights the need for extensive workup for secondary causes including bone marrow biopsy in selective cases of MPGN.

A RARE CASE OF INTERSTITIAL NEPHRITIS WITH SECONDARY MEMBRANOUS GLOMERULOPATHY DUE TO GLIMEPIRIDE

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Interstitial nephritis is known to be caused by a vast array of medications, infections and systemic disease processes. It is an immunologically induced hypersensitivity reaction to an antigen. We report a case of drug induced interstitial nephritis in a patient in whom we had suspected to have renal sarcoidosis.

A 50 year old African American male with history of DM-2 and recent diagnosis of Sarcoidosis after an inguinal lymph node biopsy was evaluated on outpatient basis for acute kidney injury. The patient was asymptomatic, and the physical exam was unremarkable except for bilateral axillary, submental and inguinal lymph nodes. Laboratory data showed a Creatinine of 2.72mg/dL, calcium of 10.3mg/dL, ACE level of 240U/L, urine with 400mg of proteinuria/24hr. All workup for HIV, protein electrophoresis was unremarkable. Renal ultrasound showed bilateral normal sized echogenic kidneys. During this whole time the only medication that the patient was taking was Glimepiride. A renal biopsy was done to rule out renal sarcoidosis. The renal biopsy showed acute inflammatory interstitial infiltrates with extensive interstitial fibrosis and eosinophils without granulomas. Electron microscopy showed epimembranous deposits suggestive of secondary membranous glomerulonephropathy. Immunofluorescence was non-contributory. Since only Churg-Strauss and interstitial nephritis due to drugs presents with this pattern of eosinophilic infiltrates, thus the patient was diagnosed with drug induced interstitial nephritis. The patient was subsequently switched to regular insulin for his diabetes, and started on Prednisone at a dose of 1mg/kg. His renal function improved in the next few weeks to a creatinine of 1.82mg/dL with calcium of 9.2mg/dL, as he was placed on a slow prednisone taper.

This case illustrates Glimepiride as a cause of drug induced interstitial nephritis in a patient with sarcoidosis which responded to steroids. This case also shows the importance of renal biopsy in patients with unexplained acute kidney injury despite a high suspicion for a specific diagnosis.

ANTI-C1Q ANTIBODY LEVELS ARE NOT RELIABLE FORECASTERS OR MARKERS AND LUPUS NEPHRITIS FLARE

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The identification of biomarkers of lupus nephritis (LN) that reliably identifies, or more importantly forecasts, a LN flare would greatly improve LN management. Anti-C1q antibodies (Ab) have been shown to associate with LN, and reports have suggested that anti-C1q Ab levels can be used to forecast or mark a LN flare. However, no study has directly tested this through serial unbiased measurements leading up to LN flare. The goal of this study was to determine this using plasma samples collected at regular bimonthly intervals from LN patients enrolled the Ohio SLE Study. Specifically, anti-C1q Ab levels were measured in samples collected 8, 6, 4, and 2 months before flare, and at the time of flare in 17 LN patients (WHO class III, IV, and V) experiencing 20 renal flares. Antibody levels were measured using ELISA methodology, and 30 matched normal control plasma samples were included for comparison. The data were analyzed by repeated measures ANOVA with post-testing. Of the 20 LN flares, 16 were positive for anti-C1q Ab. For these 16, there was a significant increase in the mean Ab level at flare, and a trend for an increase 2 months before flare, compared to 6 months or 4 months before flare. However, for each individual flare, higher Ab levels were present in only 8 of 16 at flare, and in only 7 of 16 at 2 months prior to flare, compared to the corresponding levels at 8, 6, or 4 months before flare. These data show that, despite a significant association between higher mean anti-C1q Ab levels and LN flare, individual anti-C1q levels are unreliable as markers or forecasters of LN flare.

CHRONIC BRONCHIECTASIS AND PERINUCLEAR-ANCA (pANCA) CRESCENTIC GLOMERULONEPHRITIS

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61 year old black female with a history of asthma, mitral stenosis, and hypertension was initially evaluated eight years ago for a chronic cough. She underwent a transbronchial biopsy revealing chronic inflammation and fibrosis. Infectious etiologies were ruled out, including mycobacterium and fungus.

Upon our evaluation, the patient presented with nausea and malaise. On physical exam, she had dry oral and ocular mucus membranes, and crackles at her lower lung bases. On admission, she presented with a creatinine of 13.2 mg/dL. Further work up revealed positive rheumatoid factor (472 IU/mL), ANA (1:640), pANCA (1:320), and mpo Ab (537 AU/mL). Her anti-GBM Ab, anti-dsDNA Ab, complements, hepatitis B and C, and HIV were either normal or negative. Renal biopsy revealed a cellular crescentic GN involving 90% of the glomeruli without significant tubulointerstitial involvement. Immunofluorescence revealed mesangial staining with IgG (1+), IgA (1+), IgM (trace), C1q (trace), C3 (1-2+), lambda/kappa light chains (trace). Electron microscopy revealed mild foot process effacement. She was diagnosed with primary Sjogrens syndrome based on sialadenitis, xerophthalmia, positive anti-ssA/ssB Abs, and CT sinus showing fat replacement of her parotid glands. CT chest revealed chronic bronchiectasis. The patient required renal replacement therapy, but was treated with steroids and cyclophosphamide.

There is one prior case series describing chronic bronchiectasis associated with pANCA crescentic GN. In all cases, the lung findings preceded renal involvement. This case highlights the potential relationship between bronchiectasis and ANCA mediated renal disease. Since the renal injury is potentially reversible, this condition should not be overlooked.

A CASE OF PODOCYTURIA IN A PATIENT RECEIVING ANTI-VEGF THERAPY WITH SUNITINIB

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We report a 67 year-old female presenting with persistent dry cough and unintentional weight loss over a course of 6 months. Radiologic studies (X-ray and CT) revealed multiple bilateral pulmonary nodules accompanied by a 7.2 X 6.1 cm mass involving the central and lower portion of the right kidney. Renal function was normal (serum creatinine 1 mg/dl without proteinuria). She underwent a right radical nephrectomy and retroperitoneal lymphadenectomy. Pathologic diagnosis confirmed Grade 2, T2, N0, M1 renal cell carcinoma, clear cell type. The patient responded to Sunitinib therapy. After 4 months of therapy, however, her renal function worsened with a peak serum creatinine of 1.5 mg/dl and 467 mg of urinary protein/24Hr. Angiotensin-II receptor blockade was used to treat Sunitinib related hypertension. Sunitinib related hand and foot syndrome, and drug-induced hypothyroidism were also noted. After seven months of therapy, serum creatinine improved to 1.3 mg/dl with 931 mg of urinary protein in 24 hours. A podocyturia assay was performed to ascertain the presence of urinary podocytes. Podocyturia was confirmed in the proteinuric patient while in 2 control patients on anti-VEGF therapy and without proteinuria, podocyturia was not observed.

To our knowledge, this is the first reported case of podocyturia in a patient receiving anti-VEGF therapy. Podocyturia has been observed in many renal diseases, including preeclampsia, and may be a consequence of relative decrease of free plasma VEGF. We hypothesize that anti-VEGF therapy acts similarly to decrease free VEGF levels, leading to podocyte injury, podocyturia, and proteinuria.

COEXISTENT P-ANCA AND ANTI-GBM ANTIBODY GLOMERULAR DISEASE ASSOCIATED WITH ENDOMETRIAL CANCER

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Introduction: While the association between malignancy and certain glomerular diseases is well appreciated, little is known about the risk of cancer in patients with ANCA-positive glomerulonephritis (GN). Here, we report a case of endometrial adenocarcinoma as the initial presentation of microscopic polyangiitis (MPA). We then review the current literature and potential pathophysiologic mechanisms linking malignancy to ANCA-positive glomerular disease.

Methods: A 69-year-old Caucasian woman with no significant past medical history was admitted for a two-week history of intermittent vaginal bleeding and new-onset lower extremity edema. At the time of admission, she was found to be hypertensive. Laboratory studies revealed severe renal insufficiency (serum creatinine level 8.0 mg/dl) and anemia associated with hematuria and proteinuria (4.9 gram/day). Further investigations showed positive ANA ($\geq 1/1280$), positive pANCA, positive anti-GBM antibody (32 AU/ml; N: 0-19), and low serum complement levels (C3: 82 and C4: 6 mg/dl). The patient underwent endometrial and renal biopsy.

Results: Grade-1 adenocarcinoma was found on endometrial biopsy. Renal biopsy showed focal proliferative and focal segmental necrotizing GN with crescent formation and only minimal GBM staining, compatible with MPA. Daily sessions of plasmapheresis were initiated accompanied by steroid bolus and oral cyclophosphamide. This regimen was followed by rapid improvement in renal function and proteinuria. The patient did not need renal replacement therapy.

Conclusion: To our knowledge, this is the first case in which endometrial cancer is the initial presentation of glomerular disease in a patient with co-existent ANCA and Anti-GBM antibody. Based on this case coupled with previous reports linking glomerular disease to malignancy, we suggest that patients with cancer and unexplained renal dysfunction be also evaluated for Anti-GBM or ANCA-associated glomerular disease.

IDIOPATHIC NODULAR GLOMERULOSCLEROSIS IN A NON-DIABETIC YOUNG CAUCASIAN MALE WITH METABOLIC SYNDROME: A CASE REPORT

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We report an unusual case of idiopathic nodular glomerulosclerosis (INGS) with light microscopic and ultrastructural features indistinguishable from that associated with diabetic glomerulopathy in a young Caucasian male without evidence of diabetes mellitus and no history of smoking. A 27-year-old non-diabetic Caucasian male was first documented to have uncontrolled hypertension, 1+ proteinuria and normal renal function in 2001. A routine screening in 2008 revealed a creatinine of 1.6 mg/dL associated with 3+ proteinuria. He presented at that time with BMI 28 kg/m², BP 148/96 mm Hg and HR 84 bpm. Serologic work up revealed Scr 1.6 mg/dL (eGFR=58 mL/min/1.73 m²), LDL 100 mg/dL, TG 188 mg/dL, HDL 32 mg/dL, uric acid 10 mg/dL, and fasting blood glucose of 103 mg/dL (ref. 66-90), and HbA1c 6.1 mg/dL. Random urine protein-to-creatinine ratio was 1.1 mg/gm of creatinine with a normal serologic work-up for GN including normal serum free light chains. He underwent diagnostic renal biopsy, which showed LM findings of nodular intercapillary glomerulosclerosis and mesangiolysis with mild to moderate interstitial fibrosis and unremarkable IF and EM findings, which were indistinguishable from diabetic glomerulopathy. In contrast, our patient was a young male with metabolic syndrome and possibly mild glucose intolerance but not overt type 2 DM. INGS has previously been reported to be associated with hypertension, obesity and smoking in elderly males. To our knowledge, this is the first description of idiopathic nodular glomerulosclerosis with renal impairment in a young non-diabetic adult male with metabolic syndrome.

**CYCLOSPORINE FOR TREATMENT
MEMBRANOPROLIFERATIVE
GLOMERULONEPHRITIS (MPGN) ASSOCIATED WITH
CHRONIC LYMPHOCYTIC LEUKEMIA (CLL)**

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The incidence of nephrotic syndrome in CLL is 1-2%. The most common pathological finding is MPGN (35.7%). We report a case of CLL-associated MPGN treated with cyclosporine that resulted in long-lasting remission.

60 yo female with CLL was evaluated for new onset proteinuria. Laboratory evaluation: WBC of 140k/ul, serum creatinine of 1.3 mg/dl and urine albumin excretion of 4.7 G/24 hours. HIV, hepatitis B, C, serum free light chains, cryoglobulin, serum & urine electrophoresis and ANA were negative. C4 was low. Kidney biopsy revealed global endocapillary hypercellularity and duplicated basement membranes. Immunofluorescence revealed coarse granular basement membrane staining for IgG, C1q and C3 indicating MPGN type1. Patient did not tolerate chlorambucil (pancytopenia). Started cyclosporine and protein excretion decreased to 160 mg over 6 months.

The mechanism of MPGN in CLL may be related to immune complex deposition. The effect of cyclosporine may be due to blocking of interleukin 2, 10, and TNF- α and also by lowering the auto reactive CLL cells leading to decrease in immune complex formation. Supporting this last hypothesis is that the patient's WBC has stabilized at 35k/ul. To our knowledge this is the first case demonstrating the efficacy of cyclosporine in inducing remission in CLL-associated MPGN.

MINIMAL CHANGE DISEASE (MCD) IN PATIENT OF ULCERATIVE COLITIS (UC), ADVERSE REACTION TO BALSALAZIDE OR DISEASE ASSOCIATION? LITERATURE REVIEW & CASE REPORT

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MCD is known to occur in patients of UC, though majority of available literature is from 1980s & 1990s. The mainstay of therapy for UC is sulfasalazine/5-aminosalicylate (5-ASA). The cause of MCD was mainly attributed to the sulphapyridine moiety of sulfasalazine. Mesalamine, which contains only 5-ASA, was initially thought not to cause MCD. Subsequently, there were reports of MCD in patients on mesalamine. Moreover mechanism of action remains speculative. There has been no reported case secondary to Balsalazide yet, which has 5-ASA linked to an inert unabsorbed carrier molecule.

We report a case of 56-year-old man with history of UC for about 10 years. Patient was on balsalazide for 5 years, was not on any NSAIDS & had regular colonoscopy exams. He presented with progressive leg swelling for 4 months. A 24-hour urine showed 12 grams of proteinuria. Renal biopsy was consistent with MCD without any evidence of FSGS. Balsalazide was discontinued and prednisone was initiated at 60 mg/day, both for treatment of MCD and to prevent UC flare up. Proteinuria decreased to 7 grams after 3 weeks of therapy at the time of this report.

1.This is first reported case of biopsy proven MCD, suspected of being caused by Balsalazide **2.** In absence of sulphapyridine moiety in this compound, the mechanism of action could be related to NSAID induced MCD caused by systemically absorbed 5-ASA. **3.** As only minimal systemic absorption of 5-ASA takes place, whether MCD is secondary to cumulative toxicity of 5-ASA over the years needs to be proven **4.**There could also be role of Tcell dysfunction as a common disease pathway in the pathogenesis of both diseases, which needs to be investigated.

**MEMBRANOUS NEPHROPATHY WITH
MYELOPEROXIDASE (MPO) ANTINEUTROPHIL
CYTOPLASMIC ANTIBODY (ANCA) ASSOCIATED
NECROTIZING AND CRESCENTIC
GLOMERULONEPHRITIS (NCGN)**

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The coexistence of membranous nephropathy (MN) and ANCA associated NCGN is infrequent. Here, we report such a case, who presented with rapid decline in renal function.

An 87 year old woman came to the hospital after she fell at home. Physical examination revealed hypoxemia, bilateral diffuse fine crackles on lung auscultation and trace pedal edema. Her baseline serum creatinine was 0.8 mg/dl. Initial lab tests showed BUN/Cr 67/3.9 mg/dl, albumin 2.3 g/dl and hemoglobin 6.6 g/dl. UA revealed numerous RBC's, no RBC casts and proteinuria 2.1 gm/ 24hr. CXR and CT showed diffuse bilateral alveolar infiltrates. Later a bronchoalveolar lavage confirmed the presence of pulmonary hemorrhage. Serology was remarkable for high titers of MPO ANCA with positive anti dsDNA and anticardiolipin antibodies. ANA was weakly positive. Complement assays were normal and anti GBM antibody was negative. Kidney biopsy showed MN with NCGN. There was lack of endocapillary proliferation, subendothelial deposits, tubular reticular inclusions, C1q reactivity and extraglomerular deposits. All these suggest the presence of two separate immunological processes (MN and ANCA associated NCGN) rather than lupus nephritis. The patient was treated with steroids and cyclophosphamide with improvement in renal function. In conclusion, we believe our case of MN and ANCA associated NCGN is unique, because the patient had strong seropositivity for anti dsDNA and anticardiolipin antibodies, but with no clinical or histological evidence of lupus. Such combination of findings has not been reported previously. This case also illustrates, that prompt initiation of immunosuppressive therapy can be helpful in patients with MN and ANCA associated NCGN.

A UNIQUE CASE OF COLLAPSING GLOMERULOPATHY IN A PATIENT WITH MIXED CONNECTIVE TISSUE DISEASE

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Collapsing glomerulopathy (CG) is a distinct clinicopathological entity characterized by glomerular capillary collapse, podocyte proliferation, diffuse mesangial sclerosis, and podocyte maturation arrest. Mixed connective tissue disease (MCTD) is a disease with overlapping features of systemic lupus erythematosus (SLE), progressive systemic sclerosis (PSS), and polymyositis. To our knowledge, there has been only one reported case of MCTD associated with CG and very limited data was presented with that case.

Our patient is a 60yr old AA female who was diagnosed with MCTD on the basis of findings of polymyositis, Raynaud's phenomenon, arthritis, pulmonary hypertension, gastrointestinal dysmotility, and positive ANA and anti-ribonucleoprotein antibody tests. Her disease dates back to the 1980's, but was relatively well-controlled with the use of a variety of anti-inflammatory and cytotoxic agents. In early 2008 mild proteinuria was first noted.

In February 2009, she was admitted to our institution with abdominal pain. During the admission she developed nephrotic range proteinuria, acute renal failure, and worsening Raynaud's phenomenon. A renal biopsy with three glomeruli per section showed prominent podocyte injury and hyperplasia with collapse of the underlying glomerular tuft, consistent with CG. There was also an acute tubulointerstitial nephritis and an arteriolar thrombotic microangiopathy. Immunofluorescent staining and EM showed no good evidence of an immune complex mediated process. HIV and parvovirus B19 serologies were negative. She was started on steroids and hemodialysis. She remained on dialysis for 4 weeks and then recovered renal function with her serum creatinine improving to 1.5mg% and her proteinuria decreasing to 1.3gm/day.

In summary, MCTD is a rare connective tissue disease that tends to evolve over a period of years. Our patient had MCTD for many years and then developed an acute exacerbation of disease associated with finding of CG on renal biopsy. Our case represents the first biopsy proven well documented case of CG in a patient with MCTD.

IGA NEPHROPATHY IN CIRRHOSIS; INCIDENCE, PATHOGENESIS, AND CLINICAL SIGNIFICANCE: A LITERATURE REVIEW

Hilana Hatoum¹, Fadi Rzuq².¹: McLaren Regional Medical Center, Flint, MI. ²: University of Washington, Seattle, WA. Background: Increased serum IgA levels are found in the vast majority of cirrhotic patients with resulting glomerular IgA deposition. Many theories have explained this association but the clinical significance behind this glomerular deposition remains a mystery. The aim of this review is to evaluate the incidence, pathogenesis, and clinical significance of glomerular IgA deposition in cirrhosis. Methods: An extensive literature review was conducted of studies about the dysregulation of IgA immune complex (IC) clearance in cirrhotic patients and their deposition in the glomeruli focusing on the clinical significance behind that. Results: Numerous studies described renal histology in cirrhosis (via biopsies at the time of liver transplantation or autopsies post-mortem) in addition to describing the kidney function by measuring serum creatinine whether at the time of the biopsy/autopsy or in the preceding few months. They reported glomerular IgA deposition rate of 50-90% making cirrhosis by far the most common cause of secondary IgA nephropathy (IgAN). There are two most accepted theories behind this high rate of glomerular deposition. The clearance theory focuses on the role of the liver in clearing IgA IC through the asialoglycoprotein receptor on the hepatocytes and the Fc receptor on Kupffer cells. The second theory in turn focuses more about the portacaval shunting that occurs with portal hypertension which shunts these IC and thus escaping the liver. Now, the clinical importance of secondary IgAN in cirrhotics remains the biggest dilemma. All studies reviewed agree that most patients are asymptomatic (>90%). If the disease becomes clinically active, hematuria is the most common presenting sign/symptom (exactly as primary IgAN) while nephrotic syndrome is much less common. Most studies also agree that the disease remains stable over many years with very few patients reported to progress to ESRD over years. Nonetheless, most studies remark that the activity of urine sediments predicts glomerular and mesangial cell injury. Conclusion: Secondary IgAN is the most common histological renal finding observed in cirrhotic patients that can rarely be symptomatic.

UNCOMMON ETIOLOGY OF CRYOGLOBULINEMIC RENAL VASCULITIS

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Background: West Nile (WN) virus is increasingly recognized as an important human pathogen in the North America with meningitis being the most clinical manifestations. Aim: to report a rare complication of WN virus infection. Clinical vignette: A 67-year-old gentleman with history of recurrent urinary tract infections presented to the emergency room complaining of fever and headache for two days. Upon presentation, his T was 102 F with otherwise normal physical and neurological exam. His WBC was 9.5 cells/ml with mildly elevated serum creatinine of 1.4 mg/dL from a normal baseline. Lumbar puncture was performed revealing aseptic meningitis with negative cultures but positive WN PCR so WN meningitis was diagnosed. While in the hospital, the patient continued to spike fevers with gradual worsening of serum creatinine so urinalysis was repeated showing 4+ proteinuria followed by 24 hour urine protein measurement that was 1.8 g/24 hours. At that point, complete rheumatologic work-up was performed including ANA, ANCA, C3, and C4 levels and returned negative but serum cryoglobulins were positive. Cryoglobulinemic vasculitis was the presumed etiology for the worsening in kidney function so HCV, HBV, and HIV tests were sent and came back negative so renal biopsy was performed revealing pauci-immune crescentic glomerulonephritis. WN induced cryoglobulinemic vasculitis was the presumed diagnosis and prednisone 60mg daily was started with gradual improvement in symptoms and kidney function. Two weeks later, the patient was free of symptoms with normal kidney function so he was discharged home on prednisone therapy. Conclusion: Most WN virus infections in humans are subclinical but overt disease can occur. Rare manifestations include myocarditis, pancreatitis and fulminant hepatitis where the involved organs are sites of high viral replication. Some evidence has accumulated recently suggesting renal tropism of WN virus. As far as our current knowledge extend, this is the first reported case of WN virus induced cryoglobulinemic vasculitis with classical renal affection.

CRYOGLOBULINEMIC MPGN IN A PATIENT WITH SCLERODERMA AND SJOGREN'S SYNDROME

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Cryoglobulinemic glomerulonephritis is typically associated with HCV infection (up to 90% of cases). We report a case of Hepatitis C negative cryoglobulinemic glomerulonephritis associated with scleroderma and Sjogren's syndrome.

CASE REPORT

The patient was a 54 y/o Hispanic female diagnosed with Raynaud's syndrome 8 yr ago. Later she developed features of sicca syndrome and scleroderma. Her serology was positive for high titer ANA, anti-Scl, and anti-SSA Ab and negative for anti-Sm, anti-dsDNA, anti-RNP Ab. She presented with one month of weight gain, edema, and accelerated hypertension; associated with myalgia and arthralgia. Work-ups showed hematuria, 2.5 g of proteinuria, but preserved renal function with Cr of 1 mg/dl. Additional tests demonstrated depressed C3&C4, elevated ESR (61 mm/hr), high titer RhF (105 IU/ml), and positive cryoglobulin. Hepatitis panel was negative. She also had urine free kappa light chain of 4.6g/dL and a monoclonal M-spike of 0.1g/dl of IgM kappa monoclonal protein. A bone marrow biopsy did not show overt proliferative disorder. Kidney biopsy revealed cryoglobulinemic MPGN with necrotizing arteritis.

The patient was treated with steroids and IV cyclophosphamide which after the third dose resulted in complete remission of proteinuria and resolution of monoclonal gammopathy. Subsequently, she was placed on maintenance azathioprine.

DISCUSSION

This is a case of Hep C negative type II cryoglobulinemia with positive RhF associated with scleroderma and Sjogren's syndrome.

Cryoglobulin production is also associated with disruption of B cell homeostasis that may lead to lymphoproliferative disorders. The association between cryoglobulinemia and Sjogren's syndrome has been reported with an incidence rate of as high as 16% in one case series. Its association with scleroderma is uncommon. Our patient has the clinical and serologic features of both scleroderma and Sjogren's syndrome.

MONOCLONAL GAMMOPATHY OF UNCERTAIN SIGNIFICANCE (MGUS) AND FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS)-A CASE REPORT

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FSGS incidence decreases in old age where as MGUS incidence increases with old age. It is unusual to find both conditions together.

A 72 year old Caucasian male with PMH for atrial fibrillation, HTN, hyperlipidemia and BPH, was electively admitted for evaluation of AKI. A week prior to hospitalization, he had presented to his PCP with chief complaints of malaise and worsening fatigue, and was found to have elevated serum Cr of 5.0 mg/dL (increased from 1.1 mg/dL 4 months ago). He was diagnosed with MGUS incidentally 2 years ago. Further work up revealed nephrotic range proteinuria with a spot urine sample showing protein: creatinine ratio of 4.8 g/mg. Renal US showed mildly increased echogenicity in both kidneys. Serum IgM level was 1.6g/dL. Otherwise immunoglobulin profile was normal. ANA, ANCA, Anti-GBM antibody, Hepatitis panel and HIV were negative. Renal biopsy showed severe collapsing-variant of FSGS. Cr continued to increase, with Cr = 6.4mg/dL on discharge.

The incidence of MGUS increases with age, affecting 3% of the adults ≥ 50 years. FSGS, collapsing variant has been predominantly linked to African American ethnicity, HIV status and exposure to uncommon chemicals. MGUS has been associated with Cryoglobulinemic glomerulonephritis, Immunoglobulin deposition disease, light chain nephropathy and amyloidosis *AJKD 2003; 42(1):87-95*. MGUS and collapsing-variant of FSGS are seldom seen to occur simultaneously. It may be related to the increased screening of protein electrophoresis in serum and/or urine in renal failure patients. Although, there has been one case report by Dingli et al showing an epidemiological as well as temporal relationship between MGUS and FSGS *AJKD 46:278-282*. The study proposed that they may be linked at the level of molecular pathogenesis.

MGUS molecular basis and FSGS pathogenesis are unclear. Both conditions may be found together due to the increased number of patients screened for protein electrophoresis routinely for renal failure.

MGN WITH ANCA-ASSOCIATED RPGN: A RARE DUAL GLOMERULOPATHY

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RPGN is an aggressive disease with a 1-yr mortality rate of up to 80% in the absence of immunosuppressive therapy. The prognosis of RPGN is dramatically improved by immunosuppressive regimens that include corticosteroids and cyclophosphamide.

We present a case of a 62 year old female with a history of asthma, hypertension and anemia secondary to MDS, who presented for evaluation of AKI and Nephrotic range proteinuria. Her serum creatinine had increased from 0.8 to 1.9 over a period of few weeks. She denied use of NSAIDS or other nephrotoxic agents. Urinalysis showed 3+ proteinuria and 7 RBCs/hpf with many RBC casts. A 24 hour urine collection showed a proteinuria of 7.3g. SPEP and UPEP were negative for M spike. Renal Ultrasound showed normal sized kidneys with a less than 2cm septated cyst in the right kidney. Serologic work up was positive for p-ANCA (>100 U/ml) , negative c-ANCA , normal C3, C4 , negative Hep B/C , HIV and negative ANA. She underwent a kidney biopsy which was consistent with Membranous GN with focal crescents and with no evidence of vasculitis. Work up for Malignancy revealed a normal colonoscopy, a negative Pap smear and a negative Mammogram. Given her history of smoking, she had a CT scan of chest which was also negative. MRI of kidneys revealed a benign cyst.

Treatment was initiated for ANCA associated RPGN. She was started on Prednisone after 3 days of pulse solumedrol and 2mg/kg of Oral cyclophosphamide. Anti-proteinuric therapy consisted of Cozaar 100mg daily, Lisinopril 20mg daily and tekturna 150mg daily. Cyclophosphamide was discontinued after eight months secondary to side effects, however, she remained on low dose prednisone. Her proteinuria had decreased to 1g/day and her creatinine had stabilized at 1.4 with eGFR of 46ml/min.

The co-existence of MGN and ANCA associated RPGN is rare and is likely to be coincidental.

P-ANCA ASSOCIATED CRESCENTIC GLOMERULONEPHRITIS IN SETTING OF RENAL CELL CARCINOMA

Biresh Kumar, Sheldon Chaffer. Department of Nephrology, Scott & White Hospital, Texas A&M Health Science Center, Temple, TX, USA. Renal cell carcinoma (RCC) has been described as occurring in association with a number of glomerular disorders including amyloidosis, mesangioproliferative glomerulonephritis, crescentic glomerulonephritis and extrarenal vasculitis. This represents, to the best of our knowledge, the second reported case of p-ANCA renal limited small vessel vasculitis occurring in association with RCC.

A 67 year old caucasian female with history of chronic tobacco and alcohol use presented on transfer with initial laboratory evaluation including BUN 100mg/dl, Creatinine 7.7mg/dl, platelet count of 37 and erythematous maculopapular rash over upper extremities. Five weeks before admission, patient was seen for a respiratory illness with laboratory showing Cr 3.5 and normal platelet count. One year prior to this admission, laboratory evaluation showed BUN 20 and Cr 0.8. During hospitalization serum Cr worsened to 13.0, with urine output remaining non-oliguric. Urinalysis showed 1+ proteinuria, > 50 RBC/hpf, WBC 3-9, no RBC casts. Clinically, the patient's acute kidney injury was felt to represent rapidly progressive glomerulonephritis (RPGN). No changes in mental status were noted and patient remained afebrile. Serologic evaluation included: negative ANA profile, C-ANCA, PR-3, and anti-GBM ab. Positive serologies included p-ANCA with anti MPO > 8.0. Renal US showed a 9.0 x 11.0 cm mass in the right kidney, confirmed by CT scan. Radical nephrectomy was performed with histopathology confirming pauci-immune crescentic glomerulonephritis and mixed clear and papillary cell carcinoma. The significance of ANCA in vasculitis is well established. The putative association of RPGN with RCC is rare. While the association between ANCA-positive crescentic glomerulonephritis and RCC may be a coincidental, previous reports have suggested that tumor antigens or cytokines, acting as sensitizing agents, may trigger ANCA associated small vessel vasculitis. Therefore, we report a second case of RCC occurring concomitantly with p-ANCA associated crescentic pauci-immune glomerulonephritis.

MEMBRANOUS NEPHROPATHY: A COMPLICATION OF IgG4-RELATED SYSTEMIC DISEASE

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INTRODUCTION: IgG4-related systemic disease (ISD) is an increasingly recognized autoimmune multisystem disorder that responds well to steroids.

CASE: A 72-year-old Caucasian male with a history of Castleman's disease presented with hematuria and underwent urologic work-up including a normal PSA and prostate biopsy showing acute on chronic prostatitis. Over the next few months he developed nausea, dark urine, and light stools. Physical exam demonstrated lower extremity edema, enlarged submandibular glands and inguinal lymphadenopathy. An ERCP, performed due to obstructive liver function tests and mild intra and extra hepatic biliary dilatation on CT scan, revealed a distal common bile duct stricture that was treated with a plastic stent. CA 19-9 was normal. Periapillary biopsies stained positive for IgG4, and serum IgG4 was elevated at 750 mg/dL (9-89). 24-hour urine showed nephrotic range proteinuria (>15 g/day). He was given escalating doses of diuretics and lisinopril. SCr rose to 4.7 mg/dL from normal within a month. Due to rapid progression of renal failure, kidney biopsy was performed which showed membranous nephropathy (MN) with eosinophilic and plasma cell interstitial infiltrates positive for IgG4. Retrospectively, prostate and lymph node biopsies also stained positive for IgG4. After a trial of steroids and rituximab was unsuccessful, dialysis was initiated due to progressive renal failure.

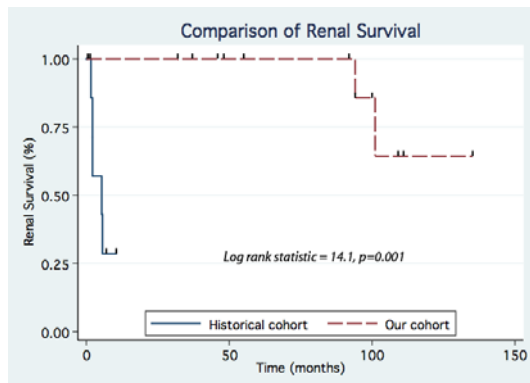
DISCUSSION: This case demonstrates the ability of ISD to affect multiple organ systems, including the rare complication of rapidly progressive MN. It implicates IgG4 in the pathogenesis of certain forms of secondary MN and suggests that ISD may mimic Castleman's disease.

CONCLUSIONS: Physician education regarding ISD needs improvement. The diagnosis of ISD is challenging and requires a high clinical suspicion as well as appropriate serologic tests and IgG4 stained biopsies. Early institution of steroid treatment may improve patient outcomes.

RENAL PROGNOSIS IN HIV HCV CO-INFECTED PATIENTS IN THE HAART ERA

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A poor prognosis among patients with glomerular disease related to HIV HCV has been previously demonstrated. We reviewed the renal diagnosis and prognosis of 13 co-infected patients with biopsy proven glomerular disease since the introduction of widespread use of HAART who had mean follow up of 81 ± 33 months and a mean follow up of 18 ± 15 months from the time of the biopsy. Among our 13 pts (7 males and 6 females), there were 6 hypertensive, 3 hypertensive and diabetic, 1 diabetic, 10 smokers, while all were illicit drug users with 9 using



both cocaine and heroin. Only 3 pts had edema and 9 pts received HAART therapy. At the time of biopsy, the mean serum creatinine was 2.1 ± 1.3 , mean urine protein 1.8 ± 1.7 gm/d, and mean urinary albumin excretion

1334 ± 1127 mg/g of creatinine. Rheumatoid factors were normal for 11/11 pts while 7 pts had low CH50. Only one pt had a low C4 and one with low C3 and cryoglobulinemia. Among our cohort, there were 4 cases of FSGS, 2 cases of diabetic nephropathy, 5 cases of mesangioproliferative GN of which 3 had features of MPGN. Three pts needed renal replacement and 3 were lost to follow up. Our cohort of patients demonstrated significantly lower need for renal replacement therapy than previously reported (see survival curve).

THREE DECADES OF PROGRESS IN TREATING CHILDHOOD ONSET LUPUS NEPHRITIS

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Treatment of severe lupus nephritis (LN) has evolved during recent decades with improved survival demonstrated in adults after the introduction of intravenous cyclophosphamide (CYC) in the 1980's and mycophenolate mofetil (MMF) in the 1990's. Childhood onset lupus nephritis carries a worse prognosis and controlled treatment trials have not been conducted.

The objective of this study was to compare renal survival in a cohort of pediatric patients followed for 3 decades according to progressive treatment regimens that included pulse solumedrol, CYC, MMF and rituximab (RTX).

A retrospective analysis was performed on 117 pediatric patients with the diagnosis of systemic lupus erythematosus (SLE) at our center. Patients with \geq WHO class 3 were designated as having severe LN. The end point for renal survival was progression to end stage kidney disease (ESKD).

Of the initial cohort, 79 (63 female; 80%) had severe LN. Average age of onset was 12.5 ± 3.0 years with a mean follow-up of 5.6 ± 3.5 years. There was a nonwhite ethnic/racial predominance of 92%. Twenty-six (33%) progressed to ESKD. During the first 2 decades, all patients received corticosteroids and CYC. When MMF was added, renal survival improved from a median of 6 to 12 years ($p < 0.003$). Patients treated with MMF alone or with RTX have experienced no patient or renal demise.

Renal survival in childhood onset LN has improved during the past 3 decades with the addition of MMF to the treatment regimen. Continued experience with this as an induction agent with or without the use of RTX requires further investigation. Collaborative trials in pediatric patients are very much warranted.

“WILL THE REAL DIAGNOSIS PLEASE LINE UP”- A RARE CASE OF ANTI-GBM NEGATIVE GOODPASTURE’S SYNDROME

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Goodpasture's syndrome (GPS) is a rare cause of rapidly progressive glomerulonephritis and pulmonary hemorrhage. The diagnosis often relies on the use of immunoassays to detect circulating anti-GBM antibodies. Our case illustrates that even in the absence of these antibodies if the clinical suspicion is high GPS cannot be ruled out.

A 29 yr old male with history of smoking, presented with hemoptysis and shortness of breath. Initial labs revealed: BUN 37mg/dl , creatinine 2.1mg/dl and hemoglobin 9.7g/dl). Urinalysis revealed red cell casts and the chest xray showed diffuse lung infiltration. Serology was negative for ANCA, ANA, and anti-GBM antibodies. The patient was empirically started on plasmapheresis, steroids and cytoxan. Hemodialysis was initiated because of worsening renal function tests (BUN 129mg/dl & creatinine 5.2mg/dl). With no firm diagnosis, a lung and kidney biopsy was performed. The lung biopsy showed diffuse alveolar hemorrhage while the renal biopsy performed showed no evidence of crescents and no other characteristic findings as it was performed after initiation of treatment. IF could not be performed on either because of insufficient tissue. Though the kidney function improved his respiratory status deteriorated and he died. An autopsy performed thereafter confirmed the diagnosis of GPS by demonstration of linear deposits in lung & kidney tissue. Lerner et al. established that anti-GBM antibodies are pathognomic of GPS. These are usually IgG in nature. However, sometimes the antibodies are non-IgG in nature. In such situations, the usual immunoassay tests are falsely negative & immunohistological evaluation of the kidney biopsy confirms the diagnosis. Alternative ways to detect such antibodies include biosensor analysis. Our case demonstrates an uncommon situation where an accurate diagnosis could not be made even though standard protocols for diagnosis and treatment were followed. This case also highlights the need to recognize anti-GBM negative GPS in the appropriate clinical situation.

A CASE REPORT OF HIV ASSOCIATED IMMUNE COMPLEX GLOMERULONEPHRITIS IN AN AFRICAN-AMERICAN MALE

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HIV-associated Nephropathy is typically described as a type of Focal Sclerosing Glomerulonephrosis, which is predominant in African-American patients infected with HIV. Patients typically present with nephrotic-range proteinuria, enlarged kidneys on renal ultrasound, and are usually normotensive. On renal biopsy patients have dilated tubules, reticular aggregates and the collapsing-variant of FSGS. Immune-complex glomerulonephritis has been described in HIV-infected patients but is typically described in Caucasian patients with concomitant Hepatitis infection. We present a case report of a young HIV + African-American patient with an immune-complex glomerulonephritis.

A 32 y/o African-American male was referred to nephrology clinic from ID clinic after multiple urinalyses over a year's time were positive for microscopic hematuria and proteinuria. His past medical history was significant for poorly controlled hypertension and HIV of 10 years duration. His last CD 4 count was 11 and he had a significant HIV viral load (approximately 240,000 copies/ml). His glomerular filtration rate was in the low 60s. Hepatitis B and C serologies, serum protein electrophoresis and urine protein electrophoresis were negative. Protein to creatinine ratio was notable for 650mg/24 hr. Complement levels were checked and both C3 and C4 were decreased significantly.

Renal biopsy was performed and was notable for immunofluorescent granular mesangial staining for C3 and IgG. He had subepithelial immune complex deposits, 10-20% fibrosis and a moderate lymphocytic infiltrate. Review of the international literature, reveals that immune complex glomerulonephritis is not uncommon in patients of African heritage associated with HIV, despite prevailing opinions otherwise in the United States. There is also evidence that immune-complex glomerulonephritis can occur without co-infection with hepatitis. The patient was urged to be compliant with both his HAART and his blood pressure medications. On repeat evaluation his microscopic hematuria had markedly improved, as had his GFR.

CLINICAL EXPERIENCE IN THE THERAPEUTIC CHALLENGE OF COLLAPSING FOCAL SEGMENTAL GLOMERULOSCLEROSIS (FSGS)

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There is no clear consensus in the management of idiopathic FSGS collapsing variant.

We report our experience with 2 patients who were diagnosed with idiopathic FSGS collapsing variant. The 1st patient is a 33 year old African American woman with massive proteinuria (urine protein/creatinine (UP/Cr) 18.6 mg/mg) and normal Sr Cr (serum creatinine) 0.8 mg/dl. She was treated with prednisone 60 mg/day for 6 months and lisinopril with a positive response (UP/Cr 8 mg/mg) and her Sr Cr remained normal. She was then initiated on Cyclosporine 150 mg twice daily and the prednisone was tapered off. Ten months later her proteinuria improved (UP/Cr 3.3 mg/mg) and Sr Cr remained stable at 1.1 mg/dl. The 2nd patient is a 20 year old Hispanic man with acute kidney injury (Sr Cr 1.5 mg/dl) and massive proteinuria (UP/Cr 28 mg/mg). He was treated with lisinopril and prednisone 60 mg/day for 6 months with a positive response (UP/Cr 12 mg/mg), but his kidney function deteriorated (Sr Cr 1.9 mg/dl). Cyclosporine 100 mg twice daily was initiated and prednisone was discontinued. After 6 months his proteinuria improved remarkably (UP/Cr 3.8 mg/mg) and Sr Cr has stabilized at 3.4 mg/dl.

Thus we report 2 cases of collapsing FSGS whom we treated similarly with a trial of steroids for 6 months followed by Cyclosporine and had partial response with stable renal function.

IMMUNECOMPLEX GLOMERULONEPHRITIS WITH LUPUS LIKE FEATURES IN A HIV POSITIVE MALE

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Immunecomplex glomerulonephritis with lupus like features without clinical or serological evidence of Lupus in HIV infected patients is an interesting pattern rarely reported in literature.

We present a 26 yr old, previously healthy African American man transferred to our facility for management of acute kidney injury. Abdominal exam revealed mild epigastric tenderness. Otherwise physical examination was unremarkable. Laboratory studies demonstrated: BUN 33 mg/dl; creatinine, 4.7 mg/dl with normal complete blood counts and liver enzymes. Urinalysis showed 100 mg/dL of protein; spot urine protein:creatinine ratio 2.1. Patient tested positive for HIV by western blot. The RNA viral load was 34,100 copies/l and CD4-positive lymphocyte count was 193/ml. Hepatitis B surface antigen was positive. ANA, ds-DNA, Hepatitis C RNA were negative. Serum C4 was low at 10.4mg/dl and C3 normal. Renal biopsy revealed findings consistent with acute tubular necrosis with mild mesangial expansion; mesangial immune complex deposition, with staining for IgG, C1q, C3, kappa and lambda light chains on immunofluorescence and mesangial, few paramesangial electron dense deposits with tubuloreticular inclusions on electron microscopy. Lack of clinical or serological evidence of lupus made mesangial lupus nephritis unlikely. A final diagnosis of acute tubular necrosis with HIV associated immune complex glomerulonephritis with “lupus” like features was made. The patient was discharged with a BUN 13mg/dl and creatinine 2.3mg/dl. A repeat renal function eight weeks later showed a dramatic improvement in kidney function (BUN 12mg/dl and creatinine 1.07mg/dl) without any active intervention.

There is presently little information known about the etiology, treatment, or outcome of lupus-like glomerulonephritis. A few sporadic case reports describe improvement in renal function with the use of anti-retroviral drugs, ACE inhibitors or corticosteroids. Better understanding of the pathophysiology of this disease will help solve this diagnostic dilemma and this entity should be considered in appropriate clinical settings.

ENDOCARDITIS AND KIDNEY MASS IN WEGENER'S GRANULOMATOSIS

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Wegener's granulomatosis is a disease that can affect many systems; upper and lower airways as well as kidneys are the most common involved organs. We report a case of Wegener's granulomatosis associated with pulmonic valve endocarditis and a kidney mass.

A 22 yr old male presented to an outside facility complaining of hemoptysis and respiratory distress associated with flank pain; he had had different chronic symptoms which included low-grade fever, fatigue, earaches and 40lb weight loss over a period of 2 months. Initial evaluation showed WBC 12.3 and urine with mild microscopic hematuria (3-4 rbc/hpf) and leukocyturia (15 - 25 wbc/hpf) and normal serum creatinine. Abdominal CT with contrast showed a right kidney mass. Chest CT showed multiple bilateral necrotic lesions suspicious for septic emboli. A TEE was performed, which visualized a thickened pulmonic valve with vegetation. Patient's clinical condition deteriorated despite empiric antibiotics and was transferred to our hospital. Upon arrival patient's physical exam revealed purpuric lesions in his extremities and severe respiratory distress requiring intubation and mechanical ventilation; repeat U/A showed 33RBC /hpf, 23 WBC/hpf, protein 100, granular casts, serum creatinine 1.6 mg/dl, WBC of 23,000, negative blood cultures and urine culture. Outside hospital serologies showed high C- ANCA titers (1: 320), and PR3 was > 100. Medical management with intravenous corticosteroids and cyclophosphamide along with plasmapheresis was started. Renal biopsy confirmed focal necrotizing glomerulonephritis. Patient had significant improvement and eventually was extubated and renal function recovered. Repeat TEE and abdominal CT scan done 2 months after initial presentation showed resolution of pulmonic valve endocarditis and decrease in size of the kidney mass.

Cardiac involvement is rare in Wegener's granulomatosis and can be affected in many different ways. Culture negative endocarditis in patients with minimal risk factors for bacterial endocarditis should raise suspicion for non-infective causes and prompt a vasculitis work up.

FIRST-TRIMESTER PREGNANCY LOSS ASSOCIATED WITH PLASMA EXCHANGE THERAPY

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A 19 year old G2P1 female with a 6 year history of systemic lupus presented to an outpatient clinic for progressive dyspnea and hemoptysis. Six months previously, a renal biopsy confirmed lupus nephritis; she was currently on mycophenolate and prednisone. A screening pregnancy test was positive, and ultrasound confirmed a five-week intrauterine gestation. Mycophenolate was discontinued, and her prednisone dose was increased. She was treated with antibiotic therapy for suspected bronchitis for three weeks with minimal improvement, and consequently was admitted for piperacillin therapy. Chest X-ray revealed bilateral infiltrates, and she was transferred to our institution at 8 weeks gestation for further evaluation.

On arrival she was noted to be hypoxemic, with a hemoglobin of 6.8 g/dL. Methylprednisolone was started, and bronchoscopy revealed extensive intra-alveolar hemorrhage. Blood and urine cultures were negative, and echocardiography revealed mild pulmonary hypertension with a right ventricular systolic pressure of 50 mmHg. The patient developed renal failure, and started a six-day course of plasma exchange (PLEX) therapy with pulse-dose methylprednisolone. Over the next four days, serial β -hCG and progesterone levels declined from 48,152 mIU/mL and 10 ng/mL to 12,686 mIU/mL and 5 ng/mL respectively, at which time she underwent a D&C for an incomplete abortion. Her pulmonary and renal function recovered; she was discharged on mycophenolate and prednisone.

This case illustrates a potential risk of PLEX therapy during the first trimester. Serial β -hCG and progesterone levels successively declined during the course of PLEX treatment, suggesting iatrogenic elimination of hormones critical to maintenance of early pregnancy. Although PLEX is generally considered to be safe during pregnancy, the majority of cases have been reported in the second and third trimesters. While her pregnancy loss may have been secondary to her underlying disease process, we suggest that physicians exercise caution when using PLEX in the first trimester.

IGA-DOMINANT STAPHYLOCOCCUS INFECTION- ASSOCIATED GLOMERULONEPHRITIS: TWO CASE REPORTS AND REVIEW OF THE LITERATURE

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The mesangial deposition of IgA is rarely described with diffuse proliferative glomerulonephritis (DPGN) associated with staphylococcus aureus (SA) infection. We hereby report 2 cases and review the 44 cases reported in the literature.

A 73 year old man with right pleural effusion and methicillin sensitive SA bacteremia developed acute kidney injury (AKI) with macroscopic hematuria and nephrotic range proteinuria. Complements were normal. Kidney biopsy showed DPGN with dominant mesangial IgA deposits. He required hemodialysis despite eradication of the infection with vancomycin and short course of steroid therapy.

A 69 year old woman developed AKI on top of chronic kidney disease after methicillin resistant SA (MRSA) bacteremia secondary to pacemaker lead infection. Urine studies showed RBC casts and nephrotic range proteinuria. Complements were normal. Kidney biopsy showed an IgA-dominant DPGN. Renal failure improved with eradication of the infection with oxacillin.

Literature review of 44 clinically similar cases shows that these patients are elderly (mean age 65) with nephrotic range proteinuria and normal complement present in 47% and 71% respectively. MRSA (66%) is the most common pathogen involved with latent period ranging from 1 to 16 weeks. Diffuse mesangial proliferation is commonly found while crescents are uncommon (18%). 2+ IgA staining is reported in 66 % of cases and IgG is absent in 32% of cases. Treatment with antibiotics yielded 60 % recovery. Renal replacement therapy was required in 26% of cases. Presence of diabetes, low serum complements, low serum creatinine, tubular atrophy and interstitial fibrosis in renal biopsy were associated with poor prognosis.

In conclusion, nephrologists should consider staphylococcal associated DPGN that can mimic primary IgA nephropathy in an elderly patient with AKI, hematuria, proteinuria and mesangial IgA deposit with recent or remote history of SA infection.

EPOETIN ALPHA ADMINISTRATION WAS REDUCED TO ONCE PER WEEK WITHOUT COMPROMISING HEMOGLOBIN TARGETS

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To enhance patient comfort, a pilot program was initiated at HUMC's chronic outpatient hemodialysis unit, to assess the feasibility of reducing the frequency of subcutaneous administration of Epoetin Alpha (EPO), while meeting the nationally recommended hemoglobin (Hgb) target value of 11 to 12 g/dl.

Nephrologists were encouraged to switch their patients from the conventional 3 times/week dose to a once weekly equivalent dose. Subsequent dose adjustments were made when necessary, in tandem with changes in the twice monthly tested Hgb values.

Investigated was the prevalent HD patient population, excluded were prevalent patients requiring less than once per week dose of EPO, and HD patients requiring more than 90,000 iu/wk.

Patients were divided into 3 groups: Group 1: One injection/week, Group 2: > 1 to < 3 inj. /week, and Group 3: 3 inj./week.

At the end 9 months, mean Hgb values for the three groups were as follows: Group 1 (n = 61): 11.33 g/dl (95% CI 11.16 – 11.50), Group 2 (n = 104): 11.27 g/dl (95% CI 11.12 – 11.42), Group 3 (n = 41): 10.85 g/dl (95% CI 10.61 – 11.09). For EPO usage, see tabular results below.

One-way Anova	Group1 (n = 61)	Group 2 (n = 104)	Group 3 (n = 41)	P value	F
Median EPO dose per week	3,174	4,168	5,934	0.0001	11

Preliminary data suggests that reducing the frequency of EPO administration from 3 times per week to an equivalent dosage once per week, did not prevent us from achieving a mean target Hgb value between 11 g to 12g/dl, and could also result in a reduction of EPO dose.

VARIANT HEMOGLOBIN MAY AFFECT ERYTHROPOIETIN RESPONSE IN AFRICAN-AMERICANS RECEIVING HEMODIALYSIS

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African-Americans receiving hemodialysis (HD) require larger doses of erythropoiesis-stimulating agents (ESA) for the treatment of anemia. We recently reported heterozygosity for variant hemoglobin to be quite common in our African-American HD population. We hypothesized abnormal hemoglobin variants could contribute to erythropoietin hyporesponsiveness among African-Americans.

We performed a cross-sectional analysis of 154 African-American HD patients with known hemoglobin phenotype. Clinical laboratory data from August 1 to October 31, 2008 were used in multivariate logistic and poisson regression models to determine the effect of abnormal hemoglobin on ESA hyporesponsiveness, defined as $\geq 6,500$ units of erythropoietin/treatment (or $\geq 19,500$ u/wk). Covariates in initial analyses included age, sex, dialysis unit, iron dose, missed treatments, dialysis vintage, dialysis adequacy, albumin, ferritin, iron saturation, iPTH, dialysis unit, smoking, alcohol abuse, history of GI bleed and diabetes. We included covariates demonstrated to be potential confounders in our data set or established in the literature. Missed treatments, dialysis vintage, Kt/V, albumin, ferritin, iron saturation, iPTH, dialysis unit, history of GI bleed were maintained in the fully adjusted model.

Of 154 patients, 34 had abnormal hemoglobin variants (sickle cell trait (24), hemoglobin C trait (9), and beta-thalassemia trait (1)). Presence of abnormal hemoglobin variants was associated with ESA hyporesponsiveness with OR 3.04 (1.18-7.82, $p=0.02$) and HR 1.99 (1.18-3.23, $p=0.009$).

Abnormal hemoglobin phenotype among African-Americans receiving HD could lead to ESA resistance. Hemoglobin phenotype may identify those at risk for receiving high-dose ESAs, which have been associated with greater morbidity and mortality. Due to the small size of our study and other limitations, our findings would be best confirmed in a larger prospective cohort.

PARENTERAL IRON THERAPY AND INFECTION RELATED HOSPITALIZATIONS IN ESRD PATIENTS ON HEMODIALYSIS

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Parenteral iron (IV Fe) is an efficacious adjunctive therapy to manage anemia in ESRD patients besides Erythropoiesis Stimulating Agents. Limited evidence exists regarding IV Fe use and risk of infections.

We conducted a retrospective observational study on 105 ESRD patients on maintenance hemodialysis between 05/2006 and 04/2009 to evaluate the association between IV Fe therapy and hospitalizations related to infection [defined as vascular access related (VAR) and non-vascular access related (NVAR)]. Those with follow up for <90 days were excluded. Cases were defined as those hospitalized for both VAR & NVAR infections while those with other disease-related hospitalization or never hospitalized served as controls. Data was analyzed using 2-tailed student t-test, chi square and logistic regression.

75% of the all patients were hospitalized at least once. Of the 372 all-cause hospitalizations, 6.5% and 18% were attributed to VAR and NVAR infections respectively. Among NVAR pulmonary, GI, urinary infections accounted for 35%, 20% and 12% respectively. Cases (n=49) and controls (n=56) had comparable median age (years) 48.2 vs. 52 and follow up (days) 852 vs. 546. The 2 groups had similar proportion of diabetes 38.8% vs. 42.8%, HIV 6% vs. 8.9%, Hepatitis C 16.3% vs. 12.5%, malignancy 12.2% vs. 8.9%, autoimmune disease and immunosuppressive therapy 10.2% vs. 3.5%, (p=NS for all the above comparisons). Both groups had similar median lab values: Hemoglobin (g/dL) 11.8 vs. 11.7, ferritin (ng/mL) 584.8 vs. 583.4, transferrin saturation 20.6% vs. 23.2%, albumin (g/dL) 3.88 vs. 3.78, Glycated (A1C) Hgb 7.1% vs. 6.5%, URR 70.6% vs. 70.8% (p=NS for all lab values comparisons). Using multivariate logistic regression adjusted for demographics, vascular access and above parameters, cases with VAR/NVAR had slightly increased odds of receiving higher doses of IV Fe replacement (103925 mg vs. 77635 mg, OR 1.0004, p=0.02) and cumulative dose (213790 mg vs. 178440 mg, OR 1.0002, p=0.01).

Patients hospitalized for infections received significantly higher amount of replacement IV Fe therapy and cumulative dose, though this should be interpreted cautiously as odd ratios are only slightly high.

COMPUTER-BASED MODEL FOR EVALUATION AND VALIDATION OF CURRENT ANEMIA MANAGEMENT PROTOCOLS IN HEMODIALYSIS

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Overwhelming evidence links patient outcomes with hemoglobin (Hgb) values in patients on hemodialysis. Hgb values below a certain target range (10.0 to 12.0 g/dL) are associated with increased risk of hospitalization and death from cardiovascular, cerebrovascular as well as infectious causes while being above this range adds no additional cardiovascular benefit and may actually increase mortality. Based on this evidence, development of a standardized erythropoietin dosing algorithm for improving anemia management in end stage renal disease (ESRD) may greatly improve patient outcomes.

We tested the hypothesis that a computer based simulation tool can be used to evaluate anemia management algorithms. We evaluated multiple currently used protocols using a model of Hgb response and computer implementation of each of the algorithms. The model incorporates three variables that are predictive of Hgb response. Estimates of these variables were determined using data collected from patients at the University of Louisville Kidney Disease Program. Data generated by simulations were analyzed by inspection of graphical displays and by summary statistics. Using these techniques, we were able to identify trends and assess the ability of each protocol to maintain Hgb within the target range. We determined that protocols which included the change in Hgb when adjusting erythropoietin doses were more likely to maintain Hgb at a stable value within target range. We also observed that protocols with higher starting doses of erythropoietin had greater variability with larger amplitude and more frequent peaks and troughs. Furthermore, we were able to show that by decreasing the starting dose we were able to improve accuracy.

Using the Hgb response model and computer implementation of the algorithms we were able to critically evaluate the ability of current protocols to effectively manage anemia in patients on hemodialysis. Ultimately, information obtained using our model may aid in revision of current protocols to construct new, more effective algorithms that can be used to improve anemia management in patients on hemodialysis and thereby improve patient outcomes.

ADMINISTRATION OF ERGOCALCIFEROL: EFFECT ON ANEMIA MANAGEMENT IN END STAGE RENAL DISEASE

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Previous studies have demonstrated that lower 25 hydroxyvitamin D (25(OH)D) levels are independently associated with lower hemoglobin (Hb) concentrations in chronic kidney disease (CKD) patients. However, the effect of Vitamin D₂ (Ergocalciferol) repletion on anemia of CKD is unknown. We sought to observe, in a single site hemodialysis center, the effects of Ergocalciferol supplementation on Hb levels and erythropoietin (EPO) dosing requirements in patients with end stage renal disease (ESRD).

Patients from a single satellite dialysis center were screened with baseline 25(OH)D values. Patients with 25(OH)D levels < 30 ng/ml were prescribed 50,000 units of Ergocalciferol weekly, while patients with levels > 30 ng/ml were given 50,000 units of Ergocalciferol monthly. Subjects were then followed prospectively with baseline and quarterly labs for 6 months. Important variables compared from baseline to month 6 included 25(OH)D, Hb, and EPO dose.

The change from month 0 to month 6 was calculated as value at month 6 minus the value at month 0. A signed rank test was used to assess the significance of change. The Spearman's correlation coefficient was computed to assess the correlation between changes of different variables. All statistical tests were two sided. A $p < 0.05$ was considered statistically significant.

Of 92 HD patients, 72 (78%) had 25(OH)D levels < 30 at baseline. During the 6 month follow up, 25(OH)D significantly increased from an average of 23.5 ± 11.8 to 29.2 ± 14.1 ng/ml, ($p = 0.0003$). The absolute change was an average of 5.74 (median: 6.05) ($p = 0.0003$). Total EPO dose significantly decreased at an average of 60,252 units monthly (median: 36,100, $p = 0.018$). Average Hb change from month 0 to 6 was not statistically significant ($p = 0.108$). As expected, Hb change from month 0 to 6 was positively correlated with the change in the total EPO dose ($p = 0.0014$). There was no significant relationship between the change in 25(OH)D levels and either Hb or total EPO dose given.

In a 6 month study, administration of Ergocalciferol was associated with patients requiring lower doses of EPO while still maintaining a stable level of Hb. In this short study, while the effect was not related directly to 25(OH)D levels, further studies are needed to explore the impact of 25(OH)D repletion on anemia in ESRD.

EFFECTS OF ERYTHROPOIETIN STIMULATING AGENT (ESA) AUTOMATED ADJUSTMENT PROTOCOLS ON HEMOGLOBIN (HGB) LEVELS AND MORTALITY IN ESRD PATIENTS.

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ESA trials have demonstrated increased mortality in patients with higher hematocrit levels. As a result, the FDA instituted a Black Box Warning for the ESA administration. We evaluated Hgb levels and mortality following alterations in ESA protocols instituted after the FDA recommendations with goals to reduce patients with Hb>13g/dl.

We performed a retrospective cohort study in over 7000 patients treated in 120 dialysis facilities at DSI Renal, Inc. from Sept., 2006 to Mar., 2008. Comparisons were made among three groups; the original ESA anemia protocol-A (Sep-April (pre-FDA)), protocol-B (May-Aug) and protocol-C (Sep-Mar (modified to optimize Hgb target 11–12g/dL)). The intent of protocol-B was to reduce the % of dialysis patients with Hb>13g/dL. The Kruskal-Wallis test was used for statistical analysis.

	DSI Renal ESA Protocols		Target
Protocol	Hold ESA	Resume ESA *	Hgb
A	Hgb>13.3 g/dl	Hgb<13.00 g/dl	12-13 g/dl
B	Hgb>12.5 g/dl	Hgb<11.75 g/dl	11-12 g/dl
C	Hgb>13.0 g/dl	Hgb<12.50 g/dl	11-12 g/dl
*Resume ESA at 2 levels below the current dose			

The Mean hemoglobin levels varied by protocol: A: 11.77 g/dL, B: 11.45g/dL and C: 11.66g/dL (p=0.01). The percentage of patients with Hb <11g/dL differed by protocol: A: 23%, B: 33% and C: 25% (p=0.01). The percentage of patients with Hb>13g/dL differed by protocol: A: 14%, B: 8%, C: 11% (p=0.01). Overall monthly mortality rate (deaths/100 patient-months) differed by protocol time: A: 1.68, B: 1.40 and C: 1.54 (p=0.01).

Overall monthly mortality rates decreased after institution of ESA automated protocols designed to decrease hemoglobin levels in patients served by a large dialysis provider.

RELATIONSHIP BETWEEN STATINS USE AND ERYTHROPOIETIN RESISTANCE IN ESRD PATIENTS

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Erythropoietin sensitivity is a variable and incompletely understood phenomenon among patients with ESRD, largely due to the known chronic inflammatory state mediated by cytokines. Approx. 10% of the patients on erythropoietin show very poor responsiveness to the drug. HMG-CoA reductase inhibitors represent the most important class of lipid-lowering agents with benefits ranging from antioxidant to anti-inflammatory and immunomodulatory properties. They also decrease serum prohepcidin levels, thus affecting plasma iron availability. Though the topic was not thoroughly investigated, 2 small sample sized studies suggested that patients who are taking statins have a reduced resistance to erythropoietin.

We conducted a retrospective case control study using data from 15 centers from the Center for Dialysis Care located in Northeast Ohio. Approx. 2,500 HD patients were included in the final sample, and the variables analyzed included demographics, medications, lipid profile, ferritin, iron, hemoglobin levels, transfusion history and relevant co-morbid conditions. Patients given less than 50 doses of erythropoietin were excluded from the final sample. SPSS was used to analyze the data. Preliminary results from one center (n=160) showed no statistically significant difference between the amount of erythropoietin used for patients on statins versus those who were not ($p = 0.64$), but significantly higher HCT values for patients on statins ($p=0.03$). Regression analysis will be used to estimate the factors that predict the quantity of erythropoietin use in the final sample controlling for confounding factors. Clarifying the relationship between two commonly used drugs in the ESRD population can be of tremendous public health importance.

	On Statins (N = 65)	No Statins (N=95)	P Value
Mean HCT	35.28	36.18	0.038
Mean Erythropoietin	7114.8	6698.1	0.643

FERUMOXYTOL IN ENDSTAGE RENAL DISEASE: REPLETION AND MAINTENANCE DOSING

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In patients with ESRD, appropriate iron utilization is integral to achieving optimal anemia management. Changes in dosing and/or frequency of iron administration may impact hemoglobin stability, epoetin utilization and/or iron parameters. Ferumoxytol is an intravenous (IV) iron that received FDA approval in 2009 for the treatment of iron deficiency anemia in patients with chronic kidney disease. 114 patients on dialysis participated in Phase 3 trials and received up to two 510 mg doses, dosed 3 – 8 days apart. IV iron is often administered as maintenance dosing after repletion for in-center hemodialysis patients. The purpose of this prospective open-label six month trial is to determine the safety and efficacy of a maintenance IV iron protocol utilizing ferumoxytol.

On October 1, 2009, all dialysis patients at two dialysis facilities (in-center N = 165, home dialysis N = 25) were converted from their existing iron protocol to a ferumoxytol repletion and maintenance protocol, consisting of two 510 mg doses for patients with TSAT < 25% and/or serum ferritin (SF) < 200, and maintenance dosing of 510 mg every other month, frequency adjusted to maintain TSAT 25 – 35% and SF 200 – 800 ng/mL. TSAT, SF, Hgb and epoetin dose were recorded at baseline and monthly. Patients were observed for adverse reactions.

To date, 129 doses of ferumoxytol 510 mg were administered to 79 patients in a 2 month period, 42 patients receiving repletion and 37 receiving maintenance doses. Three adverse events (hypotension, hypotension /itching, coughing/sneezing) were observed in three patients, one felt by the patient's physician to be possibly ferumoxytol related. Trending of laboratory results and epoetin utilization is being performed.

In conclusion, use of ferumoxytol in a dialysis facility driven protocol is easy to accomplish, and clearly has advantages for patients requiring repletion dosing. However, further study is needed to determine optimal maintenance dosing.

VITAMIN D SUPPLEMENTATION TO IMPROVE LEVELS OF ANEMIA IN ESRD PATIENTS

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Introduction: The use of ergocalciferol (VD2) supplementation on a cohort of ESRD patients on hemodialysis (HD) who are deficient in 25-hydroxyvitamin D (25-D) levels was studied to improve levels of anemia and decrease dosage requirements of recombinant human erythropoietin (EPO).

Methods: We prospectively followed a cohort of 25-D deficient HD patients. Baseline 25-D levels were obtained for patients who received HD >6 months in our unit. Patients with 25-D levels of 10-30pg/mL received 50,000IU x 4 doses. Patients with 25-D levels <10pg/mL received 50,000IU x 6 doses over 4 months. Monthly 25-D levels, Hgb levels, and mean monthly dose of EPO were recorded at baseline and after VD2 supplementation.

Results: 81 HD patients met our inclusion criteria for this study.

Mean baseline 25-D level was 15.3 ± 7.1 pg/mL for study patients compared to a mean of 28.5 ± 8.6 pg/mL after 4 months of VD2 supplementation. 36 patients (44%) achieved final 25-D levels ≥ 30 pg/mL, these patients were considered responders to VD2 treatment.

The responder subgroup had mean Hgb levels of 11.5 ± 0.7 gm/dL at baseline, 11.5 ± 0.9 gm/dL during treatment, and 11.2 ± 0.8 gm/dL at follow up. No significant statistical improvement in mean Hgb values during treatment and at follow up were found between responder subgroup compared to the non-responders subgroup to VD2 supplementation.

In the subgroup of responders mean baseline EPO dose $33,348 \pm 45,039$ units/month and mean EPO dose during follow up $29,819 \pm 31,848$ units/month. No significant statistical decrease in mean EPO dose during the follow up periods was found between the responder versus non-responder subgroups.

Conclusion: The responders to VD2 treatment did not show significant statistical improvement in Hgb levels or mean monthly EPO dosage over the non-responder subgroup.

LONG-TERM CLINICAL EXPERIENCE WITH HEMATIDE™

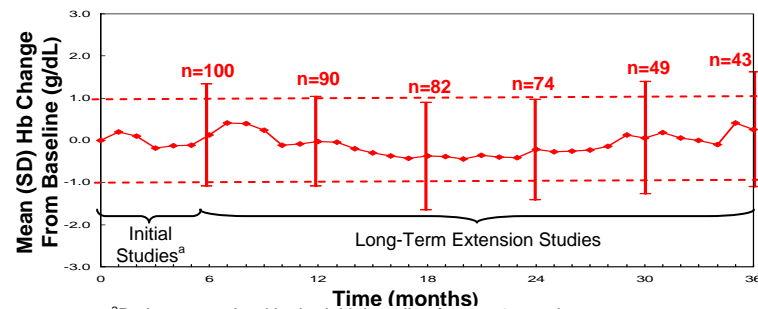
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Hematide™ is the first synthetic, peptidic ESA that is in phase 3 development for the treatment of anemia associated with CKD. Results from 2 initial phase 2 (completed) studies and 2 ongoing long-term extension studies with once-monthly Hematide are reported herein.

This unplanned analysis included 100 HD patients who had stable Hb levels on epoetin (baseline) before entry into the initial studies. Each patient received ≥ 24 weeks of Hematide treatment in 1 of 2 initial studies and continued to receive Hematide in 1 of 2 open-label extension studies; the Hematide dose was adjusted as necessary to maintain a Hb level of 10 to 12 g/dL (updated from the original 2006 protocol target of 11 to 13 g/dL).

Mean Hb levels were maintained within 1 g/dL from baseline (Figure). AEs were reported for 93 patients (93%). Of these patients, 7 (7%) had AEs possibly related to Hematide; each type of AE occurred only once. Serious AEs were reported in 67 patients (67%). A single patient experienced a serious AE that was considered possibly Hematide related (fatal pulmonary embolism).



^aPatients remained in the initial studies for 5 to 8 months.

These results indicate that long-term once-monthly Hematide treatment was generally well tolerated and maintained mean Hb levels within 1 g/dL from baseline over a long duration of treatment.

DOSE CONVERSION ANALYSES BETWEEN EPOETIN ALFA (EPO) AND DARBEPOETIN ALFA IN HOSPITAL-BASED DIALYSIS CENTERS (HBDCs)

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The dose conversion relationship between EPO and darbepoetin alfa is non-proportional across the dosing spectrum. This analysis evaluates the maintenance dose conversion ratio (DCR) between the two erythropoiesis-stimulating agents (ESAs) for chronic hemodialysis (HD) patients in HBDCs using pre and post conversion data.

This longitudinal analysis is based on retrospective chart review data from HD patients in 23 HBDCs (2004-2005) that underwent conversion from EPO to darbepoetin alfa. Patients were ≥ 18 years, had three-times-weekly (TIW) EPO dosing frequency before conversion, and a once-weekly darbepoetin alfa dosing frequency after conversion. No more than 4 consecutive missing TIW EPO dose was allowed. The mean EPO dose over an 8-week pre-conversion maintenance period (weeks -9 to -2), and mean darbepoetin alfa dose over an 8-week (weeks 21 to 28) post-conversion maintenance period were used in the analysis. A population-level mean maintenance DCR was calculated using two methods: a regression-based method using ordinary least squares regression analysis, and a ratio-based method where the DCR was calculated for each individual patient and then averaged for the study population to give a population-level DCR.

A total of 337 patients were included; 53% male, and 51.9% were 65-89 years of age. Mean (SD) hemoglobin (Hb) levels for the EPO and darbepoetin alfa maintenance periods were comparable, 11.7 (0.9) and 11.6 (0.9) g/dL, respectively. The population-level DCR (95% CI) between EPO and darbepoetin alfa was 320 (298, 344) using the regression-based method, and 350 (319, 381) using the ratio-based method.

The results of this analysis indicate that for a HBDC patient population converting from EPO to darbepoetin alfa and treated to comparable Hb levels, a mean DCR of 320 to 350 can be expected using methods that account for the non-proportional dose relationship.

EVALUATION OF THE MAINTENANCE OF HB CONTROL IN HD PATIENTS BOTH DURING AND AFTER HOSPITALIZATION WITH ONCE-MONTHLY HEMATIDE™

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During hospitalization (hosp), only 3% to 39% of HD pts receive ESA therapy (Brophy, 2007). In HD pts on rHuEPO, Yaqub et al (2001) reported mean Hb decline of 0.7 g/dL 1 mo post hosp, while Solid et al (2007) reported mean Hb decline of ~0.5 g/dL immediately post hosp; however, hosp >15 d were excluded. Hb recovery time from nadir was 2 mo. Solid et al (2004) showed HD pts on rHuEPO required up to 6 mo to regain Hb of 11 g/dL. Notably, some of the pts who were included in these historical retrospective analyses likely received ESA therapy during hosp. Hematide™ is a synthetic, peptidic ESA linked to polyethylene glycol that is in phase 3 development for the treatment of anemia associated with CKD. The purpose of the current analysis was to evaluate Hb decrease post hosp and time to recovery of Hb to prehosp levels in HD pts receiving Hematide.

Unplanned analysis of 2 ongoing open-label, long-term phase 2 extension studies in 100 pts receiving Hematide QM was performed; all hosp events (n=89) were included. Protocols prohibited pts from receiving other ESA therapy during the study, including during hosp.

Mean Hb concentrations were 11.0 g/dL prehosp and 10.4 g/dL post hosp (ie, first assessment after hosp). Mean Hb values 1- and 2-mo post hosp were 11.3 g/dL and 11.4 g/dL, respectively. Time to recover to prehosp levels was ≤1 mo (mean, 24 days). There was no Hb decrease following 38% of hosp. In a total of 100 pts, 7 (7%) had AEs considered possibly related to Hematide: 2 (2%) had diarrhea; other AEs occurred in 1 pt each. The safety profile of Hematide appears consistent with that of other ESAs (Macdougall, 2008).

Compared with published reports of treatment with other ESAs, treatment with Hematide appears to be associated with similar or slightly lower posthospitalization Hb declines and sooner recovery of prehospitalization Hb levels.

A PROSPECTIVE RANDOMIZED TRIAL ON INTERMITTENT POST-DIALYSIS DOSING OF CINACALCET: A SOLUTION TO NON-COMPLIANCE

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The calcimimetic, Cinacalcet provides an option for the medical control of secondary hyperparathyroidism (SHPT) in patients who fail vitamin D or analogue therapy precluding the need for surgery. Though shown to be effective, our experience reflects suboptimal response primarily due to noncompliance.

To enhance compliance, we undertook to evaluate the effectiveness of (thrice weekly) post dialysis dosing of Cinacalcet (group A) as compared to home daily administration (group B). This was a prospective randomized trial of chronic hemodialysis adult patients with refractory SHPT (iPTH >60pmol/l with corrected serum calcium (Sca) >2.1mmol/L, Ca x P04 > 4.4). After 2 week run-in phase, patients were randomly assigned to the two treatment groups. All were maintained on Calcitriol 0.5 mcg IV/3 x/wk titrated along with Cinacalcet to achieve control of SHPT up to a pre-assigned dose maximum of latter (240mg group A and 180mg group B), or development of any adverse effects. iPTH, Sca, phosphorus, alkaline phosphatase were followed for 16 weeks and compared to baseline in both groups. Data was analyzed using between groups linear regression for repeated measures.

Similar demographics and baseline characteristics were observed except for younger age in group A. Cinacalcet was well tolerated with average dose 120 mg (range 30-150) in group A and 62 mg (30- 120) in group B. No significant decline in PTH occurred in group A at 16 wks vs baseline compared to a significant drop in group B [with significant between group difference (p 0.006)]. However Subgroup analysis in group A showed effectiveness of dialysis dosing in patients with less severe SHPT (p 0.04).

Not surprisingly, daily dosing of Cinacalcet is more effective consistent with its pharmacokinetics and hence is optimal for treatment of SHPT. However, dialysis dosing is a viable option in patients with less severe SHPT in whom daily dosing is ineffective due to noncompliance and where parathyroidectomy needs to be avoided.

POST-PARATHYROIDECTOMY (PTX) THYROTOXICOSIS IN A PATIENT WITH ESRD. A CASE FOR CAUTION

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Despite transient hyperthyroidism reportedly occurring in about 30% of post-PTH patients with primary hyperparathyroidism, it has rarely been described in the renal literature. We report a case of a patient on hemodialysis that developed self-limited hyperthyroidism after intra-operative thyroid manipulation and excision during PTX surgery for secondary hyperparathyroidism. The mechanism is thought to be transient thyroid hormone release due to intra-operative manipulation of the thyroid. Case: A 62-year old African-American man with a history of ESRD on hemodialysis therapy and secondary hyperparathyroidism that ultimately failed multiple attempts at medical management and was referred for surgical PTX. Intraoperatively there was difficulty localizing the right superior parathyroid gland despite manipulating and carefully palpating that region of the thyroid gland, the decision was made to perform a right thyroid lobectomy.

A fragment of a parathyroid gland was implanted in the left sternocleidomastoid muscle. Two days post-operatively the patient developed atrial flutter with agitation. Neck examination revealed no signs of swelling, redness or tenderness. An electrocardiogram demonstrated new atrial flutter with a 2:1 block and a transthoracic echocardiography was unremarkable. Thyroid function tests showed elevated levels of free T3 and T4 as well as suppressed TSH. Direct current cardioversion was performed, which reverted the patient to normal sinus. Histology of the thyroid gland revealed benign tissue.

At one month follow up, the patient has biochemical evidence of hypothyroidism. Discussion: The treatment of secondary hyperparathyroidism has evolved in the past decade. Nevertheless, there remains a not-insignificant subset of patients who fail medical management and undergo surgical PTX. There is an extensive literature describing post-PTX disorders of divalent ion homeostasis but paucity on other complications. Transient hyperthyroidism from palpation thyroiditis is a fairly common under-appreciated complication of PTX. It is important that clinicians be aware of this potential complication, so as to not attribute manifestations to post-PTX divalent cation disorders, thereby allowing prompt diagnosis and treatment.

IMPACT OF SEVELAMER ON HOSPITALIZATION AND IN-CENTER DIALYSIS UTILIZATION: A MODELED STUDY BASED ON DCOR

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Over 80% of dialysis patients have hyperphosphatemia (serum phosphorus > 4.5 mg/dl). Treatment of hyperphosphatemia with phosphate binders occurs in most dialysis patients, reducing the risk of mortality, cardiovascular events and hospitalizations. The Dialysis Clinical Outcomes Revisited (DCOR) trial demonstrated that hyperphosphatemia treatment with sevelamer offered a statistically significant reduction in hospitalizations relative to treatment with calcium-based binders (CBBs). The avoidance of hospitalizations and maintenance of care with in-center dialysis sessions offers benefits to payers, health care providers, and patients.

The objective of the study was to use a Markov model to quantify the impact of phosphate binder choice on hospitalization days and the maintenance of in-center dialysis sessions among hyperphosphatemic dialysis patients. The characteristics of the hypothetical cohort represented those observed in DCOR (60 years, 54.4% male, 50.2% diabetic). The risk of mortality, hospitalization and days spent hospitalized for patients treated with sevelamer or CBBs were also derived from DCOR. Based on these risks, in any given month, a patient could die, be hospitalized, or remain in outpatient care.

The use of sevelamer vs. CBBs in a typical dialysis clinic of 75 patients would avoid 119 days of hospitalization within one year of treatment. Assuming a conventional dialysis schedule (3x per week), use of sevelamer vs. CBBs would maintain approximately 51 in-center dialysis sessions. Considering the entire US dialysis population of 367,604 patients, use of sevelamer instead of CBBs would translate into 581,163 hospitalization days avoided per year and 249,070 in-center dialysis sessions maintained per year.

Among dialysis patients, sevelamer, relative to CBBs, reduces the number of days spent hospitalized, allowing for the maintenance of in-center dialysis sessions.

TUMORAL SOFT TISSUE CALCIFICATION IN A DIALYSIS PATIENT

Bucaloiu I D, Ashouian N, Bonebrake S R, Bowen T R, Hartle J E, Norfolk E R. Geisinger Medical Center, Danville, PA

Soft tissue and vascular calcifications are frequently seen in dialysis patients and are related to abnormal bone mineral metabolism. Rarely, tumoral calcinosis can occur in such patients and can lead to pain, disability, neural compression and infection. We present the case of a patient with end stage renal disease with tumoral calcinosis involving the paraspinal tissues.

A 30 year old male nonsmoker, with end stage renal disease due to hypertensive nephrosclerosis on dialysis for five years, presented with a three week history of painful upper thoracic swelling. He denied any history of injury. Physical examination revealed a 2 x 4 cm soft tissue mass located between spinal levels C7 and T3. The mass was tender to palpation, but not affixed to the underlying bone. Range of motion in the upper extremities and neck was preserved, but limited by pain in the shoulders on flexion or abduction. Motor and sensory functions remained intact.

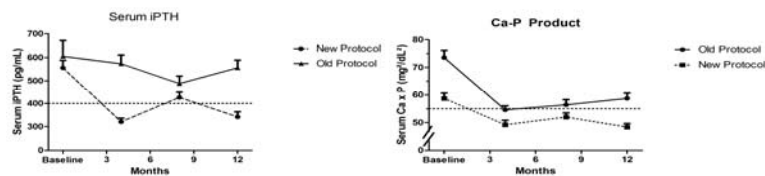
A Computed tomography scan of the area revealed a hyperdense soft tissue mass at C7-T3 level (figure 1). A whole body Bone Scan did not show abnormal uptake. The patient underwent an excisional biopsy of the paraspinal mass. Pathologic examination showed an irregularly shaped, well-circumscribed, 8.6 x 6.5 x 5.8 cm fibromuscular appearing mass. Frozen section examination revealed a fibrotic calcified benign tissue. These findings are consistent with tumoral calcinosis (figure 2). Laboratory evaluation revealed a total calcium level of 9.9 mg/dL, phosphorus level of 8.5 mg/dL and PTH level of 1430 pg/mL.

Ectopic metastatic calcification of the soft tissues has been reported in dialysis patients especially when the calcium/phosphate solubility product of the extracellular fluid is exceeded, or when dystrophic calcifications appear in areas of devitalized tissue. In the absence of a traumatic history we believe the former mechanism is responsible for the ectopic calcification seen in our patient. This case illustrates an extreme complication of disordered bone mineral metabolism in patients with end stage renal disease.

EFFICACY OF INCORPORATING CRITERIA FOR CINACALCET (SENSIPAR) USE INTO A PARICALCITOL (ZEMPLAR) PROTOCOL IN SECONDARY HYPERPARATHYROIDISM

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We recently revised our Paricalcitol (Zemlar) protocol to incorporate cinacalcet (Sensipar) use in hopes of finding more patients achieving target PTH (150-300) and with less elevations in Ca X P product. We recommend initiation of Sensipar when intact parathormone (PTH) levels >400 pg/ml and Calcium X Phosphorus (Ca X P) product >60 despite use of Zemlar at > 5 mcg per treatment. We now report outcomes after changing the protocol. A total of 55 patients were identified from our unit with enough data to evaluate (from a year before and a year after the protocol change). 12 of the 55 patients were initiated on Sensipar as defined by the protocol. In these patients, after changing the protocol mean PTH and Ca X P product fell significantly ($p<0.02$) (see graphs) Transient asymptomatic hypocalcemia occurred in 2 patients who improved with increased Zemlar dosing. Oversuppression occurred in 1 patient (PTH<80) and Sensipar was held. In the entire population, prior to changing the protocol 31/55 (56.4%) patients met goal PTH (<400) and 39/55 (70.9%) had Ca X P product <60. After changing the protocol 41/55 (74.5%) met goal PTH ($p<0.05$) and 39/55 (70.9%) had Ca X P <60. In summary, PTH goals are easier to achieve with protocols involving use of both Vitamin D analogues and calcimimetics. However there is persistent difficulty with elevated Ca X P which is likely related to patient driven diet and binder compliance issues. There may be a need to incorporate calcimimetic use at an even lower Ca X P product limit in an attempt to better achieve KDOQI guidelines. We have now further revised our protocol to recommend Sensipar use when PTH remains >400 (with Zemlar >5 mcg) and Ca x P product exceeds 55.



STRATEGIES TO IMPROVE PHOSPHORUS CONTROL IN PATIENTS WITH CHRONIC SEVERE HYPERPHOSPHATEMIA

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Hyperphosphatemia in the dialysis population remains a significant problem in spite of dietician counseling and phosphorus binding agents. The present study was designed to see if intensive physician directed education would have any additional benefit in lowering phosphorus levels in patients with persistent hyperphosphatemia. Eligible patients were those with a serum phosphorus levels of ≥ 6.5 for ≥ 3 consecutive months. The intervention group met with the study physician monthly for 3 consecutive months with review of food diaries, binder use and compliance, pictorial and written proper diet choices and pictorial and verbal description of potential vascular complications (in addition to the standard dietician visit). The control group met with the dietician monthly with review of labs and diet. Both groups were also seen monthly by their individual nephrologists with ESRD management at their discretion. 12 randomly assigned patients have completed the study, 6 in the intervention group and 6 in the control. Average PO₄ levels in the intervention group were 7.5 ± 0.75 at baseline, fell to 6.65 ± 0.61 during intervention months ($p < 0.05$) and were 6.81 ± 1.3 , NS during the 4 months post intervention. Calcium phosphorus product was 73 ± 9 at baseline, 62 ± 11 ($p = 0.058$) during intervention and 63 ± 15 (NS) 4 months post intervention. In the control group phosphorus levels were 7.14 ± 1.36 at baseline, 6.6 ± 1.27 from months 3-7 and 6.25 ± 1.4 (NS) during the subsequent 4 months. Calcium phosphorus product in the control group was 63 ± 14 , 59 ± 12 and 57 ± 13 at each time point (NS). In summary, intensive regular education with physician involvement can improve phosphorus even in patients with notoriously high levels. However, this was only partially effective, was time consuming and did not persist after intensive education was discontinued. We conclude that regular physician involvement with reinforcement of dietician counseling, review of binder use and education about possible cardiovascular consequences is important. However, ongoing strategies for simpler, more effective and less time consuming tools should be developed.

EFFECT OF ERGOCALCIFEROL TREATMENT ON MINERAL METABOLISM IN CHRONIC HEMODIALYSIS PATIENTS

Junine DeGraf, Derek Larson, Hongyan Du, Stacey Kirshenbaum, Stuart M. Sprague, Neenoo Khosla, Louisa Tammy Ho

Use of activated vitamin D (VDRA) to treat secondary hyperparathyroidism (SHPT) and mineral metabolism (MM) is considered to be an essential management tool in hemodialysis (HD) patients(pts). There is growing interest in achieving sufficient 25-hydroxyvitamin D (25D) levels in the general population. Replacement of 25D is not traditionally performed in the HD pt and there is little information regarding effects of replacement. We looked at the ability of ergocalciferol (Ergo) treatment to correct 25D insufficiency in HD pts and the effect on SHPT and markers of MM.

Pts from a single satellite HD center were screened with baseline 25D. Pts with 25D levels <30 ng/ml, were prescribed 50000 units of Ergo weekly, while patients with levels > 30 ng/ml were given 50000 units mthly. Pts were followed prospectively with baseline and quarterly labs for 6 months (mth). Measured variables included 25D, iPTH, serum alkaline phosphatase (AP), calcium (Ca), phosphorous (P), and total dosing of VDRA. Change from mth 0 to 6 was calculated from difference of values with assigned rank test to assess significance. Spearman's correlation coefficient was used to assess correlation. Statistical tests were 2 sided; $p < 0.05$ was felt significant.

Of 92 HD pts, 72 (78%) had 25D levels <30 at baseline. During the 6 mth follow up, 25D significantly \uparrow from average (avg) of 23.5 ± 11.8 ng/ml to 29.2 ± 14.1 ng/ml, ($p = 0.0003$). The avg iPTH at mth 0 was 411.7 ± 600 pg/ml and did not change significantly over the 6 mths (366 ± 724.5 pg/ml, $p = 0.1994$). No significant changes from 0 to 6 mths, in Ca (9.07 ± 0.83 vs 8.41 ± 2.41 mg/dL), P (4.77 ± 1.17 vs 4.70 ± 1.85 mg/dL), AP (151.6 ± 306.99 vs 136.8 ± 366.4 iu) were noted. However significant positive relationship between Ergo use and serum P was observed.

In this 6 mth prospective single center observational study, of wkly/mthly Ergo \uparrow 25D levels but did not impact markers of bone and MM. Further evaluation of long term administration of Ergo and achievement of higher concentrations of 25D are needed in ESRD patients.

OPTIMUM FREQUENCY OF PARATHYROID HORMONE MONITORING IN CHRONIC DIALYSIS PATIENTS

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Secondary hyperparathyroidism is a common manifestation of chronic kidney disease (CKD). KDOQI 2003 guidelines suggest monitoring parathyroid hormone (PTH) levels every 3 months in hemodialysis (HD) patients. Many dialysis centers routinely monitor PTH levels monthly without any data to support this change. This study was performed to determine the optimum frequency of monitoring PTH in patients on maintenance HD.

A cohort of 150 patients on maintenance HD at a single out patient HD center from February 2008 to March 2009 was included in this study. In phase 1 of the study , PTH was measured every 3 months as per KDOQI 2003 guidelines. In phase 2 , the frequency of monitoring PTH levels was increased from quarterly to monthly. When PTH levels were out of the target range; diet, phosphorus binders, vitamin D analogues and calcimimetics were appropriately adjusted as per standard protocols. The same protocols were used during phase one and phase two of the study period (only the frequency of adjustments changed). The primary endpoint was the percentage of patients achieving PTH level in the KDOQI target range of 150-300pg/ml. Secondary end points were calcium (Ca) and phosphorus (P) levels in target range (8.4-9.5mg/dl and 3.5-5.5mg/dl respectively). Data from the 2 phases was compared with each other and with their respective national averages (NA), using Z test for proportions .

The percentage of patients with PTH in target range increased significantly from phase 1 to phase 2 of the study (25.4% to 40.3%; $p<0.01$). There was also a significant reduction in the percentage of patients with PTH levels > 300 pg/ml in phase 2 compared with national average (37% vs 47%; $p<0.02$). The percentage of patients with Ca and P levels in normal range were similar in phase 1, phase 2 and NA. Our study demonstrates that increasing the frequency of PTH monitoring (and of medication adjustments) from every 3 months to monthly significantly improved the percentage of patients with PTH levels in target range. This improvement in PTH levels was not associated with any significant change in Ca, P, or their product.

BIOEQUIVALENCE OF A LIQUID VS. SOLID FORMULATION OF CALCIUM ACETATE

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The objective was to assess the safety and efficacy of a liquid formulation of calcium acetate (liquid PhosLo).

This study was a randomized, controlled, 3-arm, open label, cross-over study in 46 healthy subjects on liquid PhosLo, PhosLo Gelcaps (calcium acetate), or Ca citrate positive control administered 3 days with meals with 5–10 day washout period on a controlled study diet. At baseline and after 3-day drug exposure, serum Ca, phosphorus (P), glucose, insulin, and 24 hr urines were measured.

The results demonstrated that serum P and Ca from the liquid cohort were comparable to the solid based on 90% CI of ratios for C_{\max} and AUC_{0-6} using ratio adjustment to baseline. For urinary Ca and P, the lower values of the 90% CI for R_{\max} and Ae_{0-6} of the liquid fell below the lower bound, indicating the liquid cohort excreted less Ca and P than the solid. No hyperglycemia was seen, but several subjects experienced mild hypoglycemia. Insulin levels showed troughs between meals and postprandial peaks. There were no SAEs. The most common AE was mild, self-limiting diarrhea.

Liquid PhosLo and PhosLo Gelcaps showed equivalence of serum P and Ca. Less Ca and P were secreted in the urine with the liquid formulation compared to the solid, which highlights the ability of liquid PhosLo to bind P in the GI tract. Liquid PhosLo was well tolerated.

IMPROVED SURVIVAL AMONG PARICALCITOL TREATED HEMODIALYSIS PATIENTS COMPARED WITH NO TREATMENT FOR SECONDARY HYPERPARATHYROIDISM [SHPT] IS INDEPENDENT OF BASELINE iPTH LEVELS

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KDIGO BMD Guidelines suggest a new maintenance range for iPTH. We assessed the survival rate of hemodialysis patients with baseline iPTH between 150-300, 301-600, and greater than 600 pg/mL comparing paricalcitol-treated patients with patients untreated for SHPT.

A historical cohort between 2004 to 2009 was used from a large dialysis provider chain. A total of 51,265 paricalcitol-treated patients were compared with 3,485 patients untreated for SHPT.

Multivariate analyses were adjusted for pertinent variables: Adjusted 1=age, sex, race, diabetes status, and duration of dialysis; Adjusted 2=all variables in 1 + study-entry period; Adjusted 3= all variables in 1&2 + baseline calcium, phosphorus, alkaline phosphatase; and Adjusted 4= all variables in 1, 2 &3 + baseline albumin and hemoglobin.

Paricalcitol vs No VDR Activator	Unadjusted	Adjusted 1	Adjusted 2	Adjusted 3	Adjusted 4
iPTH 150-300					
n=	11,814	11,814	11,814	8,986	8,902
Hazard Ratio	0.483	0.474	0.462	0.481	0.493
95% CI	0.446 - 0.524	0.437 - 0.515	0.425 - 0.502	0.439 - 0.527	0.449 - 0.541
iPTH 301-600					
n=	27,907	27,907	27,907	23,811	23,391
Hazard Ratio	0.267	0.259	0.268	0.313	0.32
95% CI	0.230 - 0.309	0.223 - 0.300	0.231 - 0.310	0.268 - 0.366	0.274 - 0.374
iPTH >600					
n=	15,029	15,027	15,027	12,321	11,971
Hazard Ratio	0.265	0.244	0.27	0.337	0.334
95% CI	0.201 - 0.349	0.185 - 0.321	0.205 - 0.357	0.252 - 0.452	0.249 - 0.447

Use of paricalcitol among hemodialysis patients with SHPT was associated with a significant improvement of survival independent of baseline iPTH levels when compared with patients untreated for SHPT. Survival advantage for iPTH levels between 301-600 and >600 pg/mL were similar. While KDIGO BMD Guidelines are being evaluated for implementation, further studies should be conducted to assess potential implications.

**PARICALCITOL TREATED HEMODIALYSIS PATIENTS
UTILIZE LESS ERYTHROPOIETIN THAN PATIENTS NOT
RECEIVING TREATMENT FOR SECONDARY
HYPERPARATHYROIDISM [SHPT]**

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The implementation of bundling legislation may have an impact on overall medication utilization among dialysis patients. We assessed erythropoietin utilization among hemodialysis patients receiving paricalcitol versus no treatment for SHPT, during their first year of hemodialysis.

A propensity matched cohort study, from a large dialysis provider chain during 2004 to 2009 was conducted to evaluate the erythropoietin utilization among hemodialysis patients receiving paricalcitol compared with no treatment for SHPT. All adult patients who survived the first 90 days of hemodialysis, with no history of kidney transplant, with baseline intact PTH level >149 pg/mL were included in the study. No treatment for SHPT was defined as treatment with no paricalcitol, doxercalciferol, calcitriol, or cinacalcet. Paricalcitol cohort index date was the first dose; the mean time from initiation of dialysis to index date of paricalcitol cohort was applied to the no SHPT treatment cohort. A total of 5,394 matched patients, using propensity scoring for age, gender, race, diabetes status, and baseline hemoglobin level were used in this analysis. A generalized linear regression model was used to compare the mean erythropoietin utilization per patient year, adjusting for age, gender, race, diabetes status, study entry period, and baseline hemoglobin and intact PTH levels.

Erythropoietin utilization during the first year was greater among hemodialysis patients not receiving any treatment for SHPT (1,022,578 units per patient year, [95% CI, 971,197 – 1,073,958]) compared to paricalcitol treated patients (891,872 units per patient year [95% CI, 838,424 – 945,319]), p=0.0005.

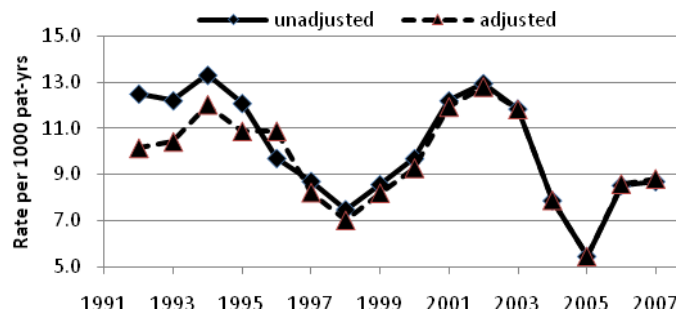
Patients who received paricalcitol while undergoing long-term hemodialysis are associated with less erythropoietin utilization per patient year compared to no treatment for SHPT. Further studies may be needed to confirm these results.

PARATHYROIDECTOMY TRENDS IN US HEMODIALYSIS PATIENTS, 1992-2007

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Parathyroidectomy (PTX) rates rose in US hemodialysis (HD) patients between 1998 and 2002. Since 2002, changes in available bone and mineral metabolism products may have affected PTX rates. The objective of this research was to examine annual trends in PTX rates, particularly from 2002. We used yearly cohorts of 1992-2007 point prevalent Medicare patients on HD on January 1st of the index year. Patients were followed onto the earliest occurrence of: December 31st of the year, PTX or death. Covariates examined included demographic characteristics, 10 comorbidities, IV vitamin D (vit D) use, previous PTX. Cox regression was used to examine baseline associations of PTX. As illustrated below, adjusted PTX rates peaked in 2002, declined steeply through 2005 and then climbed again through 2007 with a rate lower than 2003. Multivariate associations of PTX included younger age, longer vintage, and non-diabetic ESRD.



While contributions of newer vitamin D analogs, non-calcium containing (CCB) phosphorus binders and calcimimetics to the cyclical PTX trends since 1998, are unknown, the upward, then downward trend in PTX rates parallel market introduction and use of new less calcemic vit D analogs, market introduction of non-CCB phosphate binders and calcimimetics. More research is necessary to determine associations with medications and changing PTX rates.

IN-CENTER NOCTURNAL HEMODIALYSIS LEADS TO IMPROVED SERUM PHOSPHORUS (PO₄) LEVELS

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Control of serum phosphorous remains a challenge in the hemodialysis patient despite increased choices in oral phosphate binders, as well as intensive dietary counseling. Elevated serum PO₄ levels contribute to secondary hyperparathyroidism, both directly, and by limiting the use of vitamin D analogs, and are associated with an increased mortality from cardiovascular disease. Delayed transfer from intracellular fluid stores to the extracellular fluid compartment limits the contribution of in-center conventional hemodialysis (ICHD) to phosphate balance. We postulated that in-center nocturnal hemodialysis (NHD), with its longer treatment times, would result in substantially greater PO₄ removal, resulting in a lower serum PO₄. 418 NHD patients were evaluated. We compared parameters of bone and mineral metabolism prior to their conversion to NHD (baseline, mean 4, 5 and 6 months before nocturnal treatment) to these same parameters following the start of NHD (final, mean 7, 8 and 9 months post modality change). The frequency of both ICHD and NHD was 3 sessions per week; the median ICHD session was 4 hrs vs. 7.6 hrs for NHD. Mean serum PO₄ levels decreased 0.67mg/dl during the first month of NHD and, by 9 months, had fallen from a mean (baseline) of 5.79 ± 0.03 to a mean (final) of 5.09 ± 0.03 ($p < 0.001$). Ca \times Phos decreased from a mean (baseline) of 52.91 ± 0.34 to a mean (final) of 46.72 ± 0.31 ($p < 0.001$). Paracalcitol administration increased from a mean (baseline) of 43.1 ± 0.9 μ g/patient/month to a mean (final) of 51.4 ± 1.0 μ g/patient/month ($p < 0.0001$). PTH fell from a mean (baseline) of 472.3 ± 10 pg/ml to 448.9 ± 10 pg/ml ($p = 0.10$), while calcium levels were unchanged. When compared to ICHD, NHD resulted in a lower serum PO₄ consistent with enhanced PO₄ removal as a result of the longer dialysis sessions with NHD. This lower phosphate level may have encouraged more aggressive use of paracalcitol to lower PTH. The decrease in serum PO₄ Ca \times Phos, and PTH may result in long-term cardiovascular benefits for the NHD patient.

DIALYSIS HEALTH OUTCOMES ASSOCIATED WITH PRE-DIALYSIS USE OF PARICALCITOL COMPARED WITH NO VITAMIN D RECEPTOR (VDR) ACTIVATOR TREATMENT

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To research medication use, outpatient services and hospitalizations in the first year of dialysis associated with pre-dialysis paricalcitol use compared to no pre-dialysis VDRA use.

A matched cohort study of 154 dialysis patients comparing health outcomes of pre-dialysis use of paricalcitol compared to no VDR activator treatment. Patients were matched using propensity scoring for age, gender, Charlson co-morbidity Index, and pre-index total costs. Multivariate models adjusted for: age, gender, insurance, physician type, region, pre-index co-morbidities, and pre-index costs were used to evaluate the impact of pre-dialysis paricalcitol treatment on medications, outpatient services, and hospitalizations in first year of dialysis.

Descriptive matched results showed no statistically significant differences in post-dialysis healthcare outcomes. Multivariable analysis demonstrated pre-dialysis paricalcitol use was associated with statistically significant reductions in all-cause outpatient services (0.953, 95%CI: 0.933 – 0.973) and hospitalizations (0.806, 95%CI: 0.684 – 0.950), and CV-related out-patient visits (0.975, 95% CI: 0.951 – 1.000) and hospitalizations (0.816, 95% CI: 0.680 – 0.981) in the first year of dialysis compared with no predialysis VDR activator treatment.

This is the first study to assess predialysis use of paricalcitol on the first year of dialysis outcomes. Paricalcitol treatment of SHPT in predialysis patients is associated with fewer outpatient services and hospitalizations in the first year of dialysis compared to no VDR activator treatment. Further studies are needed to confirm these findings.

AMOUNT OF FLUID INGESTED WITH PHOSPHATE BINDERS (PB) IN HEMODIALYSIS-DEPENDENT CHRONIC KIDNEY DISEASE (HDD-CKD) PATIENTS

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HDD-CKD patients are prescribed many medications. Recent studies have shown that PB account for one-half of the daily pill burden. Because of the necessity of fluid restriction in HDD-CKD patients, this observational descriptive study measured the amount of fluid ingested with PB pills.

Forty-one subjects (16 M, 25 F, age 57.6 ± 11.8 yrs) were enrolled at two dialysis centers. Subjects were provided with a graduated cup along with a diary to record the total number of pills, PB pills, and fluid ingested with PB during each meal and snack for a total of 6 days. Thirty-seven subjects (13 M, 24 F) completed the study (9 PhosLo[®], 24 Renagel[®], 1 Renvela[®], 3 PB combinations). One subject withdrew consent, two were lost to follow up, and one did not complete the diary.

The average total daily pill number was 14.6 ± 7 (range 6 to 33) and 8.4 ± 4.4 for PB (range 1 to 21; PhosLo 5.9 ± 2.5 ; Renagel 8.6 ± 4.3). The average volume of fluid ingested with PB was 292 ± 200 mL (range 46 to 784 mL; PhosLo 190 ± 106 mL, Renagel 327 ± 200 mL), or 29 % of a 1 liter daily fluid restriction. The correlation between daily PB pills and fluid ingested was 0.67, and the average fluid ingested per PB pill was 39.5 ± 29.4 mL (range 6.5 to 172 mL).

These results indicate that optional fluid ingested with PB pills may account for 29% on average (range 4.65% to 78.37%) of the daily fluid restriction which may impair adherence to fluid restriction and contribute to interdialytic weight gain and its consequences.

REACTIVE THYROTOXICOSIS: UNDER DIAGNOSED COMPLICATION OF PARATHYROIDECTOMY IN THE DIALYSIS POPULATION

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Introduction: Parathyroidectomy remains the treatment of choice for patients with ESRD on renal replacement therapy with secondary or tertiary hyperparathyroidism refractory to pharmacotherapy. Although complications peri-operatively are rare and well defined; hyperthyroidism and thyrotoxicosis remains an under diagnosed post operative complication and has rarely been reported especially in the dialysis population.

Case Report: We report a 51 year old female with history of ESRD requiring long term hemodialysis. Patient developed resistant tertiary hyperparathyroidism requiring total parathyroidectomy and re-implantation of the superior parathyroid gland into the sternocleidomastoid muscle without thyroidectomy or thyroid injury. Surgery was successful with PTH dropped from 1637pg/ml to 178pg/ml. Hungry bone syndrome with hypocalcemia developed immediately post-op and was controlled with calcium and vitamin D analogue supplements. Two days post operatively she developed supra ventricular tachycardia with heart rate of 200bpm, and was treated with adenosine. She continued to have sinus tachycardia with heart rate ranging 100-120's associated with palpitations and dizziness. TSH, T3, free T4, and negative thyroid antibodies were consistent with acute reactive thyroiditis. She did not have any previous history of thyroid disease and had never been exposed to iodine, lithium, or thyroid medication previously.

Conclusion: Reactive thyroiditis leading to hyperthyroidism and thyrotoxicosis post parathyroidectomy is not well recognized and under reported. It constitutes for many co-morbidities post operatively. We suggest to routinely screen patients for symptoms and to check TSH pre and post operatively as a standard of care.

PROTON PUMP INHIBITORS AND EFFICACY OF PHOSPHATE BINDERS IN CONTROL OF SERUM PHOSPHORUS LEVELS IN HEMODIALYSIS PATIENTS

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Phosphorus levels play a major role in term of morbidity and mortality in ESRD patients on hemodialysis. Proton pump inhibitors (PPI) can alter the acid level of the stomach and thereby may affect the metabolism and effects of phosphate binders. A previous study has shown the reduced efficacy of calcium carbonate with concurrent use of lansoprazole. However no confirmatory studies are available

In this study we aim to explore the relationship between proton pump inhibitors and two forms of phosphate binders; a newer calcium based binder (calcium acetate –phoslo 667mg/tab) and a non-calcium based binder (sevelamer Hcl 800mg per tab)

We analyzed laboratory results of stable patients from an outpatient hemodialysis unit. 108 patients on stable dosage of phosphate binders over a 3-month period were identified. Patients on concurrent PPI were also noted. Patients were on different PPI. Monthly Phosphorus levels were followed for 3 months. Patients were defined as controlled if the average phosphorus level was less than 6.0 and calcium x phosphate product level less than 55. Pill burden was defined as total numbers of individual phosphate binders taken daily.

Overall there was a much higher number of subjects achieving phosphate control in the combined PPI and phosphate binders than the phosphate binders only group ($p=0.001$). More patients in the sevelamer and PPI group achieve more control of phosphate level and calcium phosphate product than patients on sevelamer group alone ($p=0.037$). There was no significant difference in number of patients achieving phosphate control in the calcium based phosphate binder (phoslo) group with PPI compared to patients without PPI. In patients taking less than 6 tablets, there was also a lower daily pill burden in the combined sevelamer and PPI than sevelamer group alone ($p=0.01$). Though mechanism still not yet known, an interaction between PPI and phosphate binders improve phosphorus control and achieve KDOQI target in hemodialysis patients

GENERALIZED OSTEITIS FIBROSA MIMICKING METASTATIC BONE DISEASE: A CASE REPORT

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INTRODUCTION: Osteitis fibrosa represents a high-turnover bone disease secondary to hyperparathyroidism and has over the years been described as part of the chronic kidney disease - mineral and bone disorders (CKD-MBD) spectrum. We present a case of generalized osteitis fibrosa mimicking metastatic bone disease.

CASE REPORT: A 46 year old African American man with hypertension and end-stage renal disease (ESRD) was admitted with progressively worsening back pain for four weeks and bilateral lower extremity numbness. He had no weight loss, fever or other constitutional symptoms. He was taking metoprolol, lisinopril, sevelamer, cinacalcet and vitamin supplements. He had been on intermittent hem dialysis for six years and had been adherent to therapy.

He had point tenderness over the ribs, thoracic and lumbar spine. Computed tomography (CT) scan of the spine showed multiple lytic lesions involving the thoraco-lumbar spine, sacrum, lower ribs and iliac bones. Head CT scan showed lytic lesions in the left parietal bone. Chest CT scan showed lytic lesions involving multiple ribs, right humeral head and left scapula, and no lesions in the lung. Abdominal CT scan was unremarkable.

Laboratory investigations revealed calcium 7.5mg/dl, phosphorus 5.7mg/dl, alkaline phosphatase 1047 units/liter, parathyroid hormone 2068 pg/ml, total protein 6.5g/dl, albumin 3.6g/dl. Serum and urine protein immuno-electrophoresis were normal. Histologic examination of the lumbar spine biopsy revealed simultaneous osteoclastic and osteoblastic proliferation without evidence of malignancy. These findings were consistent with osteitis fibrosa. He underwent a total parathyroidectomy with subsequent resolution of the symptoms.

CONCLUSION: Generalized osteitis fibrosa could mimic metastatic bone disease and should be considered in the differential diagnoses of lytic bone lesions in ESRD patients.

THE EFFECT OF SEVELAMER CARBONATE AND LANTHANUM CARBONATE ON THE PHARMACOKINETICS OF ORAL CALCITRIOL

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Lanthanum carbonate (LC) and sevelamer carbonate (SC) are used for the treatment of hyperphosphatemia in patients with CKD. It has been suggested that SC may reduce the absorption of fat soluble vitamins. We investigated whether concomitant SC or LC affected the pharmacokinetics of oral calcitriol in healthy volunteers. In this open-label study, 41 volunteers were randomized to 1 of 6 treatment sequences with each sequence consisting of 3 treatment periods: Calcitriol only ($2 \times 0.5 \mu\text{g}$ at lunch); calcitriol + LC ($1 \times 1000 \text{ mg}$ at breakfast, lunch and dinner); calcitriol + SC ($3 \times 800 \text{ mg}$ at breakfast, lunch and dinner). Treatment periods were separated by a 7-day washout and meals were standardized. Serum calcitriol concentrations were assessed by radioimmunoassay at baseline for each study period and at various time points post-dosing (up to 48 h). Exogenous calcitriol levels were calculated as total serum calcitriol minus baseline endogenous levels in each study period. Area under the curve over 48 h (AUC_{0-48}) and maximum exogenous calcitriol concentration (C_{max}) were analyzed using a mixed effect linear model with baseline endogenous calcitriol as a covariate. Mean age of the participants was 30 ± 7.6 years and 54% were men. There were no significant changes in least square mean AUC_{0-48} or C_{max} calcitriol values when LC was co-administered with calcitriol (AUC_{0-48} , calcitriol + LC vs. calcitriol alone: 429 vs. 318 pg.h/ml, $p = 0.171$; C_{max} : 47.0 vs. 49.7 pg/ml, respectively, $p = 0.313$). In contrast, co-administration of SC with calcitriol resulted in a significant reduction in least square mean AUC_{0-48} for calcitriol concentration compared with calcitriol alone (calcitriol + SC: 137 pg.h/ml vs. calcitriol alone: 318 pg.h/ml; $p = 0.024$). Co-administration with SC was associated with a reduction in least square mean C_{max} for calcitriol (calcitriol + SC: 40.1 pg/ml vs. calcitriol alone: 49.7 pg/ml; $p < 0.001$). Our data show that SC reduces the bio-availability of oral calcitriol by about 57%; LC has no significant or clinically relevant effect. This may be an important consideration in patients with CKD who often use oral vitamin D supplementation.

REDUCTION OF DIETARY PHOSPHORUS ABSORPTION WITH LANTHANUM CARBONATE OR SEVELAMER CARBONATE: A BALANCE STUDY

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Lanthanum carbonate (LC) and sevelamer carbonate (SC) are two noncalcium phosphate binders used for the treatment of hyperphosphatemia in patients with chronic kidney disease. This randomized, open-label, crossover study compared the absorption of dietary phosphorus (P) following single doses of LC or SC. Healthy volunteers underwent 4 treatment periods, separated by 7–14 day washouts. Following admission to the study center volunteers were fasted overnight. The gastrointestinal (GI) tract was cleaned by flushing with 3–4 L of mannitol solution (via nasogastric tube) starting 4 hours prior to ingestion of a meal, standardized for P and calcium content, which was consumed within a 30 minute period. Approximately 10 hours later a second 4-hour GI wash was started and rectal effluent collected. Volunteers completed periods with meal alone, meal plus LC (1000 mg of lanthanum) and meal plus SC (2400 mg); the treatment sequence was randomly assigned. All subjects were fasted in the fourth period. The P content of the duplicate meals and rectal effluent were analyzed by inductively coupled plasma-optical emission spectroscopy. Safety and tolerability were assessed. Net P absorption was analyzed using a standard mixed effect linear model in the pharmacodynamic population (n = 18). The least squares (LS) mean \pm SE absorption of P after receiving the meal without a phosphate binder was 281.7 ± 14.1 mg. P absorption after administration of 1000 mg LC was 156.0 ± 14.2 mg and after 2400 mg of SC was 221.8 ± 14.1 mg. There was significantly less absorption of P with LC than with SC ($p < 0.001$): the difference in LS means (95% CI) was -65.8 ($-96.0, -35.5$) mg. A 1000 mg dose of LC bound 135.1 ± 12.3 mg of P while a 2400 mg dose of SC bound only 63.2 ± 12.3 mg, a difference in LS means (95% CI) of 71.9 ($40.0, 103.8$) mg ($p < 0.001$). LC and SC had similar safety and tolerability profiles. A 1000 mg dose of LC bound more than twice as much P as a single 2400 mg dose of SC. These data suggest that LC may be a more effective phosphate binder than SC in clinical practice.

ASSOCIATION OF 25 HYDROXY VITAMIN D WITH HEALTH RELATED QUALITY OF LIFE AND PHYSICAL FUNCTION IN A HEMODIALYSIS POPULATION

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Aim: To describe the Vitamin D stores in a hemodialysis population and to explore 25 hydroxy vitamin D (25 OHD) association with physical performance and health-related quality of life, including cognitive function.

Methods: Subjects provided informed consent and 25 OH vitamin D levels were assessed. Physical assessment included 6 minute walk, handgrip strength and Short Physical Performance Battery (SPPB). Health questionnaires include cognitive screen, activities and instrumental activities of daily living, SF-36 and Geriatric Depression Scale.

Results: Thus far, 16 subjects (mean age 64 ± 16 years; 10 men/ 6 women; 63% Caucasian) had 25 OHD levels of 23.2 ± 7.3 ng/dl. Thirteen subjects (81%) had 25 OHD less than 30 ng/dl. There was no association between physical performance and 25 OHD in this early assessment. Most subjects scored perfectly on the cognitive scale. There was no association between 25 OHD and questions regarding general health in this population in which 58% described their health as fair or poor. There was an association between 25 OHD and physical or emotional problems interfering with normal social activities ($p=.004$).

Conclusions: Our preliminary data confirms that 25 OHD insufficiency is widely prevalent in the ESRD population. While an association between 25 OHD and physical performance was not seen, it could be due to the small sample and we will continue to recruit. There is an interesting association with questions pertaining to physical and emotional problems interfering with normal activities. Further work will be required to explore whether supplementation may improve physical and emotional health-related quality of life.

IRON ABSORPTION WITH HIGHER DOSES OF FERRIC CITRATE IN CONTROLLING SERUM PHOSPHORUS IN ESRD PATIENTS

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Ferric citrate (FC) is being developed as a phosphate binder to manage serum P level in ESRD patients on dialysis. In animal studies with rats and dogs that were administered FC at much greater doses than the proposed maximum human daily dose of approximately 12g/day, iron absorption was documented. Due to the anemia in ESRD patients on dialysis, absorption of iron from their phosphate binders may be beneficial. In a recently completed Phase II trial in 55 ESRD patients on dialysis administered doses of FC ranging from approximately 1 to 11.3 g/day for 4 weeks, changes in TSAT, serum iron, ferritin, and TIBC were assessed. Patients were eligible for enrollment if their serum phosphorus was > 2.5 mg/dL and they had a serum ferritin < 1000mcg/L and TSAT < 50%.

There was a modest increase in serum iron and TSAT with the use of FC over the 4-week treatment period in all patients. 25 of the 55 patients who received IV iron therapy during the 4-week treatment period had a 15% increase in ferritin levels at the end of the study as compared to baseline. Of the patient who did not receive IV iron therapy during the treatment period, there was no significant change in ferritin from baseline. The mean TSAT increased significantly from baseline to the end of the study in both patients who received IV iron and those who did not. These results support the supposition that longer studies examining the potential for iron absorption from using FC as a phosphate binder are warranted.

THE SAFETY AND TOLERABILITY OF HIGHER DOSES OF FERRIC CITRATE (FC) IN CONTROLLING SERUM PHOSPHORUS (P) IN ESRD PATIENTS

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Previous Phase II trials in approximately 350 ESRD patients have shown the ability of FC to control serum P. These trials administered a maximum dose of 6 g/day of FC. The purpose of this Phase II titration study in 55 ESRD patients was to evaluate the efficacy and safety of doses of FC ranging from 1 to 11.3g/day. Patients who were currently taking > 6 tablets/capsules per day of calcium acetate (667mg), lanthanum carbonate (500mg), sevelamer hydrochloride (800mg), or any combination of these agents were eligible for enrollment and immediately switched to FC. The trial consisted of two unique groups, which were based on the starting dose of FC (4.5 g/day or 6 g/day). The target range for serum P was 3.5 to 5.5 mg/dL. The serum P levels were checked weekly to guide titrations.

In the 34 patients in part 1, the mean baseline serum P immediately prior to the switch was 5.8 mg/dL and following four weeks of treatment was 5.4 mg/dL. The mean (SD) change in serum P was -0.44 (1.82) mg/dL. In the 21 patients in part 2, the mean baseline serum P immediately prior to the switch was 6.0 mg/dL and following four weeks of treatment was approximately 5.4 mg/dL. The mean (SD) change in serum P was -0.55 (1.48) mg/dL. The mean bicarbonate (Bi) at baseline in 55 patients was 22.2 mEq/L and following four weeks of treatment was 23.7 mEq/L. The mean (SD) change in Bi was 1.44 (3.04) mEq/L (p-value 0.001). The major adverse event reported was change in stool color. FC was safe and efficacious in controlling serum P in ESRD patients requiring doses > 6.0g/day.

LANTHANUM CARBONATE OFFERS SUSTAINED CONTROL OF SERUM PHOSPHORUS

Michael Smyth, Lynne Poole. Shire Pharmaceuticals, Basingstoke, UK.

Danese *et al.* (CJASN 2008) highlighted the need to sustain reduced phosphorus levels as consistently as possible. Their study showed that failure to maintain phosphorus control during 1 or more quarters in a 12-month period was associated with an increased risk of mortality in incident hemodialysis patients. Using available evidence, we evaluated how well the noncalcium-based phosphate binder, lanthanum carbonate (LC), sustains phosphorus control. In a head-to-head, cross-over study, LC reduced serum phosphorus from 7.5 mg/dL at baseline to 5.7 mg/dL at week 2, and this level was maintained until the end of the study. Sevelamer hydrochloride reduced serum phosphorus from 7.3 mg/dL to 5.8 mg/dL at week 2, but by week 4 this value had increased to 6.0 mg/dL. In a 2-year study involving over 600 patients randomized to LC, change in mean serum phosphorus from baseline (8.1 mg/dL) ranged from -1.6 to -1.7 mg/dL during maintenance treatment. A 3-year study showed that the change from baseline in serum phosphorus in patients who received at least 152 weeks of LC treatment (baseline: 8.0 mg/dL) was -2.2 mg/dL after 1 year. This reduction was maintained and increased to -2.6 mg/dL after 3 years in these patients. In a long-term assessment (n = 93), LC was able to sustain reduced phosphorus levels for up to 6 years (Table 1). These studies demonstrate that LC sustains phosphorus control. Data demonstrating the ability of phosphate binders to maintain reduced phosphorus levels in the long-term are of importance given the findings of Danese *et al.*

Table 1 Change from baseline in mean serum phosphorus levels during treatment with LC for up to 6 years

Month	n	Mean change \pm SD, mg/dL
Baseline	93	7.8 \pm 2.1
12	90	-2.0 \pm 2.4
24	85	-2.1 \pm 2.6
36	48	-2.2 \pm 2.4
48	36	-2.2 \pm 2.9
60	21	-2.4 \pm 2.2
72	5	-2.3 \pm 1.5

USE OF KINETIC MODELING TO ACHIEVE K/DOQI PHOSPHORUS TARGET AND NEUTRAL CALCIUM BALANCE IN HD PATIENTS

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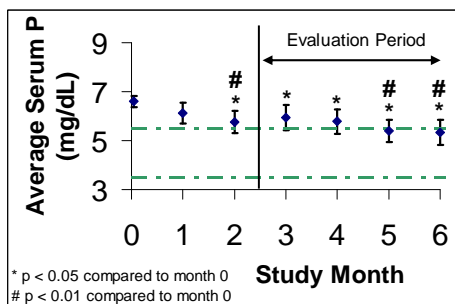
Currently <40% of hemodialysis (HD) patients achieve K/DOQI targets for phosphorus (P) and calcium (Ca). A phosphorus kinetic model (PKM) has been developed to quantify intake and removal of P and Ca. The model calculates doses of P binders, Ca Acetate (Ca Ac), required to meet the target range for serum P as well as dialysate Ca to prevent accumulation or depletion of Ca.

A 6-month, single arm interventional clinical trial has been initiated to determine the efficacy of PKM in 88 HD patients with serum P >5.5 mg/dL consistently during 6-mo baseline. Results for the evaluation period (months 3-6) will be compared to the baseline. Physicians receive monthly reports with a graphical snapshot of relevant mineral metabolism data for the previous 6 mo as well as suggested doses of Ca Ac and dialysate Ca, which are implemented at the physician's discretion.

To date, 36 patients have completed the study. Comparison of the average serum P for the 6-mo baseline vs. the evaluation period showed a statistically significant decrease from 6.3 ± 0.2 (95% CI) to 5.6 ± 0.4 mg/dL ($P=0.002$). By design, 0% of the patients fell within K/DOQI target range for average P during baseline, yet 39% fell within target during the evaluation period. Mean monthly P decreased from 6.6 to 5.3 mg/dL over

the 6 mo of the study (Figure). Mean serum Ca was unchanged, suggesting that increased Ca Ac doses were offset by lower dialysate Ca.

We conclude that PKM is an effective tool in helping patients achieve P targets and neutral Ca balance.



CALCIFIC UREMIC ARTERIOLOPATHY IN A PATIENT ON HEMODIALYSIS: ROLE OF PHARMACOTHERAPY

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Calcific uremic arteriolopathy (CUA), or calciphylaxis, is a systemic calcification syndrome occurring as a complication in some patients with ESRD. Its incidence has increased significantly in the last decade, to an estimated 5% of dialysis-dependent patients in 2008, with mortality ranging from 60% to more than 80%.

We present the case of a 48 year old white female with history of ESRD, on hemodialysis (HD) since 2005, who presented with a 4-5 day history of worsening necrotic lesions over the anterior abdominal wall and thigh. Laboratory findings included serum calcium 9.0 mg/dl, serum phosphorus 6.0 mg/dl, calcium/phosphate product (CPP) of 54.0, serum albumin 2.3 mg/dl, and PTH level of 310 pg/ml. Pathology from her wounds demonstrated medial calcific sclerosis, focal organizing fibrin thrombi in small blood vessels, and calcification in fatty tissue. She was started on daily HD (6 days/week) and sodium thiosulfate (STS) 25 grams IV three times weekly after HD. Use of a low calcium dialysate, low phosphorus diet, and cinacalcet resulted in lower CPP and PTH levels. Despite the above measures, she developed new lesions on her thighs and was eventually made comfort measures only.

CUA results from an imbalance between factors that favor calcification and those that prevent pathologic calcification. Patients who are on HD for longer than 1 year and patients on peritoneal dialysis are considered to be at increased risk. Initial therapy is directed towards wound management, improving nutritional status, correction of secondary hyperparathyroidism, and improving the calcium phosphate balance. The use of more frequent dialysis sessions, high flux HD membranes, or low calcium dialysate alone has not been shown to alter long-term outcomes. There have been several reports on the effectiveness of STS in the treatment of CUA but randomized controlled trials have so far been lacking. Successful use of bisphosphonates, hyperbaric oxygen, and ozonated autohemotherapy in treating CUA has been reported. Further research is necessary to describe the optimal use of various pharmacotherapeutic interventions, dosing strategies, and duration of therapy in treating this disorder.

RAPID REDUCTION OF SERUM PHOSPHORUS LEVELS BY LANTHANUM CARBONATE IN PATIENTS ON

DIALYSIS Rosamund Wilson,¹ Scharmen Confer,² Raymond Pratt.²

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Reducing serum phosphorus (SP) as rapidly as possible may improve patient perception of treatment requirement and avoid the need for additional titration steps. We investigated whether treatment with the non-calcium, non-resin phosphate binder, lanthanum carbonate (LC) resulted in a rapid reduction in SP by examining 4 randomized trials of patients with chronic kidney disease Stage 5 on dialysis (CKD5D).

Two of the studies started dose titration at 750 mg of LC per day and the dose could increase to 3000 mg over the titration period. In the first study (n = 126), significant decreases in mean SP levels were observed after 1 week of treatment ($p < 0.0001$). Comparison of the mean baseline level of 7.8 mg/dL with each of the 6 titration weeks also showed significant reductions ($p < 0.0001$). In the second study (n = 73), mean baseline SP levels were similar to the first study (7.7 mg/dL) and there was a significant reduction in mean SP levels from week 1. In addition, 95% confidence intervals of the means for all visits did not include the mean baseline SP value. In a study with fixed LC doses for 6 weeks, patients randomized to doses > 750 mg experienced significant reductions in SP levels ($p < 0.05$) as early as week 1 of treatment (1350 mg: n = 30, mean baseline SP 6.81 mg/dL; 2250 mg: n = 26, mean baseline SP 7.42 mg/dL). Differences in SP levels between the LC and placebo groups at the end of treatment were -1.70 and -1.88 mg/dL for the 1350 mg and 2250 mg dose groups, respectively. A recently reported cross-over study comparing LC and sevelamer hydrochloride (SH) showed reductions in SP levels with initial LC and SH doses of 2250 mg and 4800 mg, respectively. The reduction was significantly greater in the LC group than in the SH group even at this titration dose after 1 week of treatment (-1.38 vs. -1.01 mg/dL; $p = 0.024$).

LC has demonstrated rapid reductions of SP, even at low initial doses. These rapid reductions of SP may be advantageous to treat phosphate burden in patients with CKD5D.

HOW DO PATIENTS WITH FAILED KIDNEY TRANSPLANTS FARE ONCE BACK ON DIALYSIS? A GULF PRESPECTIVE

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An increasing number of failed transplant patients returning to dialysis (FTRD) have been observed with reported worse survival vs transplant-naïve dialysis (TxN) patients.

This study aimed to assess outcomes of FTRD vs matched TxN controls in a Gulf region multi-center trial of 700 HD patients.

56 FTRD and 52 controls were identified (Table 1). Interestingly, similar mortality was seen, likely due to earlier start and better HD adequacy in FTRD. Younger age, less diabetes and living donor transplantation in majority with 27% graft nephrectomy (Nx) might also confer benefits. Subgroup analysis of Nx patients showed more hospitalizations and prior rejection episodes with lower graft survival. The deaths, however, occurred only in nonNx group and are likely explained by older age, longer duration on HD, more prevalence of diabetes and CAD (Table 2).

FTRD showed similar survival to TxN. Early intensive HD might account for the benefit. Whether Nx confers advantage is unclear because of the small sample size.

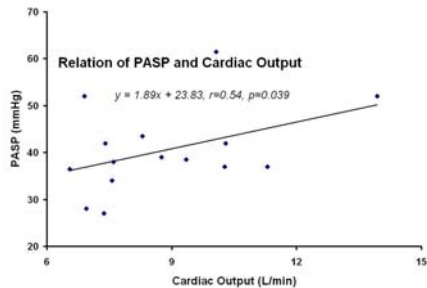
Table 1	FTRD (56)	TxN Controls (52)
Mean Age (yrs)	47.20 ±12.06 M:F 38:17	50.4 ±8.13 M:F 35:17
Causes of ESRD	GN 21: DM 6: HTN 7: Obst2: Familial 3: Unknown 17	GN 2: DM 14: HTN 2: Obst1: Unknown 33
Mean post Tx HD duration	36.63 ±29.30 mos	31.53 ±29.70 mos
KT/V	1.62 ±0.84	1.29 ±0.12
eGFR at HD start (ml/min)	8.4 ±1.6	most had no residual renal function GFR <5
Mortality (%)	3.6	3.8
Table 2	Nx (15)	Non Nx (41)
Mean Age (yrs)	44.92 ±11.8	51.07 ±9.68
# DM: # CAD (%)	1 (6.7): 2 (13)	5 (12): 7 (17)
Mean pre Tx HD duration	21.67± 17.54 mos	36.81 ± 40 mos
Graft survival (mos)	65.80 ±43.63	107.08 ±73.76
# rejections	6(40%)	11(26%)
# admissions: # deaths	5.75 ±2.06: 0	3.86 ±1.68: 2

PREVALENCE OF PULMONARY HYPERTENSION IN PATIENTS WITH HIGH FISTULA FLOW RATES

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Cardiovascular disease is prevalent in hemodialysis pts and is the leading cause of death. Pulm HTN is defined as elevated pulmonary pressures above 25mmHg and has a poor prognosis. We measured the AV fistula flow rate (Qa), cardiac output (CO), CI and pulmonary artery systolic pressure (PASP) for 16 asymptomatic hemodialysis patients with Qa >1 L/min (2.1 ± 0.8 L/min) by echocardiography and the Transonic HD-02 monitor. All pts were male with a mean age of 49.2 ± 4.9 yrs with average access duration of 42.6 ± 35.6 months, BMI 27.4 ± 7.6 kg/m², Hb 11.3 ± 1.1 g/dL, serum Alb 4.1 ± 0.4 g/dL. All pts had CO of 8.6 ± 1.9 L/min, CI of 4.3 ± 0.97 L/min/m², systolic BP of 138 ± 20 mmHg and diastolic BP of 82 ± 14 mmHg. LVH was present in 94% of pts and 87% of our asymptomatic cohort had evidence of pulmonary hypertension (PH) of which 43% were moderately severe >40mmHg. We found a significant correlation between the CO measured by the transonic ultrasound dilution technique and

the PASP ($r=0.54$, $p=0.039$). Our findings suggest a high prevalence of PH in patients with high Qa and CO and that the absence of symptoms does not rule out PH in these patients.



SERUM BETA 2 MICROGLOBULIN LEVELS IN PATIENTS ON CHRONIC HEMODIALYSIS

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Increased levels of serum beta-2 microglobulin are found in patients with kidney disease and are a major constituent of the amyloid fibrils in dialysis related amyloidosis. However, the actual levels have not been correlated to inflammatory markers, increased morbidity or mortality. The aim of this observational study is to evaluate any correlation between levels of serum beta 2 microglobulin with the length of time on hemodialysis, age, BMI along with markers of inflammation, including ferritin and albumin.

All data was collected through chart review of 32 patients dialyzed at VA Medical Center in Jackson, MS from April 2008 till October 2009 and analyzed using standard statistical methods including mean, standard deviation and analysis of variance. All patients were dialysed with a high flux polysulfone dialyzer. Inclusion criteria for the cohort included age >18yrs and chronic hemodialysis treatment for >30 days.

26 patients were African American and 6 were Caucasian. All were male. 12 of 32 patients were chronic diabetics. Mean beta-2 microglobulin was 30.1 mg/l. The mean time on hemodialysis was 40.4 months. Mean BMI was 25.6, mean age was 64.2 years and mean albumin and ferritin were 3.26gm/dl and 491.9ng/ml respectively. On analysis, none of the variables showed significant correlation with beta-2 microglobulin levels.

We felt that this result may be secondary to use of high flux dialyzers, relatively short length of time on dialysis and residual renal function. Also, levels of inflammatory markers may be dependent on other factors and thus not correlate with beta-2 microglobulin.

**REGIONAL CITRATE ANTICOAGULATION FOR
SLOW CONTINUOUS ULTRAFILTRATION (SCUF)
COMPLICATED BY SEVERE METABOLIC ALKOSIS**

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Slow Continuous Ultrafiltration (SCUF) is a safe and efficient treatment for patients with fluid overload who are hemodynamically unstable, have low urine output, and don't need dialysis or hemofiltration (HF) for solute clearance. Anticoagulation needs to be sustained for these long treatments, and this can be clinically challenging; many patients have contraindications to systemic anticoagulation with heparin. Regional Citrate Anticoagulation would be an alternative option; however, we believe this can be problematic due to citrate kinetics. We present two cases in which patients received Anticoagulant Citrate Dextrose Solution A (ACD-A 225 ml/hr) pre-filter and had net UF of 300 ml/hr: severe metabolic alkalosis developed with serum bicarbonate levels rising from 24-25 to 39-41 mmol/l [ABG: PH =7.70, PCO2=29.7, PO2=80.3, HCO3=37.3]. This is in contrast to our CVVH protocol using the same rate of ACD-A, in which there is approximately 2 L/hr of HF and no alkalosis. This emphasizes the importance of accounting for the kinetics of citrate clearance into the HF, thereby avoiding acid base disturbances. Clinicians need to be aware of the risk of metabolic alkalosis when using modalities with low rates of ultra- or HF, and decrease or discontinue the citrate infusions.

VALIDATION OF GDS-15 AS A SCREENING TOOL FOR DEPRESSION IN ELDERLY HEMODIALYSIS PATIENTS

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Depression is common and associated with increased morbidity and mortality in elderly (≥ 65 yrs) hemodialysis (HD) patients. The Beck's Depression inventory (BDI) and the Geriatric Depression scale (GDS) have been used in different cohorts to screen for depression. We aimed to evaluate the 15-item GDS (GDS-15) as such a tool in elderly HD patients and also compare with the BDI, a previously validated tool in younger HD patients.

Both tools were administered to all participants and a geriatric psychiatrist blinded to these results evaluated for depression by the gold standard psychiatric interview. The sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for both tools were assessed against the psychiatric interview.

Sixty two patients completed the study. Patients who were depressed by psychiatric interview had significantly higher GDS-15 and BDI scores compared to those not depressed ($p < 0.01$ both). ROC curves showed high predictive accuracy of the GDS-15 and BDI (area under the curve, 0.808 and 0.729) versus the psychiatric interview. The GDS-15 cutoff with the best diagnostic accuracy was 5, with a sensitivity of 63%, specificity of 82%, PPV of 60%, NPV of 83%. The BDI cutoff with the best diagnostic accuracy was 10, with a sensitivity of 68%, specificity of 77%, PPV of 57%, NPV of 85%.

These results provide evidence that the GDS-15 shows validity in comparison to a gold standard and can be used to screen for depression in the elderly hemodialysis population.

FIVE-YEAR FOLLOW UP TO THE HEMODIALYSIS INFECTION PREVENTION WITH POLYSPORIN OINTMENT (HIPPO) STUDY

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Infection is a common and serious complication in hemodialysis patients accessed via central venous catheters (CVC). This prospective study describes the long-term follow-up of a system wide implementation of topical antibiotic application at the CVC exit site for prophylaxis of CVC related infections. All patients enrolled in the Toronto General Hospital outpatient hemodialysis program dialyzed via a permanent tunneled, cuffed CVC were prospectively monitored for infection type, management and outcomes according to Health Canada standard infections definition.

Since the introduction in 2003 of prophylactic Polysporin Triple Ointment (PTO) application to the exit site during weekly dressing changes, both bacteremia and exit site infection rates have remained below 1.0 per 1000 CVC days. Gram-positive organisms accounted for the majority of exit-site infections and bacteremias and fungal infection rates were not increased.

The long term use of topical antibiotic application at CVC exit sites resulted in a sustained reduction in all CVC related infections with no evidence of antibiotic resistance. A multidisciplinary effort to monitor and track outcomes allowed the safe and effective implementation of a new prophylactic strategy.

REDUCING BEHAVIOR-BASED MISSED HEMODIALYSIS TREATMENTS

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Hemodialysis (HD) patients not receiving their full prescribed treatment or complete treatment schedule have been associated with a higher mortality risk. We examined the missed treatment rate and performed a root cause analysis for missed treatments in 11 North Carolina HD centers. We then provided focused patient education and individualized social work interventions for a period of 12 months to reduce the rate of missed treatments. This education focused on the impact of patient non-adherence on their health and included interventions such as teaching patients relaxation techniques, providing direction for substance abuse treatment, or solving scheduling issues within the clinic. Centers also offered rescheduled appointments when a treatment was missed. Our assessment found that “problems adjusting to their treatment lifestyle” was the top self-reported reason for missed treatments within a patient’s control. Previously diagnosed mental health issues were also common in patients who frequently missed treatments. Of the patients who received a Social Work intervention, missed treatments were reduced or eliminated in 71% of patients. The overall missed treatment reschedule rate doubled from 0.35% of total treatments in the clinics during July 2007 to 0.68% in June 2008. In June 2008, the combined missed treatment rate for non-adherence was 1.77% compared to a baseline rate of 4.22% in July 2007. Social work intervention reduced the rate of missed treatments and improved the reschedule rate. This improved patient adherence, especially in patients deemed “unreachable,” was a key component to improving treatment outcomes and decreasing mortality thus highlighting the valuable role of social workers within the interdisciplinary dialysis team.

VITAMIN D [25(OH)D] AND ITS RELATIONSHIP TO RISK FACTORS FOR CARDIOVASCULAR DISEASE IN MAINTENANCE HEMODIALYSIS PATIENTS

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This study examined the relationship between serum vitamin D [25(OH)D] levels and risk factors for cardiovascular disease (CVD) in maintenance hemodialysis (MHD) patients, specifically diastolic blood pressure (DBP), systolic blood pressure (SBP), pulse pressure (PP), serum total cholesterol (TC), and serum triglycerides (TG), pre- and post-supplementation with ergocalciferol. Data were collected retrospectively via a record review of MHD patients (n=344) at five of the FMC out-patient dialysis centers in western MA for the six month period following introduction of a clinical protocol in April 2006 which includes measuring serum 25(OH)D and supplementing patients with ergocalciferol (50,000IU q/week x 24 weeks) if deficient (<40 ng/mL).

Although no statistically significant relationships were found between risk factors for CVD and serum 25(OH)D at baseline for the group as a whole (21.0 ± 13.5 ng/mL, mean \pm SD) or for patients with 25(OH)D <40 ng/mL (18.4 ± 9.0 ng/mL, n=318), a significant inverse association ($r = -0.159$, $p = 0.047$) was demonstrated for patients with baseline 25(OH)D <30 ng/mL (15.7 ± 6.4 , n = 156) and PP >65 mm Hg (83.0 ± 14.7 mm Hg). For patients achieving improvement in serum 25(OH)D from <30 ng/mL at baseline to >70 ng/mL at follow-up (n=21), a significant inverse association was seen with change in PP ($r = -0.437$, $p = 0.048$) and SBP ($r = -0.438$, $p = 0.047$). Similarly, for patients not on anti-hypertensive medications at baseline (n=45) serum 25(OH)D was inversely associated with mean PP ($r = -0.327$, $p = 0.028$), which decreased (71.4, 65.5, and 51.6 mm Hg) as 25(OH)D tertile increased (≤ 13.6 , 13.7-23.3, and >23.3 ng/mL) ($p = 0.044$).

Improving serum 25(OH)D may aid in lowering PP and SBP in MHD patients who are vitamin D deficient. More research is needed to determine optimal vitamin D levels and supplementation strategies.

HELPING ADULT HEMODIALYSIS PATIENTS SELF-MANAGE ORAL MEDICATIONS: RECOMMENDATIONS FROM THE LITERATURE

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Adult hemodialysis patients take a variety of oral medications to manage their kidney disease and concurrent illnesses, with one recent study reporting that one-half of patients take 19 pills or more per day (almost half of these pills are phosphorous binders).¹ Despite the fact that self managing these medications is a critical component of good clinical outcomes, more than half of hemodialysis patients may not take their medications as prescribed. A literature review was conducted to examine adult hemodialysis patient barriers to oral medication self management. An online search was conducted from March 2009 to May 2009 using MEDLINE, PubMed, Ovid, CINAHL, and PsychLIT databases to identify research and summarize findings from meta-analyses, systematic reviews, clinical reviews, and clinical trials published in English between January 1985 and May 2009, as they relate to oral medication adherence in kidney disease and other chronically ill populations. The results of this literature search suggest that barriers to adult hemodialysis oral medication self-management are multi-faceted, and relate to the burden of taking pills, demographic and socioeconomic variables, psychosocial factors, health literacy, patient satisfaction, and health beliefs. In addition to future research in this area, hemodialysis teams can help patients ameliorate these barriers through interdisciplinary interventions related to self-management training, medication dosing, health literacy, improving communication, and increasing patient self-efficacy.

1. Chiu Y-W, Teitelbaum I, Misra M, de Leon EM, Adzize T, Mehrotra R. Pill burden, adherence, hyperphosphatemia, and quality of life in maintenance dialysis patients. *Clinical Journal Of The American Society Of Nephrology: CJASN*. 2009;4(6):1089-1096.

LIMITED HEALTH LITERACY ASSOCIATED WITH CATHETER USE FOR CHRONIC HEMODIALYSIS

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In the United States 90 million adults have basic or below basic reading skills. Limited health literacy is common and has been associated with poor medication adherence, worse glycemic control, and even mortality. Little is known about the impact of limited health literacy in patients with advanced kidney disease. The objective of this study was to examine the association between health literacy and type of dialysis access used.

We enrolled 50 prevalent chronic hemodialysis patients in a cross-sectional study from June 2009 until October 2009. Patient demographic characteristics were self-reported, and clinical data, including current dialysis access, was abstracted from the medical record. Health literacy was determined with the Rapid Estimate of Adult Literacy in Medicine (REALM) survey.

The mean (SD) age was 51 (15) years, 52% were female, 74% African American, dialysis vintage was 6.1(6.8) years, and 33% were using a catheter for dialysis access. Consistent with previous studies, 32% of the subjects had limited health literacy (<9th grade reading level). Limited health literacy was more common in males (45% vs. 19%; $p=0.04$). Catheter (vs. AVF or graft) use was more common in those with limited compared to adequate health literacy (53% vs. 23%; $p=0.04$). Patients with limited health literacy were almost 5 times more likely to use a catheter for dialysis access compared to those with adequate health literacy even after adjustment for age, gender, race, and years of dialysis (OR (95% CI): 4.8 (1.0 – 24.0); $p=0.05$).

Limited health literacy is common in patients receiving chronic hemodialysis and may identify patients at high risk for not successfully using a fistula or graft for dialysis access. Addressing health literacy may be an important part of improving permanent vascular access utilization, and possibly other clinical outcomes in chronic hemodialysis patients.

IMPROVING THE DIALYSIS EXPERIENCE WITH THE USE OF TECHNOLOGY

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Certain distraction methods have been reported to reduce pain, lower blood pressure, decrease anxiety, and improve overall sense of well-being. In dialysis units, the addition of televisions has provided a welcomed distraction for patients that sit for 3, 4 or more hours each treatment.

In order to expand upon some of these benefits, additional distraction methods were introduced at University of Michigan Dialysis Services. In particular, we made digital music players (Apple's iPod Touch™) and Internet-connected laptop computers available for patients to use during dialysis. Our initial observations show that these technologies offer potential benefits to patients.

Digital music players such as the iPod allow patients to listen to their favorite artist/genre, guided imagery, and relaxation music. Music can be a stimulus for active focus, redirection, or distraction from dialysis. As a result it may have the capacity to reduce pain, lower blood pressure, decrease anxiety and improve overall sense of well-being, all of which may decrease shortened and missed treatments.

Laptop computers provide multiple opportunities for distraction, allowing patients to watch movies, play games, check email, or do personal business. As an additional benefit, laptops in the clinic can help to enhance the technological skills of patients, allowing them to learn to use the Internet, and access renal consumer education and support websites. This can increase self-efficacy and enhance skills that have value outside the clinic.

Our project has revealed both benefits and challenges. Patients have reported several positive impacts, including increased motivation to complete their treatment, improved night time sleep, and improvements in mood. Some patients have shown reluctance to adopt the new technology, possibly due to embarrassment around their limited computer skills. While several staff members are enthusiastic, others have expressed concern about the increased burden on their time and responsibilities. Thus far, the positives have outweighed the negatives and have proved significant enough to sustain this beneficial program.

COST ASSESSMENT OF PATIENTS WITH EXCESSIVE HOSPITAL STAYS IN AN INNER CITY HOSPITAL

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The compound annual growth rate in the number of ESRD dialysis patients has been 3% - 4%. Dialysis revenue is related to payor mix and patient mix. It is impossible to choose payor mix especially in an inner city, safety net hospital system like ours. 25% of patients presenting to our hospital in need of dialysis are uninsured. We rely on hemodialysis units outside our facility for chronic dialysis as we are solely an in patient dialysis facility. This results in hospital stay until completion of insurance application and approval. A cost assessment of this problem has been done below using a typical hypothetical patient who under these circumstances has a stay of 45 days.

Average (avg) cost to community hospitals /patient / day	\$1,468
Total Stay of patient above	45 days
Cost of stay of first 20 days (D) (1,468 x20)	\$29,384
Cost of stay of remaining 25 D (\$734/D, ½ full cost)	\$18,350
Total cost of stay for patient (29,384 +18350)	\$47,734
Avg length of stay of insured dialysis patients at Jacobi	15 days
Potential number of admissions to same bed in 45 days	3 patients (45/15)
Avg payment (Medicare, 2008)/ dialysis patient (JMC)	\$22,292
Opportunity Cost of other dialysis admissions (\$22,292 x 3)	\$66,876
Potential Revenue Lost (due to long admission of patient)	\$22,292
Number of such patients at any given time	7
The potential revenue lost is	\$156,044

Opportunity Cost of diagnosis other than ESRD, may be much greater because the average length-of-stay for such patients is only 6 days. Safety net hospitals bear an inordinate financial burden to provide life sustaining therapy. This is only expected to worsen with the new proposed payment system. Strategies like providing peritoneal dialysis to such patients needs to be emphasized and looked into.

CONCEPTION OF A NATIONAL REGISTRY FOR RENAL REPLACEMENT THERAPY (RRT) IN LEBANON

Salim kabalán, samir mallat, hilal abuzeinab, hafez elzein

Lebanon: a small country on the Mediterranean, area 10452 km² and population 4.5 million. Under 100 practicing Nephrologists care for about 4000 RRT patients: 2500 on dialysis and 1500 with a kidney transplant. A national registry for RRT patients is planned over a 3 year period to drive the process of continuous improvement in quality of patient care and outcomes referenced to the Kidney Disease:

Improving Global Outcomes (K-DIGO) guidelines. Educational and training programs can be developed based on identified areas that require improvement in practices and outcomes, and provide research training opportunities in RRT for medical and graduate students.

A pilot study was kicked-off in March 2007. Data were collected from 18 dialysis centers on 1164 patients. Results were presented to each of the 18 participating centers, as well as to the Society of Nephrology in Lebanon (SNL). The study provided preliminary information, refined data collection methods and database issues and identified variations in logistics, clinical practices and record-keeping. Patients' mean age was 58 years (± 15), over 70% were married, 20% were illiterate and 18% were still working.

The SNL was approached and accepted to become the sponsor of the effort to establish a national registry. A scientific committee consisting of a representative nephrologist from each of the seven medical schools in Lebanon, the SNL and the Ministry of Health was formed to oversee the program. Data will be collected online through web-based application with alternative offline or paper-based entry allowed initially for sites without online capability. Data collection is expected to start by June 2010 for adult patients. Pediatric RRT and transplant components will be incorporated over the following 3 years.

Periodic collection of epidemiological, clinical and laboratory data makes it possible to apply continuous quality improvement measures to patient management practices and outcomes. Opportunities for special research studies, training of personnel and involvement of student trainees are also created. Prevention programs can become more focused, guided by successive observations of outcome measures over time.

PILOT STUDY OF PRACTICES AND OUTCOMES AT HEMODIALYSIS CENTERS IN LEBANON

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Study objective was to examine patient outcomes in the prevalent HD population in Lebanon at the national and center-specific levels. This pilot was intended to set the stage for a national registry.

This was a cross-sectional observational study. All consenting prevalent patients undergoing HD in the participating centers were eligible. Information on demographics, vascular access, dialysis adequacy, anemia, bone mineral metabolism, comorbidity and medications were collected through chart abstraction and patient interviews during a 9-month window starting in September 2007. Data were entered in MS-Excel by a volunteer team of 27 BS / MS level students from the Department of Nutritional Services, American University of Beirut, then read into SPSS version 16. Data validation and cleanup runs with 1% bilateral truncation in highly skewed data preceded analyses at the facility as well as at the national levels.

The study enrolled 18 dialysis centers / 1164 HD patients. Mean age was 58 years (± 15), 56% were males, over 70% were married, 20% were illiterate and 18% were still working. Primary cause of CKD was documented in 72% (diabetes, hypertension, glomerulonephritis or other kidney disease) and unknown in the others. Fistula was used for maintenance dialysis in 84%, average months on dialysis was 53 (± 59), 65% dialyzed three times weekly for 4-hour sessions, but over 40% of patients had a urea reduction ratio $< 65\%$. Mean albumin was 3.6 g/dL (± 0.6), mean hemoglobin was 10.5 g/dL (± 1.6), median ferritin was 511 ng/dL and median transferrin saturation was 26%. Mean calcium and phosphorous were 8.9 (± 0.8) and 5.4 (± 1.8) mg/dL respectively (Ca X P= 48 ± 16) and median PTH was 217.

On average, maintenance HD appears to sustain good outcomes in many aspects of patient care with the exception of hemoglobin. However, there is a wide variation in clinical and laboratory outcomes among centers. Differences in demographic and social construct among HD centers may be partially responsible for this variability, in addition to restrictions of certain medications and diagnostic testing by the type of reimbursement coverage a patient has, which may limit physician's options to provide optimal therapy and follow up to their patients.

HEALTH DISPARITIES/INEQUITIES IN END STAGE RENAL DISEASE

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The purpose of this study is to examine the differences in the rate of increase of End Stage Renal Disease between Whites, African-Americans and Hispanics and the rate of increase in diabetes and hypertension, two of the main causes of ESRD. In the United States, as of December 31, 2007, there were 527,283 people with ESRD. In 2007, 111,000 people started dialysis. There were 57,213 people with ESRD in 1980 (USRDS, 2009).

Methods: Examination of the data compiled by the USRDS in their Annual Data Report for 2007 provides information about the incidence and prevalence of ESRD by race and ethnicity and by primary cause. This information allows one to observe the differences and draw inferences from the data.

Results: The median age for ESRD patients in 2007 was 59.1 years, varying among ethnic groups from a high of 60.3 years for Whites to a low of 57.1 years for African-Americans. The point prevalence rate among African-Americans was 5,111 per million population compared to 1,911 for Asians and 1,231 for non-Hispanic Whites. The point prevalence rate for Hispanics was 2,408 per million population, almost 50% higher than that of non-Hispanic Whites (1,613). In 1980 glomerulonephritis was the leading (42%) cause of ESRD, with diabetes a distant second at 17%. In 2007, diabetes was the primary cause of ESRD. African-Americans begin dialysis at an earlier age and also have the highest rate of diabetes and hypertension, followed by Hispanics. Both groups have a higher rate of diabetes and hypertension than Whites (USRDS, 2009).

Conclusions: Based on the information in the USRDS, one can infer that the rate of ESRD, diabetes and hypertension is greater in minorities than in Whites. One can then conclude that health disparities and inequities exist between Whites and minorities living with ESRD.

ADEQUACY OF INTERMITTENT HEMODIALYSIS (IHD) IN AN INPATIENT SETTING

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Adequacy of IHD in End Stage Renal Disease (ESRD) patients is measured by Kt/v or urea reduction ratio (URR), done monthly in the outpatient hemodialysis unit. Hospitalized ESRD patients and patients with Acute Renal Failure (ARF) get IHD in the hospital setting.

Adequacy of hemodialysis is rarely measured in inpatient settings. We sought to measure hemodialysis adequacy in an inpatient setting using URR values and identify factors associated with variability in their levels. No systematic attempts to increase URR were employed.

URR values were calculated from the pre and post BUN levels obtained from 22 patients receiving IHD in Lenox Hill hospital between 8/09 to 11/09. Data from 2 sessions were used. Our study included ESRD patients who had been hospitalized and ARF patients requiring dialysis.

From the 44 URR values obtained, the mean value \pm SD was 62.47 \pm 9.6. Mean URR value for patients with a catheter was 58.86 \pm 8.3 and for patients without a catheter (fistula or graft) was 69.0 \pm 8.2 (p=0.001). When data for patients with ARF was compared to patients with ESRD, mean URR value was 55.95 \pm 8.2 versus 65.35 \pm 8.9 respectively (p=0.006). Mean URR values when categorized by blood flows of 300, 350 and 400 ml/min were 59.77 \pm 9.5, 61.41 \pm 10.7 and 66.4 \pm 7.7 respectively. The type of K bath and volume of ultrafiltration had a negative correlation with the URR value. (p=0.026, correlation coefficient-0.291 and p=0.054, correlation coefficient-0.210 respectively)

These data on measurements of adequacy in a hospital setting suggest that those patients with catheters had significantly lower clearances than those with fistulas and grafts. In addition patients with ARF had lower values than the chronic ESRD patients. Of note all patients with ARF had catheters for access. No significant association was found between low URR values and type of dialyzer or duration on IHD as routinely prescribed. It would seem that strategies to increase URR in ARF patients with catheters should be emphasized.

TENECTEPLASE FOR THE IMPROVEMENT OF BLOOD FLOW RATE IN DYSFUNCTIONAL HEMODIALYSIS CATHETERS

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To evaluate the safety and efficacy of the thrombolytic agent tenecteplase to improve blood flow rate (BFR) in dysfunctional hemodialysis (HD) catheters, we conducted two phase 3 clinical studies: the randomized, placebo-controlled TROPICS 3 trial and the open-label TROPICS 4 trial. Patients received an initial dose of tenecteplase (2 mg/lumen) or placebo (TROPICS 3 only) for a 1-hr intracatheter dwell. Treatment success was defined as BFR \geq 300 mL/min and a \geq 25 mL/min increase from baseline BFR, without line reversal, 30 min prior to and at the end of HD. For all TROPICS 4 patients and for TROPICS 3 patients enrolled after the final protocol amendment, those without treatment success at the end of the initial visit received another 2 mg instillation of tenecteplase for an extended-dwell period of up to 72 hrs. Adverse events were recorded through two HD sessions following final study drug exposure. A total of 372 patients with dysfunctional catheters were enrolled in the two studies. Of the 297 patients treated with tenecteplase at the initial visit, 31% (95% confidence interval, 26%-36%) achieved treatment success, with a mean (standard deviation) change from baseline BFR of 73 (120) mL/min. Among the 179 patients who received a 1-hr dwell of study drug followed by extended-dwell tenecteplase, 82 (46%) had treatment success at the end of the next HD session. Six catheter-related bloodstream infections and two thromboses were reported in patients following tenecteplase exposure. There were no reports of intracranial hemorrhage, major bleeding, embolic events, or catheter-related complications. In summary, tenecteplase administered as a 1-hr dwell or a 1-hr dwell followed by an extended dwell was associated with improved BFR. The safety profile of intracatheter tenecteplase was favorable, with few adverse events reported.

INCREASED OXIDATIVE SUSCEPTIBILITY OF LOW DENSITY LIPOPROTEIN (LDL) AFTER STANDARD HEMODIALYSIS PROCEDURE

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Purpose: End-stage renal disease is associated with increased oxidative stress, but little is known about the impact of hemodialysis (HD) on oxidative characteristics of plasma lipoproteins. We evaluated the effects of a standard HD procedure (4 hours) on the *ex vivo* copper-induced oxidative susceptibility of LDL and the ability of high density lipoproteins (HDL) to protect LDL from oxidative modification.

Methods: We studied 10 veterans [mean age 57.1 years, duration on HD 5.4 years, all males] undergoing chronic HD treatment. Non-fasting plasma collected immediately pre- and post-HD was fractionated into LDL and high density lipoprotein (HDL) by column chromatography. LDL oxidative susceptibility of HD patients as compared with healthy controls (n=82) was assessed by the rate of formation of conjugated dienes in the presence of copper (Cu^{++}). Autologous HDL was added to isolated LDL in a ratio LDL:HDL 3:1 based on cholesterol contents to assess ability of HDL to protect LDL from *ex vivo* oxidation.

Results: The mean pre-dialysis levels of total cholesterol, triglycerides, LDL, and HDL were 150, 96, 79, and 48 mg/dL, respectively. Pre-dialysis LDL of HD patients was more susceptible to oxidation as compared to LDL from healthy controls (mean lag time 36 versus 58 min). Mean LDL lag time was further reduced by 19.4 % after HD treatment to 29 min ($p=0.015$). Lag time of pre-HD LDL correlated negatively with levels of plasma Lp-PLA₂ activity ($p=0.03$). No correlation was observed with any other laboratory measures. In contrast to studies with HDL from healthy controls, LDL lag time remained reduced in presence of autologous HDL in HD patients.

Conclusions: LDL isolated from HD patients was more susceptible to oxidative modification as compared to LDL from healthy controls. The oxidative susceptibility was further increased following the standard HD procedure. In spite of normal levels, HDL from HD patients did not protect autologous LDL from Cu^{++} -induced oxidation.

INCIDENCE OF VASCULAR ACCESS TYPE AFTER RENAL ALLOGRAFT FAILURE.

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Arteriovenous fistulae (AVF) are widely regarded as the preferred vascular access in hemodialysis patients due to their primary patency and survival benefits. Even though allograft survival has improved based on UNOS, chronic allograft nephropathy continues to be the most prevalent cause of late transplant graft failure. Recent literature has suggested that perhaps initiation of dialysis in chronic kidney disease-transplant (CKD-T) patients may be too late and perhaps clinical practice guidelines should also be established as in CKD. Most of the data available regarding incidence of different vascular types is for failed native kidneys. As renal transplant patients are closely followed by transplant physicians, it would be expected that they have a higher incidence of AVF/AVG compared to catheters. The purpose of this study is to determine the type of vascular access in incident hemodialysis patients after a failed renal transplant.

We performed a retrospective review of all failed renal transplant patients who initiated HD between January 1991 to December 2001 at our institution. Vascular access type at one month and three months from the start of HD was recorded. A total of 104 patients were identified out of which 51.9% were male, 30.8% diabetics, 98% white and 30.8% with re-transplant.

At one month, 47.2% had a central venous catheter compared to 15.3% with AVF and 13.9% with AVG. At three months, 41.4% had a central venous catheter compared to 15.7% with AVF and 18.6% with AVG. 11.4% patients were started on PD. Multinomial logit regression showed females to have a higher likelihood of having a catheter at both one and three months ($p=0.03$ at one month and $p<0.01$ at 3 months). Diabetics also had a higher likelihood of having a catheter at one month ($p=0.04$).

Our study shows that central venous catheters are still the most prevalent access type even in failed renal transplant patients. Our results emphasize the need for earlier recognition and acceptance of a failing renal transplant and need for earlier referral for access placement.

STAPHYLOCOCCUS LUGDUNENSIS MITRAL VALVE ENDOCARDITIS IN A PATIENT ON CHRONIC HEMODIALYSIS

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Staphylococcus species is increasingly recognized as a cause of virulent infections including endocarditis. We describe a case in which coagulase-negative *staphylococcus* in blood was anything but a contaminant.

A 47-year-old African American male presented to the emergency room with complaints of shortness of breath, fever and pleuritic chest pain of one day duration. He had end stage renal disease on chronic hemodialysis, Type II diabetes mellitus and anemia. He was febrile (102.4°F), tachycardic, hypertensive and had bilateral pulmonary crackles and pedal edema. He had been hospitalized 5 weeks prior with *Staphylococcus lugdunensis* bacteremia and an infected 2-week-old arterio-venous graft which had purulent discharge. At that point, the graft and his tunneled catheter were removed and he was treated with a 4 week course of vancomycin. A temporary dialysis catheter was placed. Echocardiogram during the current visit revealed large mitral valve vegetations and severe mitral regurgitation. He underwent a mitral valve replacement and was initiated on intravenous antibiotics. He also developed right brachial artery emboli that required embolectomy. He was subsequently discharged in a stable condition.

S. lugdunensis is a coagulase-negative staphylococcus (CNS). It is unique among CNS because of its propensity for causing aggressive native valve infective endocarditis. A proteinaceous biofilm formation is believed to play a major role in the pathogenesis, especially in the setting of catheters. The mortality rate of *S. lugdunensis* endocarditis rivals that of *Staphylococcus aureus*. *S. lugdunensis* is generally susceptible to beta-lactam agents. If speciation is not performed, these bacteria might be mistaken for *Staphylococcus epidermidis*, a relatively avirulent bacterium that is a common contaminant of cultures. Endocarditis due to this organism is characterized by high mortality, rapid tissue destruction, and a predilection for native valves. Because the clinical outcome is much more favorable with valvular replacement, speciation of the organism assumes great importance in defining the therapeutic approach and resultant outcomes.

HEMOPERFUSION (HP) VS HEMODIALYSIS (HD) IN A CASE OF COMBINED OVERDOSE (OD) WITH ACETAMINOPHEN AND VALPROIC ACID (VPA): IMPLICATIONS OF OUR FINDINGS

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Acetaminophen (N-acetyl-p-aminophenol) or APAP ODs are frequent, as it is the most widely used analgesic/antipyretic drug sold as a single medication or in drug combinations. In the U.S. APAP OD is the major cause of acute liver failure and second leading cause of hepatic failure requiring liver transplantation. Although HD clears APAP, its benefit is questionable in single APAP ODs, where N-acetyl-cysteine (NAC) is the antidote of choice, as it foils the toxic metabolites. VPA ODs have recently increased, in part because of its expanding clinical use. For mild to moderate VPA ODs, supportive care is the sole treatment. Guidelines lack as to whether more aggressive approaches should be used in severe VPA ODs. In severe cases with coma and impending cardiovascular compromise, HD, HP, and continuous renal replacement therapies (CRRT), or combinations thereof, have been used. In severe combined VPA/APAP ODs, extracorporeal therapies may be indicated, since both drugs can be cleared and are metabolized by the liver, but in turn, become hepatotoxic at OD levels. Herein we report such a case. Because of co-ingestion of ibuprofen, four psychiatric medications, and illicit drugs, we offered first charcoal HP, followed by HD, in addition to the usual NAC protocol. Both HP and HD decreased the half life of VPA from > 30 , to 3.03 and 3.60 hours, respectively, and improved the patient's condition. While VPA, a fatty acid ($pK_a \sim 5$), has limited water solubility, the valproate anion (VP^-), its circulating form, is soluble in plasma ($pH > 7$), and displays saturable protein binding. The free VPA fractions ($54.4 \pm 1.1\%$) were identical at initiation of HP and HD. The similar clearances with HP or HD suggest that the equilibrium between bound and free VPA is not rate limiting. Thus extra-corporal modalities such as the molecular adsorbent recirculating system (MARS), HP, or HD with albumin supplemented dialysate, are unlikely to perform any better than HD alone in treating a single, severe VPA OD. However, in severe VPA ODs complicated by other drug ODs, including APAP, a clear hepatotoxin, an initial HP, followed by HD, is a logical strategy.

IMPROVING ADHERENCE IN ADOLESCENTS WITH ESRD: A CASE STUDY

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For teenagers undergoing chronic hemodialysis, the recommended medication routines, dietary restrictions and the stress of undergoing three times weekly hemodialysis treatments in-center may result in significant depression and nonadherence. Until this nonadherence is corrected, the adolescent is at considerable risk for life threatening complications and may not be accepted on the active kidney transplantation waiting list.

A 17 year old male was receiving outpatient hemodialysis at a large university program. His attitudes and behaviors resulted in significant nonadherence with his care and ineligibility for kidney transplant. To improve his compliance, the dialysis team designed a program targeted for teens.

Purpose. The purpose of the research was to improve understanding of how to improve compliance in adolescents undergoing hemodialysis to improve their quality of life and improve transplantation rates.

Method. With an urgent goal of helping him get listed on the kidney transplant list before his approaching 18th birthday, the renal team used a multidisciplinary approach to assess his needs and set treatment goals. The team included the pediatric nephrologist, a mental health professional to treat undiagnosed depression, the renal dietitian, and the nurse practitioner at dialysis. A simple, positive only behavioral reinforcement program was developed using a point system based on compliance with acceptable blood pressure, fluid gains, serum phosphorus levels and monthly medication reviews. Using his interest in hockey, the team developed a notebook and calendar using this theme to map his compliance. He became enthusiastic about earning points and with these points he was able to earn the prize of a hockey souvenir within 3 months and also became eligible for transplant.

Results. His compliance improved significantly and he was listed and received a kidney transplant 7 days before his birthday and was discharged home on his 18th birthday. He continues to do well.

Conclusion. Renal professionals may find the techniques used in this case study may improve adherence in ESRD adolescent patients, improving quality of life and increasing transplantation rates.

EXTRA-ARTICULAR MANIFESTATIONS OF DIALYSIS RELATED AMYLOIDOSIS: A CASE REPORT

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Dialysis related amyloidosis (DRA) is a disorder caused by tissue deposition of β -2 microglobulin amyloid fibrils preferentially in the bone, joint and synovium in end-stage renal disease (ESRD) patients on long-term dialysis. Hence, it commonly manifests as an arthropathy involving the large joints or as carpal tunnel syndrome. Although other organ systems may be involved, they rarely cause clinical manifestation. We report a rare case of cutaneous, muscular and intestinal manifestation of DRA.

A 52-year-old African American male with ESRD secondary to focal segmental glomerulosclerosis (FSGS) has been on hemodialysis for 24 years. He also has a history of a failed deceased-donor kidney transplant, renal osteodystrophy, hepatitis C, and bilateral wrist surgeries for carpal tunnel syndrome as well as debilitating arthropathy of the wrists, shoulders, hips elbows, knees and metacarpophalangeal joints that were attributed to DRA. He presented to our institution with non-healing bilateral buttock ulcers that were previously treated as “pressure ulcers” with multiple sessions of incision and debridement. He also reported persistent diarrhea with negative evaluation for infectious source. On examination, a deep ulcer with discharge was found in the gluteal region. The ulcer beds revealed a firm mass with no tenderness to palpation. A computed tomography scan of the gluteal region revealed diffuse enlargement and heterogeneous appearance of the gluteus maximus muscles bilaterally with ill defined blurring and stranding. Flexible sigmoidoscopy revealed a thickened and nodular appearing rectal wall. The β -2 microglobulin level was high at 25.2 mg/L. Biopsy of both gluteal mass and rectal lesions showed amyloid deposits by Congo red staining. Mass spectrometry confirmed the presence of beta 2 microglobulin-type.

This case illustrates the rare systemic manifestation of DRA in a long term hemodialysis patient and highlights the importance of obtaining tissue samples with special staining of unusual lesions involving the soft tissue and/or gastrointestinal tract.

TIMING OF HEMODIALYSIS VASCULAR ACCESS PLACEMENT DETERMINES ACCESS OUTCOMES

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Predicting outcomes of hemodialysis vascular access (HDVA) is critical. Multivariate models used in many studies have not been perfect in predicting HDVA outcomes, suggesting roles for risk factors other than patient demographics and comorbid conditions.

In this cross sectional study, we investigated the role of timing of HDVA placement on outcomes. Patients with either arteriovenous fistulae (AVF) or grafts (AVG) were included in this study. Medical records of all prevalent hemodialysis (HD) patients at any of the seven University of Virginia hemodialysis facilities were reviewed. Data was collected on patient age, gender, race, comorbid conditions, HDVA type and location, timing of access placement (Pre or Post ESRD), status of each HDVA (in use, maturing, failed) and the number of months used.

A total of 750 HDVA were included (60% AVF and 40% AVG) in this study. Mean (SD) age of patients was 61.3 (13.7). 41% were females, 60% African-American, and 38% Caucasian. Diabetes was present in 51%, cardiovascular disease including heart failure, coronary, peripheral vascular and cerebrovascular disease in 40% and hypertension 62% of all patients. Overall success rate and longevity of all HDVA were 66% and 22.4 months, respectively. Two hundred and twenty four (30%) of all HDVA were placed before initiation of HD. The rate of success for HDVA was significantly higher in the Pre as compared to the Post group (74.6% vs. 62.9%, $p = 0.002$). The number of AVF failure to mature (7.6% vs. 12.0%, $p = 0.01$) were significantly lower in the Pre group. Mean number of month an HDVA was used was 29.4 in the Pre and 19.2 in the Post group ($p < 0.0001$). Hypertension was more common in the post HD group (66.7% vs. 49.1%, $p < 0.0001$). No differences in age, gender, race or comorbid conditions were found between groups.

Placement of vascular access before initiation of hemodialysis is associated with higher success rates and longer survival of HDVA.

EVALUATION OF MEDICATION RECONCILIATION PROCESS IN PREVENTING MEDICATION ERRORS IN ESRD PATIENTS

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ESRD patients are frequently admitted to the hospital, and generally take a large number of medications. Medication reconciliation may be an effective process to reduce medication errors at the time of hospital discharge. Few studies have evaluated the impact of medication reconciliation process in the ESRD population. We aim to evaluate the impact of medication reconciliation process on medication errors in ESRD patients at the time of hospital discharge.

This retrospective observational study was conducted at the Ohio State University. A hospital based medication reconciliation process was implemented on September 1st 2006. Fifty consecutive ESRD patient admissions were reviewed. Half occurred before and half after the implementation date. The numbers of discharges with medication errors were recorded. Medications errors were then divided into errors of omission, duplication or inaccurate dosage.

In the pre-medication reconciliation period, ten (40%) discharge instructions had medication errors. In the post-medication reconciliation period, three (12%) discharge instructions had medication errors. The most common error was an omission. Using the Fisher's Exact Probability test the one-tail p-value was 0.025.

The number of medication errors for ESRD patients at the time of discharge decreased after a formal medication reconciliation process was initiated. Medication reconciliation is an effective tool in reducing medication errors.

ADEQUATE ESTIMATION OF TUMOR MARKERS IN HEMODIALYSIS (HD) PATIENTS

Miho Kando, Nozomi Okada, Daisuke Okita, Emi Kihara, Koichi Sasaki, Yasufumi Kiyota, Chiharu Kawamoto, Yuka Orita, Kazuko Arita, Katsutoshi Maeda, Hiroaki Oda Oda Medical Clinic, Hiroshima, Japan. The purpose of this study is to clarify clinical characteristics affecting the serum levels of tumor markers, consequently to evaluate the levels of the markers adequately in HD patients. One hundred and twelve HD patients (68 males and 44 females) with clinically no malignancies were subjected to this study. Serum CEA, AFP, CA19-9 in males and females as well as those of PSA in males and SCC in females were measured and investigated a relationship to clinical features of age, duration of HD therapy, and primary kidney diseases which caused end stage renal disease (ESRD). Serum levels of CEA, AFP, CA19-9, PSA and SCC in the HD patients were 4.9 ± 0.4 ng/ml, 3.0 ± 0.2 ng/ml, 13.3 ± 1.4 U/ml, 1.2 ± 0.1 ng/ml and 4.5 ± 1.1 ng/ml, respectively (mean \pm SEM). The mean CEA level in HD patients were within the normal range (≤ 5.0 ng/ml), however, 33% of HD patients showed higher CEA levels than the normal range. No HD patient showed a higher AFP level than the normal range (≤ 10 ng/ml) and a positive correlation between AFP levels and HD duration was found suggesting an accumulation of AFP molecule over time. Patients with chronic glomerulonephritis (CGN) patients (3.5 ± 1.7 ng/ml) showed higher AFP levels than those with diabetic nephropathy (DN) patients (2.5 ± 1.6 ng/ml). CA19-9 levels in 96.4% of HD patients were within the normal range (≤ 37 U/ml) and those in DN patients (16.0 ± 2.3 U/ml) showed twice as high as those in CGN patients (8.2 ± 1.2 U/ml). Within the normal range (≤ 4.0 ng/ml), mean PSA levels significantly elevated according to aging by every decade. A positive correlation between serum SCC and creatinine levels indicated an impaired clearance of SCC molecule may elevate serum SCC levels. In summary, normal ranges of AFP and CA19-9 are applicable to HD patients, although one-third of HD patients show higher CEA levels than the normal range. Relevant consideration of HD duration and primary diseases of ESRD for AFP, while primary diseases of ESRD for CA19-9, ages for PSA and serum creatinine levels for SCC may be indispensable for adequate estimation of these markers.

ANALYSIS OF HEMOGLOBIN STABILITY WITH HEMATIDE™ IN HEMODIALYSIS PATIENTS

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Hb variability has been linked to increased mortality in HD pts (Yang, 2007). Methods used to characterize variability include (1) standard deviation (SD): for each pt, monthly Hb used to calculate mean Hb; distance of each monthly Hb from mean used to determine SD; each pt's SDs then averaged across population; (2) residual SD (RSD): regression line fit to each pt's monthly Hb values, with the SD calculated around that line; (3) Hb cycling: cycle=2 consecutive excursions in different directions; excursion=decreasing or increasing monthly Hb levels by ≥ 1.5 g/dL (Arneson, 2009). Using monthly Hb values over 6 mo, Arneson (2009) reported on Hb variability in prevalent HD pts receiving an ESA: mean of the SD was 0.96, mean of the RSD was 0.75, and Hb cycling occurred in 37.8% of pts. Fishbane (2005) reported Hb cycling in >90% of pts in this study of 281 HD pts over 12 mo with Hb values collected every 2 wks, but excluded pts with hosp >10 d. Hematide is a synthetic, peptidic ESA linked to polyethylene glycol that is in phase 3 development for the treatment of anemia associated with CKD. The purpose of the current analysis is to characterize Hb stability/variability with Hematide in HD pts.

This unplanned analysis of effectiveness included a subset of 61 HD pts receiving Hematide QM (Q4W) for 12 mo in an ongoing, long-term phase 2 extension study (N=81). Hb variability was evaluated using the 3 methods described above. Pts hospitalized >10 d were not excluded.

Mean of the SD was 0.68. Mean of the RSD was 0.40. Hb cycling occurred in 21.3% of pts. Of the total 81 pts in the study, 7 (8.6%) had AEs possibly related to Hematide. Two had diarrhea; other AEs occurred in 1 pt each. The safety profile of Hematide appears consistent with that of other ESAs (Macdougall, 2008).

Initial results for stability of Hb in HD pts on Hematide for 12 months appear to compare favorably with those reported for other ESAs. Controlled studies that compare Hematide against other ESAs are ongoing.

MANAGING WORKLOAD ON DIALYSIS

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To determine the quality of dialysis care it is essential to have the adequate database.

Fresenius Medical Care with a clinical database called Euclid collects medical patient data, fully assuring confidentiality. Each participating dialysis center receives benchmarking reports at the regular interval. On October 2009 the database includes 32,685 patients with 425,098 chronic renal replacement treatments from 430 dialysis centers. Since the year 2007 all Nefrodial centers in Slovenia have been entered into the database. This is the opportunity to register the magnitude of the work to a complete database performed by medical doctors.

During the period of one year (from December 2008 until December 2009) we analyzed the number of changes in the dialysis protocols. These were made by medical doctors in stabile patients in our dialysis center in order to improve chronic renal replacement therapy.

We analyzed data for 47 patients, age 65,6 \pm 13,2 years, females 41,2%, diabetics 23,1%, time on dialysis 7,01 \pm 5,93, catheter 8,4%, with neoplasm 14,5%. We recorded 1168 changes in dialysis protocols, in total, 597 in men and 571 in women, during the observational period. The average number of changes made in dialysis protocols per patient was 25,3 \pm 5, 2 SD.

Together with the strict dialysis protocols and following the clinical practice guidelines, we need to make changes in about 16,7 percent of patients, on every dialysis session. To do so, the permanent engagement of medical doctors is needed. To be effective, a database must be integrated into operations of the dialysis center as a continuous activity, involving all staff members without the working burden of medical doctors.

INTRADIALYTIC USE OF HEPARIN: IS HEPARIN NECESSARY FOR STABLE CHRONIC HEMODIALYSIS TREATMENTS?

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It is common practice to use heparin during hemodialysis (HD) to prevent clotting. However, the use of heparin is associated with several adverse effects including bleeding, heparin induced thrombocytopenia and its consequences and bone loss (with long term use). The goal of the study was to determine if heparin use influences dialyzer clotting, dialysis clearance and hemoglobin level. Charts of 18 chronic stable HD patients who received at least 20 HD on the same admission, from June 2007 to November 2009 were reviewed. All patients received 3.5 hours of HD, three times a week, with an average blood flow of 400 ml/min using Fresenius polysulfone dialyzers. Patients were categorized into two groups based on the use of intradialytic heparin. Statistical methods used include chi-square test, paired and unpaired t-tests. P values < 0.05 are considered to be significant. Twelve of the 18 patients were males. Average age of patients in the heparin group was 45 years and 52.6 years in the heparin free group.

	Heparin group	Heparin Free group	Comments
Number of Patients	6	12	
Total number of HD	270	569	
Number of dialyzer clotting	3 (1.1%)	8(1.4%)	P=0.76
Hemoglobin	10.8±0.77	10.03±0.77	P=0.06
Urea reduction ratio	66.5±4.24	65±5.9	P=0.48

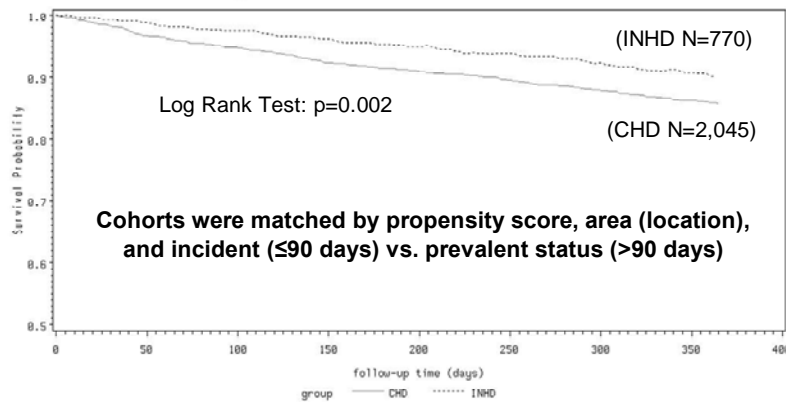
The risk of dialyzer clotting as well as average hemoglobin and dialysis adequacy were not statistically significant between groups. The risk of dialyzer clotting reported in our study is lower than previous studies. Intradialytic heparin use should be individualized and used only in patients with a tendency of higher clotting.

COMPARISON OF MORTALITY RISK BETWEEN INCENTER NOCTURNAL AND CONVENTIONAL HEMODIALYSIS

Eduardo Lacson, Jr.,¹ Jianglin Xu,¹ Keith Lester,¹ Norma Ofsthun,¹ Robert Lindsay,² Rita Suri,² Gihad Nesrallah,² Michael Lazarus,¹ Raymond Hakim, MD.¹

¹Fresenius Medical Care, North America, Waltham, Massachusetts, USA; and ²University of Western Ontario, London, Canada

We identified 770 patients from 77 Fresenius Medical Care, North America facilities treated by In-center Nocturnal Hemodialysis (INHD) 3x/week, for the first time between 1/1/06 - 12/31/07, who matched ~1:3 with conventional hemodialysis (CHD) patients by propensity score (using age, gender, race, diabetes, vintage, body mass index, vascular access, albumin, hemoglobin, phosphorus, calcium, & white cell count), area (location) and incident/prevalent status (≤ 90 days vs. >90 days). Mortality was tracked for up to 1 year. Resulting matched CHD/INHD cohorts were ~8 years younger than average, two-thirds male, half African American, and half with diabetes – not typical of the US HD population. Comparative 1-year survival curves are shown:



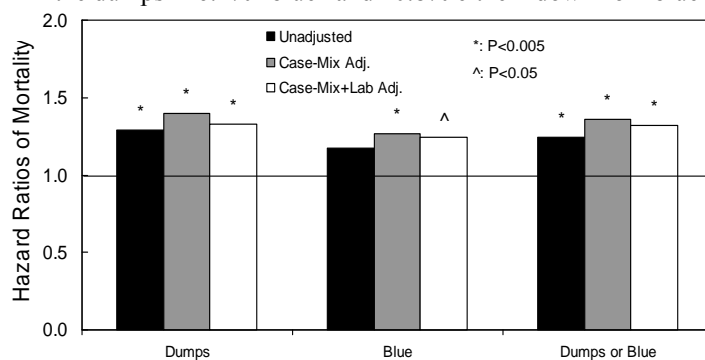
Although we could not eliminate residual confounding, INHD patients exhibited a survival advantage over matched CHD patients with hazard risk ratio of 0.66 ($p=0.002$). INHD is a novel viable option for dialysis therapy. How outcomes compare between INHD and non-conventional (e.g. more frequent) dialysis regimens remains to be elucidated.

DEPRESSION IS ASSOCIATED WITH HIGHER MORTALITY RISK IN INCIDENT HEMODIALYSIS PATIENTS

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We studied 6,415 incident (vintage \leq 90 days) HD patients treated in Fresenius Medical Care North America facilities with scorable SF-36 surveys obtained between 1/1/06 to 12/31/06. Scores <3 (“more than a good bit of the time”) for SF-36 questions #25 (“down in the dumps”) and #28 (downhearted and blue”) were used as indicators of depression. Mortality was tracked for 1-year from the date of survey. Cox models were constructed, including adjustment for case-mix (age, gender, race, diabetes), and case-mix+lab (calcium, albumin, creatinine, phosphorus, hemoglobin, transferrin saturation). Mean cohort age was 62.3 ± 15.2 , 45% female, 69% white, 58% diabetic with 14.1% classified as “down in the dumps” 16.1% “blue” and 20.8% either “down” or “blue”.



Hazard ratios (HR) were 1.24, 1.36 and 1.32 for unadjusted, case-mix adjusted and case-mix+lab adjusted markers of depression (all $p<0.005$). In the subset of 284 patients who withdrew from dialysis, corresponding HR were 1.37, 1.56 and 1.62, respectively (all $p<0.05$).

Depression were associated with ~30% greater risk of death for incident HD patients (similar to prevalent patients), with an even greater risk for withdrawal from dialysis. Whether or not treatment of depression decreases death risk or defers withdrawal from dialysis remains to be elucidated.

INCENTER NOCTURNAL HEMODIALYSIS CONVERSION- ASSOCIATED CHANGES IN LABORATORY MARKERS

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Robert Lindsay,² Rita Suri,² Gihad Nesrallah,² Michael Lazarus,¹
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We reviewed lab results of 556 patients from 77 Fresenius Medical Care, North America outpatient dialysis units on prior conventional hemodialysis (CHD) for <6 hours/treatment for >90 days, then converted for the first time between 1/1/06 – 12/31/07 to 3x/week In-center Nocturnal Hemodialysis (INHD) for >90 days. Mean laboratory results for albumin, hemoglobin, standard weekly Kt/V, phosphorus, calcium, white blood cell count, and transferrin saturation for the prior 90 days on CHD were each compared to that of the 1st 90 days and up to 180 days after conversion to INHD.

The cohort's mean age was 52±13 years, 68% male, 54% African American, 51% diabetic, and vintage was 3.4±3.8 years. Results shown are for ~86% of patients contributing data in all three periods:

Lab Markers	CHD Prior 90 days	INHD 1 st 90 Days	INHD 91-180 days
Albumin (g/dL)	3.89±0.38	3.94±0.37*	3.94±0.36*
Hemoglobin (g/dL)	11.75±1.24	12.04±1.20*	12.13±1.13*
Transferrin Sat. (%)	25.45±8.92	25.30±9.04	25.25±8.59
Std. Weekly Kt/V	2.32±0.28	2.89±0.24*	2.89±0.24*
Phosphorus (mg/dL)	5.85±1.48	5.00±1.28*	5.12±1.44*
Calcium (mg/dL)	9.06±0.76	9.24±0.70*	9.25±0.68*
WBC Count (x10 ³)	7.33±2.23	7.08±2.18**	7.08±2.08**
* p<0.0001; ** p<0.01 (all compared to the CHD period)			

Sustained statistically significant increases in albumin, hemoglobin, dialysis dose, and calcium with declines in phosphorus and WBC count were observed upon conversion to INHD during the 1st 6 months. The direction of changes observed was all favorable (with the possible exception of calcium), although their clinical significance remains to be elucidated.

EVALUATING DEFICIENCIES IN PROCESSES OF CARE IN INCIDENT FISTULAS IN A VETERANS ADMINISTRATION HEALTHCARE SYSTEM

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The 2009 USRDS has reported a very low AVF incident rate of 15%. The K/DOQI Clinical Practice Guideline 1 for vascular access targets a greater than 50% incident AVF rate. The purpose of this study was to determine the proportion patients meeting GFR benchmarks in the necessary steps in pre-dialysis vascular access care.

We performed a retrospective study at the Cincinnati Veterans Administration (VA), using data extraction, based on outpatient creatinines, from a VA database of identified patients with a GFR ≤ 30 from 2006 to 2007 seen in our weekly CKD clinics and performed a 2-year follow-up. We identified 366 patients who met the above criteria. 99% were male, 74% white, 60% diabetics, and 37% had peripheral vascular disease.

84% (n=307) had an initial nephrology consult from a primary care physician. Median GFR in these patients at the time of initial nephrology clinic referral was 30 ± 13 . 31.4% (n= 115) of patients had referral for pre-operative access mapping during the follow-up period. Mean GFR at the time of vein mapping referral was 15 ± 4.8 with only 2.5% of patients having a GFR ≥ 25 at the time of vein mapping referral. 24.3% (n=89) had referral to a surgeon for vascular access surgery, and 55% (n=53) of these referred patients had documented permanent access placement. Mean GFR at the time of vascular access placement was 12.9 ± 4.5 . Only 10% of the patients with vascular access placement had GFR ≥ 20 at the time of vascular access placement. Among the initial 366 patients, 23% (n=84) initiated hemodialysis during the follow-up period. The patient distribution of access at the initiation of dialysis was 26% AVFs, 1% AVGs, and 73% catheters.

Even in a VA health care system, we see delayed referral for pre-operative vascular mapping and surgical evaluation, placement of AVF at low GFRs, and low rates of AVFs at dialysis initiation.

EFFECT OF HEMODIALYSIS (HD) ON BODY COMPOSITION IN ESRD PATIENTS (PTS)

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Bioimpedance analysis (BIA) is a simple method for assessing body composition and nutritional status, utilized in dialysis pts by our group and others. We have previously correlated several BIA-derived parameters with outcomes, including survival, in this population. In this study, we investigated the effect of a single hemodialysis on body composition in HD patients. Sixteen HD pts were enrolled in this study. Demographic, clinical and biochemical data were recorded, and body composition parameters were determined by BIA before and after a routine dialysis. The mean age of the pts was 64 ± 17 (SD) years. Pre and post dialysis body composition data are shown in the Table:

Variable	Predialysis	Postdialysis	P
Weight (LBS)	166 \pm 36	160 \pm 34	<0.0001
BMI(Lbs/inch ²)	27.6 \pm 6.6	26.8 \pm 6.2	<0.0001
Phase angle (Degrees)	5.07 \pm 1.2	5.0 \pm 1.1	0.87
ICW (Liter)	21 \pm 5.1	19 \pm 5.0	0.006
ECW (Liter)	18.8 \pm 3.1	17.3 \pm 2.8	0.003
TBW (Liter)	39.5 \pm 7.5	36.8 \pm 6.9	<0.0001

Not surprisingly, body weight, body mass index (BMI), intracellular water (ICW), extracellular water (ECW), and total body water (TBW) were significantly lower post-dialysis. However, the ECW/ICW ratio did not change ($p=0.68$ by paired t-test). The change in body weight correlated with the change in TBW ($r=0.57, p=0.02$) and in ECW ($r=0.49, p=0.05$). Pre ($r=0.59, p=0.016$) and post dialysis ICW ($r=0.7, p=0.003$) and the post-dialysis change in ECW ($r=0.59, p=0.016$) were directly correlated with serum albumin, consistent with expected hemoconcentration. Interestingly, the ECW/ICW ratio (a marker of relative lean body mass) and the phase angle (marker of “cellular health”) were unchanged after hemodialysis. We have previously reported that these two markers were highly correlated with survival, and these results further validate their utility as independent outcome predictors in this population.

LONG-TERM USE OF FERRIC CITRATE IN ESRD PATIENTS

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After completion of a Phase II multi-national (US and Taiwan), multi-center, randomized, double-blind, placebo-controlled, dose-ranging study in approximately 135 patients treated with a fixed-dose of placebo, 2, 4, or 6g/day of ferric citrate (FC) for 28 days to control serum phosphorus (P) in ESRD patients undergoing dialysis; 29 patients from the site in Taiwan were admitted into a one-year, open-label extension (OLE) trial to assess the long-term efficacy and safety of FC in controlling serum P for up to one year. The patients in the OLE trial were titrated with doses ranging from 2 to 6 g/day of FC throughout the one-year treatment period to maintain serum P between 3.5 and 5.5 mg/dL. The average duration of the patients' participation in the OLE trial was 306±85 days. This OLE trial was the first trial to examine the long-term use of FC as a phosphate binder in ESRD patients on dialysis. The mean serum P level throughout the trial was 5.22±0.18 mg/dL, and the mean CaxP product was 49.06±2.15 mg²/dL². There were approximately 8 patients that had intermittent IV iron and/EPO holidays throughout the 1-year treatment period. There were no ferric citrate-related serious adverse events in the OLE trial. The most common adverse event in the OLE was stool color change and this adverse event was expected. FC was well tolerated in the trial. The results of this trial indicate that FC has the potential to be an effective and safe long-term phosphate binder that controls serum P in ESRD patients on dialysis.

CITRATE (ACD) VS HEPARIN LOCKS: COMPARING CATHETER OUTCOMES FOR SHORT TERM INPATIENT HEMODIALYSIS

Ruba Nijmeh, Samir Parikh, Udayan Bhatt, Anil Agarwal., The Ohio State University, Columbus, OH.

Heparin has traditionally been the agent of choice to lock hemodialysis (HD) catheters (CVC) to maintain patency, but has known disadvantages. ACD is an alternative anticoagulant and its use is becoming more prevalent. There is no data comparing the two agents in patients undergoing HD in the inpatient setting. It is a quality assurance controlled study to assess differences in the efficacy of ACD vs. heparin lock in both non tunneled (NTDCs) and tunneled dialysis catheters (TDCs) in the inpatient setting.

We prospectively collected data for ACD and heparin locks in newly inserted NTDCs and TDCs, between 08/09 and 11/09 at two sites within The Ohio State University system. ACD lock was used in all CVC at site 1, while heparin lock was used in all CVC at site 2. Outcomes of CVC dysfunction and CVC infection were recorded.

We evaluated 120 CVC and 84 met inclusion criteria with 691 catheter days and 333 treatments (tx). There were 50 CVC in ACD group (gp), and 35 CVC in heparin gp. The event rate for the NTDCs was 0.048 per tx in ACD gp and 0.128 per tx in heparin gp ($p=0.05$). The event rate for the TDCs was 0.023 per tx in the ACD gp and 0.05 per tx in the heparin gp ($p=0.17$). For combined TDCs and NTDCs, the event rate was 0.043 per tx in ACD gp and 0.079 per tx in the heparin gp ($p=0.47$).

	Cath Days	TX #	Infxn	Dysfxn	Event rate
ACD NTDC(40)	300	165	1	7	0.048
Hep NTDC(15)	108	47	0	6	0.128
ACD TDC(10)	83	42	0	1	0.023
Hep TDC(20)	200	79	0	4	0.050

The preliminary data from this prospective, controlled study suggests that there is no significant difference in CVC outcomes between heparin and ACD as catheter lock for TDC; however, there appear to be better outcomes with ACD in NTDCs in inpatient setup for short term HD. Data collection is ongoing.

IMPROVING MEDICATION ACCESS TO REDUCE DISPARITIES BY A LARGE DIALYSIS PROVIDER

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¹DaVita Inc., Lakewood, CO, United States

There are clear disparities in access and delivery of care to racial and ethnic minorities with CKD (Norris K, Nissenson AR: *CJASN* 2008) which may also extend to those with ESRD. Achieving access to medications is a challenge for many ESRD patients who on average are taking 7-9 different medications each day. We developed an innovative program, DaVita Rx which delivers prescribed medications to dialysis patient at the dialysis center. We postulated that use of DaVita Rx would improve medication adherence, particularly for subsets of patients for whom access to medications was suboptimal. We hypothesized that access to and use of medications may be a significant area of disparity that impacts clinical outcomes. This study analyzed serum phosphorus for 8,120 DaVita patients across 373 centers. A subgroup analysis was performed on 1094 patients to analyze phosphorus results by race and ethnicity. Mean serum phosphorus levels were analyzed over 2 time periods: 9-months prior to enrollment in DaVita Rx and 1 year post-enrollment. A mixed-model analysis was performed so that all available data would be used for the estimates of mean values at each quarterly time point. The mean values for phosphorus were calculated as least squares estimates to allow for the inclusion of all patients. As of January 2009, Black and Hispanic populations were over represented in the DaVita Rx population, with 72% of those using DaVita Rx in one of these groups, compared to only half of all DaVita patients in these groups. After 1 year of using DaVita Rx, patients were more likely to move from suboptimal (>5.5 mg/dl) to optimal (< 5.5 mg/dl) serum phosphorus levels compared to non-DaVita Rx users (6% vs. 3%) and the effect was even more pronounced in Blacks (8% DaVita Rx vs. 2% non-DaVita Rx). These results indicate that DaVita Rx is used disproportionately more by Black and Hispanic populations suggesting an unmet need in these groups. Significant improvements in phosphorus control, particularly in Black patients using DaVita Rx, illustrates that timely access to needed medications can improve clinical outcomes in this group, and thus fills an important gap in care.

DIALYSIS RELATED TRANSIENT METHEMOGLOBINEMIA- A CASE OF TOPICAL PRILOCAINE INDUCED SYSTEMIC TOXICITY

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Flushing and Trude Weishaupt dialysis Satellite Unit, Flushing, NY

Symptoms attributable to methemoglobinemia in dialysis patients are now rare especially in the absence of dialysis water contamination.

Patients on hemodialysis, to minimize the pain discomfort of access cannulation, often use topical anesthetics. In high dosage administered Prilocaine can result in toxicity with features of methemoglobinemia.

We present the case of a 78 yr old female with ESRD due to HTN with no residual renal function who have been previously stable on hemodialysis for 2yrs. Patient developed a new intra-dialytic hypotension, sensation of alteration in body temperature, peri-oral numbness and bluish discoloration of the lips and hands at the end of her dialysis session. There was also a report of new post -dialysis weakness and confusion. Symptoms started to improve after administration of fluid and resolved completely within 4 hours.

There was no change in her home medication of atenolol, folic acid and nephrocap and patient was again observed to have similar symptoms 2 days later on her next hemodialysis. On further questioning patient reported a new use of topical Prilocaine-Lidocaine cream with cellophane wrapping after application.

Based on a high suspicion of Prilocaine-induced methemoglobinemia, the EMLA cream was discontinued. There was no further recurrence of symptoms with the next series of hemodialysis sessions.

Due in part to its high protein binding, the levels of prilocaine may transient be elevated during dialysis, particularly with ultra filtration in patients with ESRD without residual renal function. Topical Prilocaine applied cutaneously can be absorbed into the systemic circulation in sufficient amount to cause systemic symptoms. This systemic absorption is increased with application of a wrap over the applied skin site.

In our patient, the cellophane wrapping technique after application may had led to increase systemic absorption and features suggestive of transient methemoglobinemia as well as other features of Prilocaine toxicity

BILATERAL LOWER EXTREMITY SEQUENTIAL COMPRESSION DEVICES (SCDS) FOR THE MANAGEMENT OF INTRA-DIALYTIC HYPOTENSION – A NEW APPROACH TO AN OLD PROBLEM

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Intra-dialytic hypotension (IDH) is common, affecting up to 15%-50% of patients during HD. Various treatment/preventative modalities are currently available. These therapeutic options are often ineffective, or potentially hazardous (drug adverse effects). Moreover poor cardiac reserve, a common causative factor, is hardly amenable to any therapy.

Enhanced external counter pulsation (EECP) is increasingly being utilized in the long-term management of chronic CHF and otherwise refractory angina. EECP mechanistically improves venous return, enhances peripheral resistance and cardiac index. We hypothesized that bilateral lower extremity sequential compression devices (SCDs), presently used for DVT prophylaxis, could serve as “mini EECP” devices and stabilize the CVS during HD and prevent IDH.

We carried out an out-patient pilot study of bilateral SCDs to prevent IDH in 3 patients who otherwise had failed other treatment approaches (Table).

Age /Sex	Cause of ESRD	Other factors contributing to IDH	Result
68/M	Ischemic CM Sepsis	Reduced cardiac reserve Anemia Low albumin (2.9) Persistently Low SBP	Good.
42/F	ESLD HRS	Alcoholic cirrhosis Low albumin (1.8) Persistently Low SBP	Excellent
86/F	Hypertension	Low albumin (2.5) Gr 2 Diastolic dysfunction	Good.

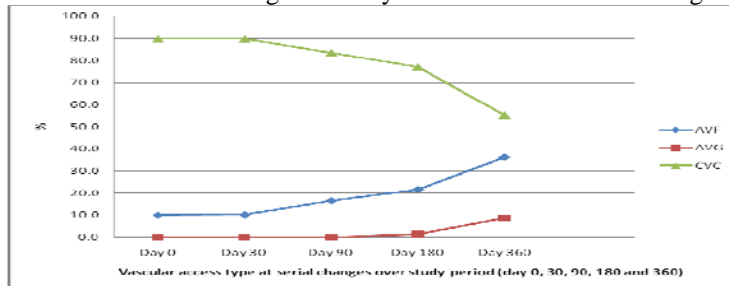
The SCDs were effective, convenient, and safe. We achieved ultra-filtration goals of 1-3 kg during hemodialysis sessions in all 3 patients, consistently, for months, a feat that was not possible without the SCDs. This new modality of preventing IDH is complementary to current existing therapies. Larger multicenter studies are warranted.

TIME AVERAGED VARIATIONS IN HEMODIALYSIS VASCULAR ACCESS TYPES AND THEIR DETERMINANTS AT AN INNER-CITY HEMODIALYSIS FACILITY

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Vascular access has a central role as the lifeline for hemodialysis (HD) patients. An arteriovenous fistula (AVF) is recommended as the access of first choice by many different HD guideline committees. However, despite these recommendations, there remains much variation in vascular access use among dialysis units.

We conducted a cohort study of HD vascular access types in 80 patients and their rates of change from one type to another over a 12 month period. The mean rates of vascular access use were: AVF, 44%; grafts [AVG], 5%; while central venous catheter (CVC) use was 51%. The rates of access change from days 0 to 360 is shown in the figure.



Whereas late presentation was the major determinant of initial vascular access at day 0, prolonged surgical wait time was the major determinant at day 30, 90 and 180. Economic difficulty was the major determinant at day 360. Other factors are: AVF failure; delayed AVF maturation; patient refusal; and patient preference.

Our AVF utilization rates fall short of the KDOQI/CMS goal of 66%. Efforts are underway to mitigate this deficiency.

IMPACT OF A NURSE-DRIVEN VASCULAR ACCESS MANAGEMENT PROGRAM ON ACHIEVING AND MAINTAINING OPTIMAL VASCULAR ACCESS FOR A CHRONIC HEMODIALYSIS (HD) POPULATION

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The purpose of this evidence-based practice improvement project is to monitor the impact of use of aggressive vascular access monitoring in addition to promotion of timely access placement on the overall percentage of patients who are actively using an arteriovenous fistulae (AVF) in the chronic hemodialysis (HD) unit.

The methods included analysis of the data collected since the opening of the unit for length of time between initiation of HD in the unit and placement of a permanent vascular access. Access monitoring techniques, measures of dialysis adequacy (URR and Kt/V), and nursing assessment of the access were analyzed to determine whether early intervention deemed necessary by these monitoring techniques had an impact on AV access function. Patient and staff education initiatives on improved access assessment and care were established.

The results of the continuous data analysis showed that the percentage of AVFs in use in the UCH Chronic unit in 2006 was 40% and in 2007, 60%. In 2008, the percentage of AVFs was 66% which exceeded the 65% goal of the Fistula First initiative. The time interval for permanent access placement decreased significantly over the years 2006-2008. In 2006, 15% were placed in 0-30 days and 8% in 31-90days; in 2008, 26% were placed within the first 30 days with 64% placed 31-90 days after starting chronic HD. At the end of 2008, 97.7 % of patients exceeded the goal URR of 70% and 98% had a Kt/V of > 1.4; 100% of access flow rates met or exceeded the goals of 400 ml/min (AVgraft) and 600 ml/min (AVF).

Our results indicate that a nurse in the role of vascular access manager provides a continuum of care and, in collaboration with the entire staff, can make an impact on the timeliness of vascular access placement as well as maintenance of access function. Utilizing new evidence (access assessment technique), technology (access flow monitoring) and patient education techniques (printed materials in addition to verbal instruction) can optimize patient outcomes.

U.S. INVESTIGATIONS OF HEALTHCARE-RELATED ADVERSE EVENTS IN HEMODIALYSIS PATIENTS, 1999-2009

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Frequent healthcare exposures and underlying illness may place maintenance hemodialysis patients at increased risk for healthcare-related adverse events. Investigation of these events can improve patient safety and inform prevention efforts. To determine the number and type of investigations of hemodialysis-related adverse events (HAE) in the United States, we reviewed published and unpublished reports of HAE investigations involving CDC during 1999-2009.

Twenty-one HAE investigations were conducted and a total of 441 HAEs were identified during the study period. Fifteen (71%) of the 21 investigations involved infectious disease outbreaks, resulting in 151 HAEs. These included investigations of hepatitis C virus transmission (n=7), bacterial and fungal bloodstream infections (n=5), infections caused by *S. aureus* not susceptible to vancomycin (n=2), and transmission of *Mycobacterium tuberculosis* (n=1). The remaining 6 (29%) investigations involved noninfectious outcomes that accounted for 290 HAEs. These included investigations of chemical intoxications (n=4), fatal vascular access hemorrhage (n=1), and allergic-type reactions from contaminated heparin (n=1). Failing to adhere to recommended screening, equipment maintenance, and/or infection control practices was implicated in 15 HAE investigations. Six investigations characterized novel disease mechanisms and allowed determination of appropriate control measures. Delays in reporting HAEs to public health authorities occurred in at least 5 investigations. Among dialysis providers, rapid detection and reporting of HAEs are necessary to improve patient safety and facilitate prevention efforts.

THE DOPPS PRACTICE MONITOR: A NEW INITIATIVE FOR TIMELY REPORTING OF US HEMODIALYSIS PRACTICES

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The Dialysis Outcomes and Practice Patterns Study (DOPPS) is a prospective cohort study of hemodialysis (HD) practices and patient outcomes based upon nationally representative samples of facilities and patients across 12 countries. In order to publicly provide reporting of a broad array of country-level HD practices, the DOPPS recently published the first DOPPS Annual Report (www.dopps.org). The DOPPS is now implementing a complementary initiative for the US, known as the DOPPS Practice Monitor (DOPPS PM), in which many aspects of US HD practice will be reported every 4 months. The DOPPS PM will be based on data collected from prevalent cross-sections of HD patients at >140 US HD facilities, with each report reflecting highly contemporaneous data collected ≤ 4 months prior to the Report.

A stratified random sampling design will allow the DOPPS PM to report **representative** findings for US in-center HD practice as a whole and also for facility strata (e.g., non-rural facilities, rural facilities, and facilities located >1 hr travel time to an urban center; hospital-based facilities; smaller and larger clinics; geographic region, etc). The DOPPS PM will follow major practice areas, including achievement of clinical practice guidelines and performance measures, other laboratory measures, medication use, staffing and services provided, changes between dialysis modalities, and transplant waitlisting. Provider surveys, as well as patient-reported results regarding satisfaction with care, out of pocket expenses, and quality of life will also be collected.

An important focus of the DOPPS PM will be to provide timely trends across many areas of HD practice and to evaluate the impact of the CMS fully bundled prospective payment system (2011), value based purchasing (2012), and other health policies which may impact delivery of HD care. The first DOPPS PM report is planned for autumn, 2010. To this end, the DOPPS welcomes input from the community regarding DOPPS PM scope and additional objectives.

A RANDOMIZED CONTROLLED TRIAL TO EVALUATE THE EFFECTS OF CINACALCET PLUS LOW-DOSE VITAMIN D ON VASCULAR CALCIFICATION IN HEMODIALYSIS PATIENTS

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Coronary artery calcification (CAC) is common and progressive in patients on dialysis; it may be aggravated by elevated plasma parathyroid hormone (PTH) and/or calcium (Ca) and phosphorus (P) levels. This prospective, randomized, controlled trial was designed to determine whether the treatment of hemodialysis patients with secondary hyperparathyroidism (sHPT) using cinacalcet and low dose vitamin D sterols attenuated the progression of CAC compared with flexible doses of vitamin D sterols alone. Hemodialysis patients with sHPT and detectable CAC (N=360), were randomized to treatment with cinacalcet (30-180 mg/day) plus low-dose vitamin D (≤ 2 μ g paricalcitol equivalent/dialysis session) or to flexible vitamin D therapy. sHPT was defined as PTH ≥ 300 pg/mL or PTH 150-300 pg/mL with Ca \times P > 50 mg²/dL² while receiving vitamin D. In both groups, calcium-based phosphate binders were used exclusively and the therapeutic target for PTH was 150-300 pg/mL. The primary end-point was % change in Agatston CAC score from baseline to week 52; additional analyses were also conducted using the volume score. We also report the change in Agatston calcium score for the aorta. Agatston CAC scores [median, (Q1, Q3)] increased by 31% (8%, 81%) from baseline in the flexible vitamin D group and by 24% (-1%, 63%) in the cinacalcet group, p=0.073. Volume CAC scores increased by 30% (10%, 78%) from baseline in the flexible vitamin D group and by 22% (2%, 52%) in the cinacalcet group, p=0.009. Agatston aorta scores increased by 33% (5%, 69%) and 20% (7%, 47%), respectively, in each group, p=0.073. Median plasma PTH levels decreased by 65 pg/mL (-184, 62) from baseline at week 52 in the flexible vitamin D group and by 132 pg/mL (-276, -24) in the cinacalcet group, p=0.018. In contrast, mean (95% CI) serum Ca increased by 0.17 mg/dL (0.06, 0.28) in the flexible vitamin D group but decreased by 0.51 mg/dL (-0.65, -0.37) in the cinacalcet group, p<0.001. Serum P decreased by 0.24 mg/dL (-0.55, 0.07) in the flexible vitamin D group and by 0.92 mg/dL (-1.28, -0.55) in the cinacalcet group, p=0.025. Total daily calcium intake did not differ between groups.

Cinacalcet with low dose vitamin D therapy attenuated progression of CAC although the difference in Agatston scores between groups did not reach statistical significance. Cinacalcet with low dose vitamin D provided better biochemical control of sHPT than flexible vitamin D alone.

EFFECT OF MEDICAID COVERAGE IN DIALYSIS

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Medicaid reimbursement by different HMO's is adversely impacting the dialysis units. In the absence of good monitoring system it is not uncommon to discover that frequently prescribed medications are not covered. Denial of home medications are usually reported by the patients but the denial of medications that are given intravenously during dialysis may get unnoticed. Most Medicaid payers do not pay for the intravenous dialysis medications and the routine monthly blood work. This can be a burden when numbers of patients on Medicaid is increased in any unit.

We decided to look for the unsuspected loss of revenue by giving uncovered medications to Medicaid patients in an inner city Philadelphia dialysis unit which is managed by a national chain and run by University Physicians. The unit has 139 patients and 40 (28.7%) of them have Medicaid coverage and 19 of them have only Medicaid without any secondary coverage. The vitamin D analog administration during dialysis is reviewed in 19 (13.6%) patients who have primary Medicaid. The Cost of blood work also reviewed

In just one month the unit lost \$4300 for all 19 Medicaid patients for only Vitamin D analog and loss of \$7200 per month for blood work for all uncovered Medicaid patients.

The alternate choices to avoid loss of revenue may be giving generic medications & or converting them to oral form. The loss of revenue from monthly blood work may be more difficult to resolve but contracting competitive local laboratory may provide some relief.

K-DOQI GUIDELINE GOAL ATTAINMENT AT HEMODIALYSIS INITIATION AND FIRST-YEAR SURVIVAL

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Individuals with end stage kidney disease have high mortality rate. In order to improve outcomes, the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (K-DOQI) developed evidence based guidelines for the care for patients with kidney disease. We aimed to determine, in a national cohort of incident hemodialysis patients, whether attainment of a greater number of K-DOQI guideline goals (dialysis vascular access, hemoglobin, and serum albumin) at dialysis initiation was independently associated, in a graded manner, with higher first-year survival rates.

Overall, 192,307 patients 20 years and older who initiated hemodialysis between June 1, 2005 and May 31, 2007 in the US were included and followed for 365 days from the first dialysis date. Guideline attainment was determined by physician report. The primary predictor variable was the number of guideline goals (0, 1, 2, or 3) met at dialysis initiation. The guidelines examined in the analysis were 1) arterio-venous (AV) fistula or AV graft used at initiation, 2) hemoglobin ≥ 11 gm/dl, 3) albumin at goal. Cox regression analysis was used to compare survival times adjusting for baseline characteristics.

At dialysis initiation, 59%, 30%, 9% and 2% of patients attained 0, 1, 2, or 3 guideline goals. After multivariate adjustment, mortality hazard ratios (95% CI) were 0.81(0.80-0.83) for those who attained 1, 0.53(0.51-0.56) for those who attained 2, and 0.34(0.30-0.39) for those who attained all 3 guideline goals, compared to those who did not attain any of the goals. Attainment of each of the individual goals was also associated with improved survival: 0.94(0.92-0.96) for hemoglobin at goal; 0.67(0.65-0.70) for albumin at goal; and 0.56(0.54-0.57) for those initiating dialysis with an AV graft or fistula.

Our findings suggest a graded association between greater attainment of evidence based guideline goals at dialysis initiation and lower risk of death during the first year on dialysis.

STREAMLINE BLOODLINES IMPROVE KT/V WHILE LOWERING DIALYSATE USAGE

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Blood flow and dialysate flow are critical inputs to achieve required dialysis dose (Kt/V). Blood flow for a given patient and their access is not generally different based on bloodline. We evaluated the Medisystems Streamline® bloodline (SL), designed to allow higher blood flows, and its impact on Kt/V and dialysate usage.

We compared Streamline to Medisystems ReadySet® bloodlines (RS) in a cross-over evaluation of 117 patients over 3 months. Data was collected once each month during lab draw days. After 1 month, patients were switched to SL. Blood flow and dialysate flow prescriptions were changed. No significant changes were made to dialyzers, delivered treatment time or needle sizes.

We found Streamline improved blood flows significantly while lowering arterial pressures across all access types. Because of the higher blood flows, we were able to maximize our adequacy target (100% of patients with Kt/V \geq 1.2) while significantly lowering dialysate usage and conserving fresh water for our facility.

	RS (Mar. '08)	SL (May '08)	Change	N	P
Average Kt/V	1.64	1.73	+5%	117	<0.001
% of Patients Kt/V \geq 1.2	96%	100%	+4%	117	
Blood Flow (mL/min): All	440	459	+4%	117	<0.001
Catheters	380	399	+5%	20	0.071
Fistulas	453	468	+3%	61	0.004
Grafts	470	479	+2%	32	0.098
Arterial Pressure (mmHg)	-196	-173	-12%	117	<0.001
Dialysate Flow (mL/min)	669	625	-7%	117	<0.001
Treatment Time	215	216	0%	117	0.179
Assumed Monthly Water Usage (L)*	404,000	377,000	-7%		

REPLACEMENT OF 25-OH VITAMIN D IN AN INDIGENT HEMODIALYSIS POPULATION

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Introduction: 25-OH vitamin D deficiency is highly prevalent in the dialysis population and is an independent risk factor for inflammation and cardiovascular disease. Following a recent policy change at our HD center, we surveyed all patients for 25-OH vitamin D deficiency and monitored outcomes after standard replacement therapy during a 6 month period.

Methods: 154 HD patients were studied. 92% African-American, 35% diabetic. 99% were deficient in 25-OH vitamin D. Ergocalciferol 50000u was administered once weekly for 4 weeks, then once monthly for 5 months. 25-OH vitamin D, alkaline phosphatase (AlkPhos), PTH, Albumin and reticulocyte hemoglobin content were measured. Paired T test employed for analysis.

Results: 43% were 25-OH vitamin D replete (serum level > 30ng/ml) after 6 months. 1 Pre- replacement , 2 Post- replacement

n 43	mean pre 1	mean post 2	mean diff	P value
AlkPhos	132.23	129.81	-2.42	0.805
PTH	653.02	636.65	-16.37	0.749
RetHgb	33.26	33.02	0.24	0.509
Albumin	3.94	3.96	0.02	0.590

Conclusion: In an indigent, largely African American dialysis cohort with close to 100% 25-OH vitamin D deficiency, we accomplished repletion in 43%. Markers of bone health and albumin showed a minimal trend towards improvement. We propose a larger study to further investigate these relationships.

Disclosure of Financial Relationships: nothing to disclose

CLINICAL OUTCOMES OF DIALYSIS CATHETER-RELATED BACTEREMIA (CRB) WITH CONCURRENT EXIT SITE INFECTION (ESI)

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CRB can frequently be treated with systemic antibiotics, in conjunction with an antibiotic lock, in an attempt to salvage the catheter. Catheters are replaced only in patients with persistent fever or bacteremia. It is unknown whether CRB associated with an ESI can be treated with this approach. To investigate this question, we retrospectively queried a prospective, computerized vascular access database, and identified 1436 episodes of CRB in 5 yr, of which 64 cases (4.4%) had a concurrent ESI. *Staphylococcus aureus* and *Staphylococcus epidermidis* accounted for 88% of CRBs in this group. Five serious complications (4 major sepses, 1 endocarditis) occurred in patients with CRB+ESI. These complications occurred in 20% (5 of 24) of *S. aureus* infections, but not with other organisms (Table 1). Early catheter removal (≤ 4 days) was more likely for *S. aureus* than *S. epidermidis* CRB+ESI (33 vs. 12.5%, $P=0.06$).

In conclusion, ESI occurs in a small proportion of CRB episodes, primarily those caused by *Staphylococcus*. Delay in catheter replacement is safe with *S. epidermidis*. However, catheters should be replaced promptly in *S. aureus* infections, due to the high risk of major complications.

Table 1	All CRB's	CRB + ESI	% with ESI	Serious complications
Bacteria				
All episodes	1436	64	4.4	5
<i>S. aureus</i>	393	24	6.1	5
<i>S. epidermidis</i>	335	32	9.6	0
<i>Enterococcus</i>	191	3	1.6	0
All Gram negative	436	3	0.7	0

HEMATIDE™ DOSE ADJUSTMENTS IN THE MAINTENANCE OF HB IN HEMODIALYSIS PATIENTS

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Hb variability in pts on hemodialysis (HD) may be associated with increased mortality (Yang, 2007). In a published retrospective analysis of 281 HD pts receiving rHuEPO for 1 yr (pts hospitalized >10 d were excluded), a mean of 6.3 (SD, 3.3) dose changes/pt/yr (0.53 dose changes/pt/mo) were reported (Fishbane, 2005). Frequent ESA dose changes may contribute to further Hb cycling and subsequent adverse outcomes (Fishbane, 2005). Fewer dose adjustments would thus be a desirable goal when managing HD pts. Hematide™ is a synthetic, peptidic ESA linked to polyethylene glycol that is in phase 3 development for the treatment of anemia associated with CKD. The purpose of the current analysis is to examine the frequency of Hematide dose adjustments in HD pts.

Eighty-one pts received Hematide QM (Q4W) in an ongoing, open-label, phase 2 extension study. Inclusion criteria included completion of a prior 24-wk, phase 2 dose-finding study. This unplanned analysis included 56 HD pts who received Hematide for ≥18 mo. We analyzed frequency of dose changes required to maintain Hb within target range (10-12 g/dL after protocol amendment in 2007; 11-13 g/dL in 2006) over 18 mo; results were then annualized. Patients hospitalized >10 d were not excluded.

Fifty-six pts receiving Hematide required a mean of 3.9 (SD, 1.6) dose changes/pt/yr (0.33 dose changes/pt/mo) to maintain Hb levels within target. Mean Hb level over 18 mo was 11.28 g/dL. Seven of 81 pts (8.6%) had AEs considered possibly related to Hematide; 2 pts had diarrhea; other AEs occurred in 1 pt each. The safety profile of Hematide appears consistent with that of other ESAs (Macdougall, 2008).

Compared with published reports of treatment with other ESAs, results from this analysis suggest that treatment with Hematide is associated with fewer dose adjustments to maintain Hb in HD pts. Controlled studies comparing Hematide with other ESAs are ongoing.

RENAL CELL CARCINOMA IN VON-HIPPEL LINDAU SYNDROME: TO SEVER OR TO SPARE

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Von Hippel-Lindau (VHL) disease is a rare autosomal dominant inherited disorder manifested by multiple cysts in different organ systems with the potential for malignant transformation. About 76% of patients with VHL have renal cysts and 70% of patients are at risk of developing renal cell carcinoma by age 60.

We present an unusual case of a thirty two year old black male who presented with flank pain and hematuria. He was seen two years earlier with similar complaints in the out-patient clinic and computed tomography (CT) scan done at that time showed bilateral renal masses; however, he was lost to follow up. His brother had undergone a nephrectomy and was on hemodialysis. To our patient's knowledge, his father had an unknown cancer. His physical examination revealed left costovertebral angle tenderness with a palpable left kidney. Complete metabolic panel showed creatinine of 1.9mg/dl which was elevated from his baseline 1.0mg/dl two years prior. Complete blood count was normal, urinalysis showed more than fifty red blood cells. A repeat abdominal CT scan revealed multiple cystic and solid lesions involving the pancreas and kidneys; he had four masses on his right kidney and three masses on his left, the largest kidney mass measured 5.5cm x 4.6 cm which had increased from the previous scan. At this time a strong suspicion for Von Hippel Lindau syndrome was entertained. A magnetic resonance imaging of the brain showed multiple lesions in the medulla and cerebral hemispheres which were consistent with hemangioblastomas. Subsequently genetic testing for VHL was positive. The treatment options were discussed with the patient and he opted for bilateral nephrectomy. The pathology was positive clear cell renal carcinoma.

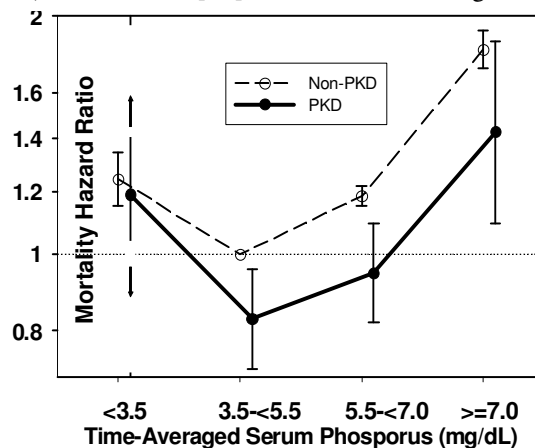
The optimal management of renal cell carcinoma patients with Von-Hippel Lindau is still unclear. The presence of multiple complex renal cystic lesions, rapidly enlarging renal masses, deteriorating kidney function, and poor compliance combined to make a compelling case for bilateral nephrectomy in our patient. The merits of total nephrectomy versus nephron sparing surgery are discussed in this report.

COMPARING MORTALITY-PREDICTABILITY OF HYPER-PHOSPHATEMIA IN MAINTENANCE HEMODIALYSIS PATIENTS WITH AND WITHOUT POLYCYSTIC KIDNEY DISEASE (PKD)

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Although observational studies show consistent associations between high serum phosphorus (P) & death risk in maintenance hemodialysis (MHD) patients (pts), it is not known whether PKD-MHD pts exhibit same or different associations. We examined a 3-yr (7/01-6/04) cohort of 58,917 MHD pts including 1,562 PKD pts in DaVita clinics using Cox models adjusted for case-mix & malnutrition-inflammation complex syndrome (MICS) including serum calcium and PTH. For each pt we calculated 3-yr-averaged P values based on weekly to monthly measured P levels over 3 yrs. PKD & non-PKD pts were 58.2±13.6 & 61.5±15.4 years old (mean±SD) & included 49% & 46% women, & 8% & 47% diabetics, respectively. In fully adjusted models across 4 P increments (<3.5, 3.5-<5.5 [ref], 5.5-<7.0 & ≥7.0 mg/dL, P in 3.5 to 5.5

mg/dL range was associated with greatest survival in both PKD & non-PCKD pts. Incremental association between hyper-P >5.5 mg/dL & increased death risk were similar in both groups (see Figure):



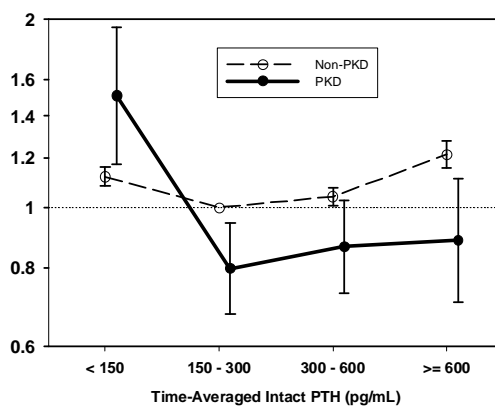
Hence, associations of 3-yr time-averaged serum P with death risk in PKD pts are similar to their non-PKD counterparts.

SECONDARY HYPERPARATHYROIDISM & SURVIVAL IN HEMODIALYSIS PATIENTS WITH & WITHOUT POLYCYSTIC KIDNEY

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Background: It is not known whether PKD patients (pts) who undergo hemodialysis (HD) treatment exhibit similar death risks pattern related to secondary hyperparathyroidism (SHPT) as non-PKD HD patients. **Methods:** We examined a 3-yr (7/01-6/04) cohort of 58,917 HD pts including 1562 PKD pts in DaVita dialysis clinics, whose survival was followed up to 6/06, using Cox models adjusted for case-mix, surrogates of Malnutrition-Inflammation Complex Syndrome and minerals. For each pt we calculated a 3-yr-averaged PTH value based on monthly to quarterly measured intact PTH over the entire 3 yrs. **Results:** PKD & non-PKD pts were 58.2 ± 13.6 & 61.5 ± 15.4 yrs old & included 49% & 46% women & 8% & 47% diabetics, respectively. In fully adjusted models across 4 PTH increments of <150, 150-<300 (reference), 300-

<600 & ≥ 600 pg/mL, PTH in 150 to 300 pg/mL range was associated with the greatest survival in both PKD and non-PKD pts. However, marked differences were noticed with both high and low



PTH levels between the 2 populations (see Figure):

Conclusions: The associations of 3-yr time-averaged PTH with survival in PKD pts, in whom $PTH < 150$ pg/ml is associated with highest mortality, appears different from non-PKD ps in whom $PTH > 600$ pg/ml is associated with the highest death risk.

CATASTROPHIC HYPERCALCEMIA AS A TECHNICAL COMPLICATION IN HOME HEMODIALYSIS

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Nocturnal home hemodialysis is associated with improved quality of life. We present a case where a life threatening event resulted from a technical error. The patient is a 46 year old woman with diabetic nephropathy, on home nocturnal hemodialysis for 6 years. She presented to the Emergency Room with history of vomiting, profuse sweating, and progressive deterioration in sensorium, which started 1 hour after initiation of dialysis the previous night. Because of the change in her status, her husband stopped her dialysis 3 hours after initiation. In the ER, she was stuporous, had a HR 48bpm and BP 98/56 mm Hg. She had no focal neurological deficits and systemic examination was normal. Serum calcium was 26 mg/dl (6.5 mmol/L). The corrected QT interval was shortened on the electrocardiogram. She was initiated on urgent hemodialysis with a low calcium bath. Over a period of next 6 hours, her clinical status improved commensurate with improvement in serum calcium. At the end of 6 hours of dialysis, her calcium had normalized to 10.3 mg/dl (2.57mmol/L).

Investigation as to the cause of the hypercalcemia revealed that there was a recent change in the Reverse Osmosis (RO) machine used for the dialysis. It was found that the drain port of the RO machine was connected to the dialysis machine and the product water was connected to the drain .The reversed lines resulted in high conductivity of the water samples connected to the dialysis machine. Thus the drain water from the RO which contained a very high concentration of calcium was used in the dialysate, resulting in acute hypercalcemia. This case illustrates the complexities involved in the care of a patient on home hemodialysis and how an error can lead to catastrophic outcomes. It also emphasises the importance of quality assurance in home hemodialysis.

ANALYSIS OF RENAL FIBROSIS AND WEIGHT OF SPONTANEOUSLY HYPERTENSIVE RATS (SHRS) PREGNANT EXPOSED TO PHYSICAL TRAINING

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Background: To evaluate the effects of exercise training in hypertensive pregnant rats on blood pressure (BP), heart rate (HR) and to correlate values of kidney weight with the percentage of renal fibrosis found. Study Design: We used SHR matched by age (240 ± 02 days) and weight (245 ± 12 g), and WKY, all also matched for age (250 ± 02 days) and weight (320 ± 20 g.) We divided the experimental groups: a) Group hypertensive sedentary (HS)(n=5), b) Group hypertensive trained (HC)(n=6), c) Group normotensive sedentary (NS)(n=5) d) Group normotensive trained (NC)(n=6). On the 20th day of gestation the animals were euthanized, and kidneys were removed and sent to heavy staining for making slides. Results: No significant differences were observed when we evaluated the heart rate (HR) of pregnant rats ($F=2.693$, $p=0.127$). The rats trained had lower values of MAP in comparison to the sedentary group ($T=89.000$, $p=0.028$). We observed a significant positive correlation between weight and kidney weight of rats ($rS=0.596$, $p=0.0115$). The rats belonging to the HS had lower kidney weights ($F=17.440$, $p=0.001$) when compared with the four groups. The hypertensive rats showed a higher percentage of fibrosis when compared with normotensive rats ($F = 9778$, $p < 0.001$). Conclusions: The present study indicates that physical training by swimming cause attenuation of PA and decrease in percentage of fibrosis in hypertensive pregnant rats.

RATIONAL SYNTHESIS OF A SELECTIVE RENAL OUTER MEDULLARY POTASSIUM CHANNEL (ROMK) ANTAGONIST

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ROMK critically regulates sodium and potassium homeostasis and may represent a drug target for a novel potent and potassium sparing diuretic. However, the molecular pharmacology of ROMK and the inward rectifier potassium channel family is essentially undeveloped. Thus, direct evidence for the proposed unique diuretic action of ROMK remains elusive. The purpose of this study was to identify selective, small molecule ROMK antagonists.

We screened approximately 225,000 small molecule compounds searching for ROMK modulators and identified several antagonists. One compound termed VU590 inhibits ROMK with an IC₅₀ of 300 nM, but also inhibits Kir7.1 at low micromolar concentrations (Lewis et al., 2009). While VU590 shows reasonable potency with suboptimal selectivity at ROMK, a structurally related compound denoted as BNBI selectively inhibited ROMK over Kir7.1 but with 10-fold less potency. Thus, selectivity and potency could be dissociated and suggested the possibility that a hybrid compound of VU590 and BNBI could act as both a selective and potent ROMK antagonist. Using this rationale, we synthesized VU591 and found that it not only inhibits ROMK with an IC₅₀ of 240 nM but is selective over Kir7.1 and several other related inward rectifiers (Kir2.1, 2.3, 4.1, and 6.2), voltage gated sodium, potassium, and calcium channels, calcium activated potassium channels (hSlo1/beta1) as well as a panel of 65 other potential off-targets. Patch clamp electrophysiology studies suggest that VU591 blocks the cytoplasmic pore of ROMK pointing to a selective binding site in this region. Taken together, VU591 represents a starting point to characterize a selective ROMK drug binding site and address the therapeutic potential of ROMK.

SODIUM (NA) INTAKE VARIES ACROSS THE AFRICAN DIASPORA AND IS ASSOCIATED WITH BMI

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Differences in Na intake may explain the wide range of hypertension (HTN) prevalence across the African Diaspora. Methods: The Sodium Study is a cross-sectional survey of adults of African ancestry from 3 different countries [Maywood, IL, U.S. (n=949), Kingston, Jamaica (n=1033), and Igbo-ora, Nigeria (n=964)] conducted from Dec 19, 2000 to May 30, 2003 and aims to explore the interaction between Na intake and genetic factors on blood pressure across the African Diaspora. We present the findings on Na intake and its association with BMI. Participants were adults >25 years old not receiving treatment for HTN and without diabetes. Blood pressure was measured using standardized methods and the average of 3 measurements was used. Na intake was defined as the mean Na excretion in three 24-hour urine collections on a usual diet. Participants were grouped by World Health Organization BMI categories and average Na intake and blood pressure were compared across BMI categories within each country using analysis of covariance.

Mean age (years) was 42 in U.S., 39.5 in Jamaica and 37.7 in Nigeria. Average SBP (mmHg) and daily Na intake were highest in U.S. (122.3 mmHg and 172 meq) and lowest in Nigeria (119.2 mmHg and 120.0 meq). Across BMI groups, average Na intake was consistently highest in the U.S. (figure 1). Among adults with BMI ≥ 30 kg/m², Na intake was 60 meq higher in U.S. adults compared to adults from Nigeria or Jamaica after adjustment for age and sex. Conclusion: Across the African Diaspora, Na intake is substantially higher in the U.S. compared to Nigeria or Jamaica, especially among the obese.

THE ASSOCIATION BETWEEN SODIUM (Na) AND POTASSIUM (K) INTAKE AND SYSTOLIC BLOOD PRESSURE (SBP) ACROSS THE AFRICAN DIASPORA

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Hypertension prevalence varies substantially across the African Diaspora and dietary factors likely account for some of this divergence.

We examined Na and K intake and BP among adults across the African Diaspora using data from the Sodium Study, a cross-sectional survey of adults of African ancestry, conducted from December 19, 2000 to May 30, 2003. Adults > 25 years old without hypertension treatment were enrolled in Maywood, IL (n=949), Kingston, Jamaica (n=1033), and Igbo-ora, Nigeria (n=964) with similar mean age at 42, 40, and 38 years respectively. Na intake was defined as the mean Na excretion in three 24-hour urine collections on a usual diet and BP was an average of 3 measurements.

The association between SBP and Na and K intake was determined using linear regression while adjusting for age, sex, BMI and regression dilution bias. Mean Na (meq) and K (meq) intake were 172 and 44 in U.S., 133 and 40 in Jamaica, and 120 and 45 in Nigeria. Mean Na intake was significantly associated with SBP in the U.S. and Jamaica but not Nigeria (Table 1). Na and K intake showed the strongest associations with SBP in the U.S. even after standardizing the coefficients for blood pressure variance across sites.

Differences in Na and K intake may explain in part the higher prevalence of hypertension in US Blacks compared to adults in Nigeria or Jamaica.

Regression Coefficients for Urinary Na and K excretion after adjustment for age, sex, BMI (sample of data)				
Site	Dependent	Independent	Coeff	95% CI
Maywood	SBP (mmHg)	Na (meq)	0.046	0.024, 0.067
		K (meq)	-0.689	-0.783, -0.596
Jamaica	SBP	Na	0.029	0.010, 0.048
		K	-0.032	-0.089, 0.025
Nigeria	SBP	Na	0.019	-0.007, 0.045
		K	-0.163	-0.227, -0.098

CORRELATION BETWEEN ROUTINE PERI-HEMODIALYSIS BLOOD PRESSURE MEASUREMENTS AND AMBULATORY BLOOD PRESSURE MEASUREMENT IN PATIENTS WITH END-STAGE RENAL DISEASE.

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Nephrologists routinely use blood pressure (BP) measurements during hemodialysis (HD) to assess BP control in patients with End-Stage Renal Disease (ESRD) on dialysis. Our study objective was to assess correlation between routine pre/post BP values on HD and results on 48-hour ambulatory blood pressure monitoring (ABPM).

Forty-three patients were recruited from the University of Mississippi Medical Center Outpatient Hemodialysis Unit. Data obtained included: age, race, gender, co-morbid illnesses vintage on dialysis, last 6 previous dialysis logs and 48 hour ambulatory blood pressure measurements. Data are presented as either percentage or means with \pm SD and analyzed with SPSS v.16 using ANOVA and logistic regression

Statistical significant correlation existed between diastolic pre-HD BP, systolic and diastolic post-HD BP and average systolic and diastolic BP on ABPM (p 0.01, <0.001 and 0.037 respectively). Systolic BP on ABPM and pre-HD did not correlate

Post-HD BP measurements correlate with ABPM measurements and should be used preferentially when assessing BP control in ESRD patients.

EFFECTS OF ALDOSTERONE RECEPTOR BLOCKERS IN RESISTANT HYPERTENSION AND STAGE III CHRONIC KIDNEY DISEASE

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Aldosterone blockade is not widely used in subjects with advanced chronic kidney disease (CKD) because of the risk of hyperkalemia.

We retrospectively reviewed our Hypertension Clinic database to evaluate the efficacy and safety of aldosterone receptor blockers (AldoRBs) added to a pre-existing antihypertensive regimen that included a diuretic and a renin angiotensin system (RAS) inhibitor in subjects with resistant hypertension and stage III CKD. Demographics, laboratory and medication data were extracted from the medical record for baseline (before starting AldoRBs) and most recent follow-up visits. The primary end-point was change in systolic blood pressure (SBP). Secondary end-points included serum potassium (K) and creatinine (Cr), eGFR, diastolic blood pressure (DBP), and tolerability. Statistical analysis was performed using paired t-test.

The analysis included thirty patients on spironolactone and six on eplerenone (mean dose 23 ± 10 and 50 ± 34 mg/day, respectively). Median follow up was 312 days. Mean age was 63 ± 11 years; 64% were male, 53% African-American and 28% diabetics. The mean number of antihypertensive agents was 5 ± 2 . AldoRBs induced a significant decrease in SBP from 162 ± 22 to 138 ± 14 mmHg ($p < 0.00001$). K increased from 4.0 ± 0.5 to 4.4 ± 0.5 mEq/l ($p < 0.0001$), with the highest value being 5.8 mEq/l. Serum Cr increased from 1.5 ± 0.3 to 1.8 ± 0.5 mg/dl ($p < 0.0004$) and eGFR decreased from 48.6 ± 9.0 to 41.2 ± 11.0 ml/min/1.73 m² ($p < 0.0001$). DBP decreased from 87 ± 17 to 74 ± 12 mmHg ($p < 0.00001$). Five patients could not tolerate spironolactone due to side effects and one developed acute renal failure that partially recovered after stopping spironolactone.

AldoRBs significantly reduced blood pressure in subjects with resistant hypertension and stage III CKD. They were generally safe and well tolerated although there was one case of acute renal failure highlighting the need for close biochemical monitoring.

RENAL ARTERY STENTING WORKS - IN THE RIGHT PATIENT

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Atherosclerotic renal artery stenosis (RAS) accounts for approximately 90% of all cases of RAS. It classically manifests after the age of 50 years and its presence carries significant morbidity and mortality. We present the case of a woman with significant bilateral RAS who had a favorable response to renal artery intervention (RAI).

A 74-year-old female with hypertension, and coronary artery disease was admitted to our hospital with a blood pressure of 200/100 mm Hg. Her serum creatinine was elevated at 2.5 mg/dl compared to a normal baseline. Her hospital course was complicated by multiple episodes of flash pulmonary edema. An MRA revealed bilateral 90% ostial renal artery stenosis. She underwent left renal artery angioplasty and stent placement. However, she continued to have episodes of flash pulmonary edema. A repeat angiogram performed revealed a 50% stenosis and dissection distal to the left renal artery stent with a failed attempt at revascularization. She developed another episode of pulmonary edema, became anuric and her creatinine increased to 5.6 mg/dl. A third attempt at revascularization, albeit considered very challenging, was offered to the patient. Both renal arteries were successfully stented using a brachial artery approach. Her urine output improved immediately after the procedure with decline in her serum creatinine to 1.2 mg/dl over the next 3 weeks.

Despite significant advances in techniques of RAI, the indication for renal artery stenting remains controversial due to an unproven benefit of RAI compared to best medical therapy. Furthermore, consistent and reliable predictors of a favorable response to RAI in terms of renal function remain unknown making it very challenging to select the right patients for RAI. Our patient had a favorable response to the RAI as suggested by significantly lowered requirement of antihypertensive medications and rapid improvement in renal function after the procedure. This case illustrates that careful patient selection and an adequately skilled interventionist are of paramount importance in managing renal artery stenosis by a vascular intervention.

NEW ONSET REFRACTORY HYPERTENSION AND HYPOKALEMIA ASSOCIATED WITH ECTOPIC RENIN SECRETION DURING THE FIRST TRIMESTER OF PREGNANCY

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The objective of this report is to describe a case of refractory hypertension and hypokalemia during first trimester of pregnancy resulting from ectopic renin production from molar pregnancy.

A 34-year-old female presented to hospital with headache, nasal bleeding and vaginal spotting. Physical examination was remarkable for BP 200/110 mmHg, rales at the bases of both lung fields, and gravid abdomen. Laboratory data were significant for potassium 2.7 mmol/L, bicarbonate 25 mmol/L, sodium 131 mmol/L, B-type natriuretic peptide 1820 and random urine potassium 104. Moreover, an echocardiogram revealed severe left ventricular dysfunction with ejection fraction (EF) of 25%. Further work up for secondary causes of hypertension showed an aldosterone and random renin levels of 98ng/dL and 32 ng/ml/hr, respectively. Additionally, pelvic sonography showed a single intrauterine fetus (estimated gestational age 12 weeks) and a complex vascular mass within the endometrium consistent with complete molar pregnancy. To further investigate the source of renin production, an MRI of the abdomen and pelvis showed a complex vascular mass within the endometrium consistent with complete molar pregnancy. These findings were confirmed by pelvic sonography. Subsequently, the patient underwent dilatation and curettage (D&C). Pathologic examination of D&C was consistent with molar pregnancy. Soon after D&C, blood pressure and serum potassium normalized. Three month later, stress echocardiogram showed normalization of EF (65%).

This case highlights the complexity of hypertension work up during the first trimester of pregnancy. Moreover, molar pregnancy should be considered in the differential diagnosis of hypertension and hypokalemia in pregnant women.

VALIDATION OF A PAPER BASED DIETARY POTASSIUM TEST FOR ADULTS WITH CHRONIC KIDNEY DISEASE

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High plasma potassium levels are common in patients with chronic kidney disease (CKD) and cause life threatening cardiac dysrhythmia. Self management by dietary restriction of potassium is critical. Currently, there is no valid instrument to quickly assess patient's knowledge of dietary potassium. The purpose of this observational study was to develop and validate a novel paper-based test of knowledge of foods containing high versus low potassium (test), designed for clinical use in adult patients with CKD who need to restrict dietary potassium intake. The 21 item test allows individuals to choose from two food items, asking them to select the item lower in dietary potassium. Two forms (A) and (B) of the test were designed as parallel forms. Statistical analysis was determined using SAS, version 9.1. 34 participants with high knowledge and 34 participants with low knowledge of dietary potassium and 10 renal dietitians participated in the research by taking the potassium test. 10 renal dietitians also rated each item as essential/non-essential for patient knowledge.

	Form A	Form B	P value
Kuder-Richardson 20	0.71	0.68	
Pearson's Correlation	r = 0.81		< .0001
Kruskal-Wallis Test	Mean (s.d.)	Mean (s.d.)	
High Knowledge Patient	16.8 (2.4)	16.7 (2.3)	<.0001 (A)
Low Knowledge Patient	12.3 (3.1)	12.9 (3.0)	<.0001 (B)
Dietitian Knowledge	20.9 (0.3)		<.0001

In conclusion, among the VA patients with moderate to severe CKD, the 21 item test (form A) has good reliability, excellent construct validity and internal consistency. The majority of the items were rated as essential knowledge by the majority of the renal dietitian raters. It is easy to administer and score, and was well-accepted by participants. This novel instrument may be useful in clinical, educational, and research settings to test patients' knowledge of dietary potassium restriction, which is a critical domain of self-management for patients with CKD.

C-REACTIVE PROTEIN (CRP) PREDICTS RESPONSE TO NUTRITIONAL ENHANCEMENT IN HEMODIALYSIS PATIENTS WITH LOW SERUM ALBUMIN

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We studied whether CRP levels in hypoalbuminemic HD pts predict response to nutritional enhancement.

HD pts are at high risk for hypoalbuminemia from malnutrition and chronic inflammation. Studies report poor outcomes in ESRD pts with low albumin levels.

169 HD pts with serum albumin < 4 g/L were studied. CRP cut off level was < 5 mg/dl. The Low-Albumin-Low-CRP (LALCRP) group (presumed dietary etiology) and Low-Albumin-High-CRP (LAHCRP) group (presumed inflammatory etiology) were compared for response to dietary and protein supplement enhancement. Kt/V was monitored for adequacy and consistency. Primary and secondary outcomes were #1) achieving serum albumin level > 4 g/l and #2) all-cause mortality respectively.

Our results showed 92/169 (54%) pts had low serum albumin. 34/92 (37%) and 58/92 (63%) of pts comprised LALCRP and LAHCRP groups respectively. All had monthly albumin checked for 9 months. 77.42 % of LALCRP vs. 41.9% of LAHCRP pts achieved the Primary Outcome of Alb level response (> 4g/l). Fisher's Exact test: p= 0.004, (95 % Two sided CI = 58.9%, 90.4% and 28.5%, 60.3% for LALCRP and LAHCRP groups respectively). Fisher Exact test for survival tended to favor LALCRP but was not significant, p = 0.12.

We conclude that normal CRP levels in hypoalbuminemic HD pts predict higher likelihood of albumin normalization with enhanced dietary and supplemental protein. Similarly, elevated CRP in this setting portends less probability of success. We hypothesize that this difference helps delineate a dietary vs. inflammatory etiology of hypoalbuminemia in this setting.

THE EFFECT OF NUTRITION COUNSELING ON MALNUTRITION AND AZOTEMIA IN ADULT VETERANS WITH CHRONIC KIDNEY DISEASE (CKD)

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Dietary counseling in patients with an estimated glomerular filtration rate (eGFR) ≤ 60 mL/min/1.73 m² is an integral part of therapy as it can prevent or improve symptoms of uremia. The purpose of this retrospective study was to investigate the effect of dietary counseling on body mass index (BMI), serum albumin, and blood urea nitrogen (BUN) in pre-dialysis patients.

Medical records of male veterans within VAGLAHS with Stages 3-5 CKD were randomly examined until four hundred patients who met study criteria were identified and entered into the study: 200 who had received nutrition counseling and 200 who had not.

Study subjects ranged in age from 36 to 92 years, with a mean of 70.5 years. Results of two-tailed independent *t*-tests showed no significant differences in BMI, serum albumin, or BUN in subjects who received nutrition counseling versus those who received no nutrition counseling. In subjects who received low protein diet counseling versus those who did not receive low protein diet counseling, results of 3-way ANOVA indicated no significant differences in BUN or serum albumin levels.

Of the 1,430 patient records reviewed, 200 subjects (14%) received nutrition counseling by a Registered Dietitian. Based on Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines (2000), all 1430 patients should have received nutrition counseling since they had a eGFR < 60 mL/min/1.73m². All study subjects had a mean BUN level above the normal range, suggesting that on average, subjects who received nutrition counseling were referred to an RD with late, rather than early, stages of CKD.

Results of this study indicate that KDOQI guidelines (2000) to evaluate nutrition status and monitor a panel of nutrition measures in patients with a eGFR < 60 mL/min/1.73 m², such as albumin and body weight, are not being implemented at VA GLAHS on a regular basis.

ASSESSMENT OF EXTRACELLULAR FLUID VOLUMES OBTAINED BY DIFFERENT METHODS IN MULTI-ETHNIC ASIAN CHRONIC KIDNEY DISEASE PATIENTS

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The measurement of extracellular fluid volume (ECFV) in chronic kidney disease (CKD) patients can be determined by non-invasive multi-frequency bioimpedance analysis (BIA) or radio-isotope clearance measurements (GFR). Use of GFR-ECFV is limited by time, repeated plasma sampling, and radiation exposure; whereas BIA-ECFV is a calculated measure from a reference using proprietary data, of which it is unknown if data from CKD patients was included. We hypothesize that BIA-ECFV will be biased when compared to GFR-ECFV in Asian patients with CKD.

We used data from a prospectively recruited cohort of 232 CKD patients (52% male, Chinese 40.5%, Malay 32%, others 27.5%). GFR and GFR-ECFV were measured simultaneously by three-sample plasma clearance of ^{99m}Tc -DTPA followed by normalization to du Bois body surface area and Brochner-Mortenson correction. BIA-ECFV was measured on the day of the GFR test by multi-frequency BIA using Bodystat Quadscan 4000 (Isle of Man, British Isles). The results were compared across gender, ethnicity, and GFR above and below 50 mL/min. Statistical analyses were performed with JMP 7 (Cary, NC, USA). Bias in BIA-ECFV was determined by Bland-Altman analysis of agreement.

The overall means were: age 58.4 ± 12.8 years, measured GFR 51.7 ± 27.5 mL/min/ 1.73m^2 , BIA-ECFV 15.9 ± 2.7 L, and GFR-ECFV 16.3 ± 4.8 L. There was no difference in ECFV by either method (mean difference = -0.41 ± 4 L, $P = \text{NS}$). There was no difference in ECFV by ethnicity and in males, but was lower in females by BIA (mean difference: -0.86 ± 4.4 L, $P = 0.04$), and when GFR was < 50 mL/min (0.97 ± 3.0 L, $P < 0.001$). However, mean GFR-ECFV was greater than BIA-ECFV by 2.2 ± 4.5 L ($P < 0.001$) when GFR was greater than 50 mL/min.

BIA-ECFV determination is similar to invasive GFR-ECFV overall. There is greater bias at earlier stages of CKD ($\text{GFR} > 50$ mL/min).

IMPROVED PATIENT PHOSPHORUS OUTCOMES WITH THE USE OF LANTHANUM CARBONATE

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Maintaining serum phosphorus can be difficult for hemodialysis (HD) patients even with dietary counseling and oral phosphorus binder therapy. Through observation in our clinics, we noted that patients on the non-calcium binder lanthanum carbonate experienced better phosphorus control than other non-calcium resin based binders.

To determine if lanthanum carbonate helped patients within our clinic maintain phosphorus with KDOQI guidelines of 3.5-5.5 mg/dL, we assessed phosphorus, calcium, PTH and albumin levels in patients starting on lanthanum carbonate between January 2009 and December 2009. A total of 33 patients who either switched to lanthanum carbonate from a non-calcium resin based binder (n=14) or calcium-based binder (n=5) or who started on a phosphate binder for the first time (n=14) were included in the study. The last lab values prior to the switch to lanthanum carbonate (baseline) were compared to lab values 2 months after starting on lanthanum carbonate.

The mean phosphorus levels decreased from baseline levels of 6.4 ± 1.8 mg/dL to 4.7 ± 1.0 mg/dL, $p < 0.001$ after 2 months on lanthanum carbonate. PTH also decreased significantly from 466 ± 311 pg/ml (baseline) to 319 ± 180 pg/ml (lanthanum carbonate), $p < 0.05$. After 2 months on lanthanum carbonate, the percent of patients meeting KDOQI guidelines increased from a baseline of 33% to 80%.

Comparing only patients who switched from a resin-based binder to lanthanum carbonate showed decreased phosphorus (6.4 ± 1.7 vs. 4.7 ± 1.3 , $p < 0.01$) and increased percent of patients within KDOQI guidelines (36% vs. 71%). Calcium and albumin did not change significantly. Within our clinics, the type of phosphate binder positively affected phosphorus levels but calcium and albumin levels were unchanged.

IMPLEMENTATION OF VITAMIN D FOR STAGE 3 TO STAGE 5 CHRONIC KIDNEY DISEASE PATIENTS

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The kidney plays an essential role in vitamin D (vit D) metabolism by activating 25-hydroxy D₃ (25 (OH)D₃) to 1,25 dihydroxy D₃. In chronic kidney disease (CKD) vit D deficiency could contribute to secondary hyperparathyroidism (HPT). We monitored 25 (OH)D₃, parathyroid hormone (PTH), serum albumin, calcium and phosphorus levels among 441 stage 3- 5 CKD patients in our regular renal clinics and initiated ergocalciferol (D₂) 50,000 IU weekly for 12 weeks for patients whose 25(OH)D₃ levels were \leq 15ng/ml and cholecalciferol (D₃) 1000 IU daily for patients whose 25(OH)D₃ levels were 16 – 30ng/ml. Patients were followed for 6-9 months and data analyzed on 361 patients (265 stage 3, 85 stage 4 and 11 stage 5) who were not receiving active vit D.

Average age was 66.2 ± 13.7 (range 21-95) years. 25 (OH)D₃ levels improved after vit D supplementation: 18.5 ± 7.6 ng/ml to 36.5 ± 11.1 ng/ml ($p < 0.05$) in stage 3, 22.1 ± 10.4 ng/ml to 39.2 ± 11.9 ng/ml ($p < 0.05$) in stage 4 and 15.2 ± 5.6 ng/ml to 42.4 ± 15.3 ng/ml ($p < 0.05$) in stage 5 patients. PTH levels significantly improved in stage 3; 95.8 ± 57.8 pg/ml to 83.7 ± 55.2 pg/ml ($p < 0.0001$) but were not significantly changed in stage 4 (162.2 ± 91.6 pg/ml to 160.1 ± 95.1 pg/ml (NS) or stage 5 patients (207.8 ± 135.7 pg/ml to 192.4 ± 145.9 pg/ml (NS). There were no significant changes in serum albumin, adjusted calcium, and phosphorus in all stages of CKD. Linear regression predicting PTH levels show a significant inverse association between 25(OH)D₃ and PTH levels in stage 3 CKD group ($R = -.173$, $R^2 = .030$, $p < 0.01$). Stage 4 and 5 patients had increased or no changes in PTH levels despite vit D supplementation, presumably due to severity of CKD and decreased conversion to 1,25 dihydroxy D₃.

These findings suggest that vit D supplement may be more effective for the treatment of secondary HPT in early stages of CKD. Although there may be other benefits of vit D supplements in stage 4 and 5 CKD, these findings suggest active vit D therapy will be needed.

PICA: AN IMPORTANT AND UNRECOGNIZED PROBLEM IN PEDIATRIC DIALYSIS PATIENTS

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Pica, defined as the compulsive consumption of non-nutritive substances, is thought to be increased in the dialysis population. Little is known regarding the incidence or the metabolic complications resulting from pica, particularly in children. The objective of this study was to determine the prevalence of pica among patients in our pediatric dialysis center.

Eighty-seven patients followed on chronic dialysis therapy were surveyed for consumption of non-nutritive substances. Those with pica were assessed for demographic, nutritional and metabolic characteristics. Dialysis efficiency was estimated by calculating urea clearance per patient volume (Kt/V). Seventy-seven percent of patients were receiving hemodialysis (HD) 3-4 x weekly on hollow fiber dialyzers. Twenty-three percent of patients were maintained on peritoneal dialysis using nightly cycling (CCPD).

The race/ethnicity of the population was predominantly non-white (93%). The patients' mean age was 17.2 ± 7.2 years. Dialysis efficiency reflected by Kt/V averaged 1.5 ± 0.5 . The survey indicated that 46% of the patients experienced some form of pica divided into simple "ice" pica (34.5%) versus "hard" pica (12.6%). Hard pica included the compulsive consumption of chalk, starch, soap, sand, clay, ajax cleanser, sponge and potting soil. Those on HD were 8.3 times more likely to have hard pica compared to those on CCPD. Greater than 5 years on dialysis was associated with a 3.2 odds ratio (OR) of having pica ($p=0.02$). Anemia was the most significant morbid association with pica, occurring at an $OR=4.4$ ($p=0.001$) for all pica and 6.5 ($p=0.02$) for hard pica. Once pica was initiated, an "addictive" nature to the consumption became apparent. Intervention consisted of behavioral modification employing substitution strategies by child psychology.

In conclusion, our data indicate that pica is a prevalent and potentially harmful affliction that needs further attention in the nutritional management of dialysis patients.

**RCT OF PERSONAL DIGITAL ASSISTANT (PDA)
SUPPORTED DIETARY INTERVENTION TO REDUCE
SODIUM INTAKE IN PD**

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Reducing sodium intake is critical in PD patients (pts) to reduce intravascular volume expansion, hypertension, left ventricular hypertrophy (LVH), and permit the use of lower dextrose dialysis fluid. This RCT investigated whether an individualized PDA-assisted dietary adherence enhancement program based on Social Cognitive Theory reduced sodium intake in PD patients.

This 4-mo RCT randomized 26 PD pts with an average age of 51.7 years (SD=16.4) and average duration of PD of 3.4 years (SD=4.0). Intervention pts monitored dietary intake with a PDA programmed with their dietary prescription and received PDA feedback regarding % of daily targets consumed and counseling based on Social Cognitive Theory. Both groups reviewed computer-based dietary educational programs. Outcomes were evaluated at baseline and 4 mo from dietary recalls, blood pressures, and the PD Dietary Problems questionnaire. Change scores were compared using Student's t test. Data regarding adherence and acceptability were obtained from intervention pts.

Nineteen pts (10 intervention and 9 controls) completed the study. Intervention pts reduced their intake of Na⁺ by 187mg (SD=662), control pts increased Na⁺ by 44mg (SD=1,209; delta 231 mg/d, p=0.082). Blood pressures were unchanged. Mean score on the PD Dietary Problems questionnaire increased 0.5 points (SD=20.1) in control pts and decreased 10.5 points (SD=16.2) in intervention pts (p=0.194). Intervention pts entered an average 212.1 meals (SD=112.1) over 16-weeks or 1.9 meals per day. 86.7% of intervention pts said they would use the PDA and BalanceLog to monitor their diet after the study ended.

This small pilot study showed a PDA-supported dietary intervention to be feasible and acceptable in PD patients. Further study with a larger sample is needed to determine if this approach results in fewer dietary problems and reduces dietary Na⁺ consumption.

SUPERMARKET TOURS AS A NOVEL TOOL FOR TEACHING RENAL NUTRITION TO CKD STAGE 3, 4, AND 5 PATIENTS

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Dietary education is important for managing the nutritional well-being of CKD patients. This typically occurs in a 1:1 clinic setting. However, patients often express problems in finding kidney friendly food products on supermarket shelves and difficulty in understanding how to read labels. To help patients address these issues in a familiar setting, a supermarket tour was developed by a renal dietitian at St. Paul's Hospital. The free 90 minute tour was lead by a renal dietitian, at a local supermarket. Aisles containing breads, cereals, crackers, cereal bars, cookies, beverages, and soups provided the best learning opportunities. Label reading focused on sodium and phosphorus content of foods based on the guidelines from the National Kidney Foundation "Your Guide to the New Food Label, 2008". Participants were able to ask the dietitian questions "on the spot" about specific food products along the aisles. Responses to a questionnaire given at the end of each tour were positive. One hundred percent responded they were introduced to new kidney friendly products and 100% felt they would be able to locate them in the store. Ninety-three percent felt more comfortable reading labels. Participants felt the tour provided opportunity to dialogue with other patients. They felt "special" that a program was individualized for them. Overall, the tour met the objective of participants identifying kidney friendly foods on the supermarket shelf and increasing their ability to read labels. To generate more participants, future tours may be offered in different geographical locations and in ethnic supermarkets.

**INDIVIDUALIZED INTRADIALYTIC PARENTERAL
NUTRITION (IDPN) SOLUTIONS INCREASE ALBUMIN
LEVELS IN PATIENTS WITH CHRONIC KIDNEY DISEASE
(CKD) STAGE FIVE**

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Background: Many CKD Stage 5 patients are nutritionally compromised, reflected by low serum albumin levels. Research has shown that improvement in albumin levels decreases mortality in this population. IDPN is an available alternative therapy for patients who require a more aggressive approach for improving nutritional status, specifically weight and albumin status, when oral intake is insufficient. **Purpose of the study:** Determine if albumin levels would increase in patients well managed on IDPN therapy.

Methods: A retrospective, longitudinal analysis of patients receiving IDPN solutions using data from the NPR electronic medical record was conducted. Inclusion criteria were the initiation of IDPN therapy from August 8, 2008 through March 31, 2009 and six months of continuous therapy with no breaks greater than three weeks. Patients were followed from August 1, 2008 through September 30, 2009.

Results: There were 145 patients that met the inclusion criteria and that were reviewed. Initial albumin levels for all patients had a mean of 2.93 (95% CI 2.85-3.02) and a mean albumin level at the end of six months of 3.40 (95% CI 3.33 -3.47) $p<0.0001$. Patient weights at the beginning of the study period had a mean of 154.6 pounds (95% CI 147.9-161.3) and at the end of six months patient weights were average 151.9 pounds (95% CI 145.6 – 158.2), $p=.17$. There was not a significant loss of weight in the study population.

Conclusions: IDPN is an important alternative for patients with CKD Stage 5. In this study use of these solutions in a well managed approach indicate that albumin, a primary marker of patient nutritional status and perhaps mortality, was improved. Further longitudinal studies are needed to explore the impact of IDPN on patient mortality.

BIOTIN DEFICIENCY AND RESTLESS LEGS SYNDROME IN DIALYSIS

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¹Missoula, MT, USA, ²Little Rock, AR, USA, Studies suggest that micronutrient depletion may contribute to neuropathic problems in ESRD patients. We assessed the relationship of RLS in individuals receiving chronic dialysis to their biotin status as judged by activity of a biotin-dependent enzyme, propionyl-CoA carboxylase (PCC), in peripheral blood lymphocytes (PBL). 70 patients and 8 control subjects participated. Diagnosis of RLS was made using the International Restless Legs Scale. PCC activity was measured by a ¹⁴C-HCO₃ incorporation assay and PCC activation coefficient (AC) was measured after ex vivo incubation of PBL with 10 nM biotin. PCC activity and PCC AC were not significantly different between the dialysis group and the control group. However, PCC AC did approach significance (p = 0.06). For PCC AC, 19/61 (31%) of the subjects had activation coefficients of greater than 1.1 suggesting impaired biotin status. Accordingly, we examined the biotin status of the subgroups with and without RLS. PCC AC was significantly greater (p = 0.012) in the subgroup with RLS. The prevalence of RLS was 35/61 (57%) in the dialysis group. It appears that RLS symptoms in this population may be an indicator of biotin deficiency.

EFFECT OF PROTEIN SUPPLEMENTATION IN CHRONIC DIALYSIS

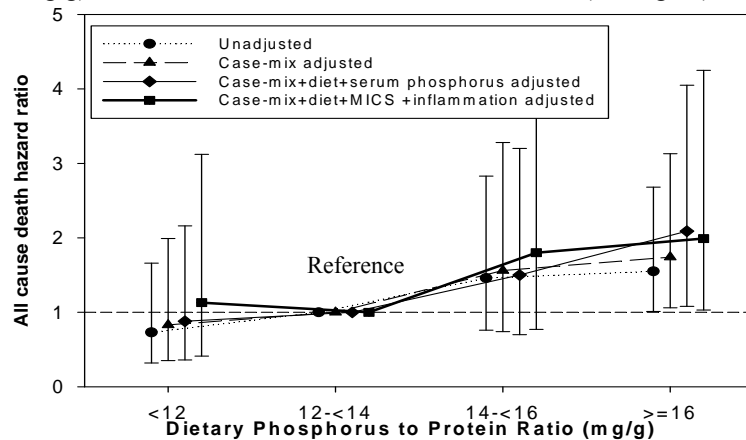
Heidi D. Moretti, Missoula, Montana, USA, Andrea M. Johnson, Tammy J. Keeling-Hathaway.

Our aim was to evaluate the impact of oral protein supplementation given during hemodialysis and peritoneal dialysis on nutrition status, number of hospitalizations, and length of stay. We used a randomized crossover design in which serum albumin, normalized protein catabolic rate (nPCR), total hospitalizations, and length of stay were compared in patients receiving protein supplements to those who did not. The study was conducted for a period of one year. Subjects included 49 patients treated with hemodialysis or peritoneal dialysis for at least three months. nPCR significantly increased by the fourth month of treatment from baseline of 1.05 to 1.16 ($P=0.007$). The control group had a significant decline in nPCR during the first six months and second six months, ($P=0.038$ and 0.024 , respectively). Improvement was seen in albumin in the treatment group by month 3 from 3.49 to 3.52 ($P=0.035$), but this was not sustained. In the second six months, the control group had a significant drop from 3.35 to 3.19 ($P=0.014$) and the difference between protein and control groups was significant in the second six months as well ($P=0.037$). When protein supplementation ended, weight significantly dropped for those with BMI less than 20. Trends for reduction in hospitalization admissions and hospital days were seen in both crossover treatment groups.

ASSOCIATION OF DIETARY PHOSPHORUS TO PROTEIN RATIO WITH MORTALITY IN HEMODIALYSIS PATIENTS

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Epidemiologic studies show an association between higher pre-dialysis serum phosphorus (P) and death risk in maintenance hemodialysis (MHD) patients (pts). There are little data about the effects of the ratio of dietary P to protein (P/P) on outcomes. We examined 5-year (2001-06) survival predictability of dietary P/P ratio, estimated from the Block's food frequency questionnaires, at the start of a cohort of 224 MHD pts. The P/P ratio was divided in 4 increments: <12, 12 to <14 (reference), 14 to <16 & ≥ 16 mg/g. We adjusted for: (1) case-mix, sevelamer or calcium- binders & residual urine; (2) dietary energy, protein & K; (3) Malnutrition inflammation complex syndrome (MICS), EPO & vitamin D doses, nPCR, & BMI; & (4) inflammatory markers (CRP, IL-6, TNF α). MHD pts in the highest P/P group (≥ 16 mg/g) exhibited almost 2-times increased death risk (see Figure):



Hence, higher dietary P/P ratio is associated with increased death risk in MHD pts, even after adjustments for serum P, type of P-binder & dietary protein, energy & K intake.

WALKING DISABILITIES AND NUTRITIONAL STATUS IN LONG-TERM HEMODIALYSIS PATIENTS

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According to USRDS, walking disabilities (WD) are prevalent in the hemodialysis (HD) population and predict increased mortality in these patients. Malnutrition, also common in HD patients, confers similar morbidity and mortality risk. However, there have been no studies to determine whether WD are a cause or consequence of poor nutritional status in HD patients. This retrospective study investigates the relationship between acquired WD and nutritional status in HD patients.

Medical records of HD patients who received treatments at our dialysis facility between July 1, 2004 and April 1, 2009, were screened for documentation of a WD. WD was defined as the use of a cane, walker, or wheelchair. Mean serum albumin levels from up to 6 months before and after the documented onset of the WD were compared using a paired t-test.

Onset of WD was documented in 144 of 740 (20%) hemodialysis patients. Mean serum albumin levels before and after onset of WD were 3.77 ± 0.37 and 3.71 ± 0.47 g/dL, respectively, $p=0.06$.

Serum albumin levels are low and tend to worsen in HD patients who develop WD. As a result, nutritional status is further compromised and mortality risk increased. Thus, patients with WD will require additional nutrition intervention.

BENEFICIAL EFFECT OF EXERCISE ON BODY COMPOSITION-A HEMODIALYSIS CASE STUDY

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It has been well documented that exercise is beneficial for dialysis patients. However, few participate in any sustained exercise program, aside from informal activities of daily living. An exception to this is JG, a renal replacement consumer since 1994.

In 2003, after treatments with both PD and transplant, JG was receiving in-center HD. She participated in a six month study to evaluate the benefit of exercise while dialyzing. The intervention was riding a stationary bike for 30 minutes three times a week during the first two hours of dialysis. Participants were encouraged to maximize their exercise time to two hours each HD session as tolerated, a goal that JG was able to achieve. Total body DEXA scans were obtained pre-study and six months later (end of study) to evaluate body composition.

JG felt better while participating in the study so she decided to continue to ride the bike for two hours each HD even after the study was completed. She also added other regular exercises over time including water aerobics, yoga and tai chi. Prior to the study, JG's exercise consisted of walking her dog and caring for her lawn, informal activities she continued as she added structured exercise to her life.

In 2008, JG participated in a second research protocol where a baseline DEXA was performed. A comparison of the two pre-study DEXAs illustrated the long-term benefit of her exercise efforts. In 2003 at age 48 years, JG weighed 68 kg (BMI 28.4). Her lean mass was 41.8 kg, fat mass 24.0 kg, and body fat 36.5%. In 2008 at age 53 years, JG weighed 59 kg (BMI 24.5) with a lean mass of 39.5 kg, a fat mass of 17.5 kg, and a body fat of 30.7%.

JG's weight loss and reduction of body fat while maintaining her lean mass, impressive for the healthy population, is even more impressive considering her ESRD and ongoing HD therapy. JG is an excellent example of a motivated HD patient achieving beneficial changes to her body composition from her commitment to sustained exercise.

INNOVATIVE COLLABORATION BETWEEN A DIETETIC INTERNSHIP PROGRAM AND THE MTCRN TO ENHANCE A RENAL DIET EDUCATION TOOL

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In early 2009, the Middle Tennessee Council on Renal Nutrition (MTCRN) decided to update their Basic Renal Diet Education Tool.

This tool is marketed to hospitals and long-term care facilities nationwide as a way for the MTCRN to raise funds. While not intended as a comprehensive guide to the renal diet, its purpose is to provide an overview of diet guidelines for patients new to dialysis.

The tool was brought to a preceptor of the dietetic internship program at VUMC as a possible project for the incoming interns. The project was accepted and assigned to two interns. Following principles for clear health communication, modifications included:

- Using font size, formatting, and space to improve readability
- Rewording phrases to highlight a positive emphasis
- Consolidating two sample menus into one
- Providing diabetic suggestions where appropriate
- Categorizing foods within groups (i.e. chunking) for readability
- Adding color and graphics to improve and clarify the message

The interns presented the revised diet education tool at the October 2009 meeting to the approval of the MTCRN members.

This project is an excellent example of an innovative collaboration between these two groups. The MTCRN provided a basic education piece in need of revision. The interns had the knowledge to utilize marketing concepts, readability guidelines, and computer software tools (Microsoft Publisher) to enhance this basic renal diet education tool.

In the end, all groups, including the patients that will be exposed to this Basic Renal Diet Education Tool, have benefited. This collaboration between the MTCRN renal dietitians and the VUMC dietetic interns has resulted in a comprehensive and attractive diet education tool that offers high readability to its target audience.

EFFECTS OF USING AN INTERACTIVE APPROACH TO PHOSPHORUS EDUCATION

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Education is extremely important for patients to succeed and stay healthy on hemodialysis. They need reinforcement on topics that they have already been exposed to, to promote motivation and continued compliance. Patients in the dialysis unit were experiencing elevated levels of serum phosphorus (P).

The registered dietitian (RD) created a P wheel to aid patients in their reinforcement of P education. The wheel was an education tool by showing not just how much P was in a food item, but also the serving size, calories, protein, potassium, sodium and fat contents of each food. The particular foods that were chosen were foods that are most commonly eaten and asked about by the patients. A small window in the wheel reveals the nutritional facts for each food as you turn the top of the wheel. Each food was categorized in separate sections, grains, dairy, nuts and beans, prepared foods, beverages and desserts. This helped the patient to find what food they were looking for more efficiently. In addition to specific foods, on the back of the wheel there is supplementary P education, such as, alternatives to high P foods, what happens when eating too much P and why P binders are needed and so important to take.

The P wheel was created to help the patients serum P levels fit within goal of 3.5-5.5 mg/dL. Baseline values reported in August 2009, 39% of 115 patients had a serum P of 3.5-5.5 mg/dL. The wheel had been used with the patients for a full month. Each patient was able to look and use the wheel along with teaching from their RD or their patient care tech (PCT). The patients could also engage the RD or PCT in a conversation to ask them questions or specific foods that they could use as an alternative to high phosphorus foods. In September 2009, 55% of patients had a serum P of 3.3-5.5 mg/dL. The percentage of patients that had met goal in September 2009 for serum P since August 2009 had increased by 16%.

Using specialized teaching tools greatly enhances the hemodialysis patients understanding on particular topics. The outcome was not only decreased serum P levels, but also lower levels of calcification, mortality and well being in this population.

NUTRITIONAL STATUS OF PERITONEAL DIALYSIS (PD) PATIENTS AFTER MEDICAL NUTRITION THERAPY AT SINGAPORE GENERAL HOSPITAL PD CENTRE

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This retrospective study assessed the nutritional status of newly initiated PD patients after Medical Nutrition Therapy (MNT) at PD centre.

Patients were recruited from May 2007 to Sep 2009 at Singapore General Hospital (SGH) PD Centre. All patients' dietary protein intake (DPI), high biological value (HBV) protein and serum albumin were assessed and recommendations were given during the initial MNT session. Follow-up session was arranged 6-8 weeks post initial MNT which DPI & HBV intake were reassessed and reinforced. Patients' protein intake, serum albumin and nPNA were recorded.

A total of hundred and forty-nine patients recruited in this study. A hundred and seven patients (71.8%) had shown up at dietitian follow-up session with DPI increased from $44\pm 13\text{g}$ to $48\pm 16\text{g}$, ($p<0.05$); HBV protein increased from 4 to 5 exchanges ($p<0.05$); the percentage of HBV met requirement increased from 68.5% to 79% ($p<0.05$). Although there was an increased in DPI and HBV intake, but it did not correlate with serum albumin ($p=0.12$) or nPNA ($p=0.35$). Ninety-four (63.1%) patients had history of diabetes and the mean HbA1c was $6.5\pm 1.3\%$ post initial MNT. Of this cohort, the mean DPI ($46\pm 16\text{g}$ vs $52\pm 14\text{g}$, $p<0.05$) and HBV protein intake (5 ± 2 exchanges vs 6 ± 2 exchanges, $p<0.05$) were lower than patients without diabetes. There was also an association between history of diabetes and hospitalization incident (OR=1.76, CI 95%).

An increased in DPI and HBV protein intake among new PD patients was seen post MNT. However, the increment did not correlate with serum albumin level and nPNA. Patients with diabetes had an overall lower DPI and the increment of DPI and HBV intake post MNT also shown significantly lower than patients without diabetes. An association was seen between patients with diabetes and hospitalization incident. It could be important to this patient group since lower dietary intake in long term will affect their nutritional status and may lead to higher mortality risk in the future. Therefore, MNT with regular assessment and reinforcement should continue and as part of management to improve nutritional status of all PD patients.

A RARE CASE OF COCAINE INDUCED BILATERAL RENAL INFARCTION

Waqas Ahmed, Madan Gowda, Naseer Khan, Piam Shanesaz, Puneet Bains. Jewish Hospital Cincinnati, OH. Introduction: Cocaine-induced renal damage is well known to be caused by rhabdomyolysis induced ARF & by hypertensive nephro- sclerosis. Cocaine induced renal infarction (CIRI), however, has been rarely reported in the literature and is mostly unilateral. Only two cases of bilateral CIRI have been reported so far, we report the third one. Case Report: A 46 year old male with no significant past medical history except for cocaine abuse presented to ER with complaint of constant bilateral flank pain which worsened with deep inspiration. Review of systems was otherwise unremarkable. Physical examination revealed tachycardia and right upper quadrant tenderness. EKG showed sinus tachycardia. Basic lab investigations were unremarkable with serum Cr 1.2. However D-Dimer and serum LDH were high. Urine drug screen was positive for cocaine. CTPA ruled out PE whereas CT abdomen & pelvis with contrast revealed bilateral renal infarcts. Transthoracic as well as transesophageal echocardiograms did not show any evidence of mural thrombus or vegetations. Abdominal aortogram revealed bilateral patent renal arteries. Further screening tests for hypercoagulability, collagen vascular disorders and lipid profile were within normal limits. Based on this extensive work up diagnosis of cocaine induced renal infarction was made. Patient was started on heparin and then switched to Coumadin. Discussion: Cocaine abuse is an epidemic in US & its toxicity affects multi-systems. The mechanism of action of CIRI is mainly due to cocaine's vasoconstrictive and thrombotic effects. CT with contrast is the initial diagnostic modality while renal angiogram remains the gold standard. There is no consensus on the treatment of CIRI. Due to its non-specific symptoms (abdominal /flank pain, nausea & vomiting, fever etc) diagnosis of CIRI can be missed easily & should be considered in differential diagnosis in cocaine abuse patients who present with these symptoms.

IRON REPLETION DECREASES PLATELET COUNTS (PLT) IN NON-DIALYSIS CKD PATIENTS

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Iron deficiency may produce “relative” thrombocytosis that may contribute to thrombotic complications noted in ESA anemia clinical trials. PLT in > 40,000 MHD pts were found to be inversely related to severity of iron deficiency. Post-hoc analysis of the DRIVE Study noted a reduction in PLT following iron administration (1000 mg over 8 dialysis sessions). We dosed outpatients with 500 or 1000 mg of low molecular weight iron dextran (ID) during 2-4 hr sessions to correct iron deficiency [TSAT < 20%, ferritin <200] and analyzed hematologic responses of Hb, iron indices, and PLT in 132 subjects without intercurrent events who were receiving once-monthly darbepoetin (DA) using our CAMP[®] program. Of these, 86 had complete data for all parameters of interest: 500 mg (N=36) and 1000 mg (N=50). Mean age 69.9 yr; mean Cr 2.3±0.1 mg/dL; mean MDRD-GFR 34.4±1.4 mL/min/1.73 m². Table (mean±sem) shows results over time.

* significant change from basal value, # difference between 2 ID doses.

Parameter	Iron Dose	Time			
		-1 mo	0 (basal)	+1 mo	+2 mo
Hb (g/dL)	500	10.1±0.2	10.0±0.2	10.8±0.3*	10.9±0.3*
	1000	10.3±0.2.	10.4±0.2	11.7±0.2*#	11.7±0.2*#
PLTS (x 1000)	500	265±15	280±16	235±12*	227±10*
	1000	328±19#	328±18#	306±23*#	274±13*#
TSAT (%)	500	18.7±2.0	12.9±1.0	26.8±2.9*	26.9±3.0*
	1000	15.5±1.1	14.0±1.0	24.5±1.9*	25.6±2.0*
Ferritin (ng/mL)	500	52±9	37±5	157±26*	127±30*
	1000	66±8	53±7	237±31*	193±28*
DA dose (mcg/mo)	500	151±17	166±17	170±21*	137±28*
	1000	97±11#	173±14	150±14*	104±20*

MANOVA analysis: iron dose determined magnitude of change from baseline in PLT (< 0.001), DA dose (<0.001), Hb (0.032), and ferritin (0.049) but not TSAT (0.42). PLT decrease was greatest in those with basal counts > 300 K, varied inversely with initial basal PLT (R=0.70,) and was independent of basal ferritin, TSAT, and Hb.

We conclude that correction of iron deficiency lowers PLTS and improves erythropoiesis resulting in higher Hb with lower DA doses. Clinical significance of PLT reduction to other outcomes requires additional study

COMPARISON OF RADIOCEPHALIC FISTULAS (AVF) PLACED IN THE PROXIMAL FOREARM AND IN THE WRIST

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Non-maturation is a common problem with AVFs. In pts with a failed distal radio-cephalic fistula (dRCF) at the wrist or those with vessels unsuitable for a dRCF, a brachio-cephalic AVF is usually placed. Proximal forearm radio-cephalic fistulas (pRCF) may permit a second forearm AVF before proceeding to the upper arm. We retrospectively analyzed a prospective, computerized access database to compare the outcomes of 19 pRCF and 39 dRCF placed during a six-month period. Primary failure was defined as inability to cannulate a fistula reproducibly for dialysis. Cumulative survival was defined as the time from fistula creation to its permanent failure. The baseline characteristics of the two patient groups were similar, except that those with a pRCF were more likely to have had a previous access and less likely to be female. Primary failure (non-maturation) was lower for pRCF than dRCF (32 vs 59%, $P=0.05$). After excluding primary failures, cumulative fistula survival was similar for pRCF and dRCF (92 vs 86% at 1 yr and 74 vs 76% at 2 yr, $p=0.56$).

In conclusion, a pRCF may be an attractive alternative to a brachiocephalic AVF in patients who cannot receive a dRCF. A pRCF has a lower non-maturation rate to that of a dRCF, and a comparable cumulative survival once it is used successfully for dialysis.

TRENDS IN STROKE RATES AMONG HEMODIALYSIS PATIENTS

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Recent trends in hospitalizations for stroke in US hemodialysis (HD) pts have not been reported. Using USRDS data, we examined hosp claims from 1995-2007 to compute rates for stroke using ICD9 codes 430.X -438.X (CVA/TIA). For comparison, we computed overall hospitalization rates for CVD in HD pts, and used a Medicare 5% sample of people aged 65+ to compute CVA/TIA and CVD rates.

CVA/TIA hosp rates in HD pts increased from 1995-1999 and began to decrease in 2002; 20% from 2001 to 2007. CVA/TIA rates in the Medicare 65+ population were generally stable until 2001 and then declined; 24% from 2001 to 2007. Rates of CVD hosps in the HD population increased until 2004 and then began to decline. CVD rates in the Medicare 65+ population increased somewhat from 1995-2001, and then showed a similar pattern of decline in 2002.

The pattern of declining CVA/TIA and CVD hosp rates in the general Medicare population may be associated with improved awareness and control of hypertension, hypercholesterolemia, and declining smoking rates. Among HD patients, the earlier decline in CVA/TIA hosp rates compared to CVD hosp rates is more challenging to explain, and may be due in part to improved management of ultrafiltration and improved treatment of hypertension in these pts, which may have a greater impact on CVA/TIA rates compared to CVD rates in HD patients.

Rates of Hospitalization for CVA/TIA CVD, per 1,000 pt yrs

Year	HD - CVA/TIA	HD - CVD	Medicare Age 65+ CVA/TIA	Medicare Age 65+ CVD
1995	48.4	560.7	19.5	93.9
1996	50.2	585.1	19.8	95.9
1997	52.5	589.9	20.1	98.8
1998	53.7	591.1	19.6	98.3
1999	55.6	603.2	19.5	98.9
2000	55.4	605.1	19.6	100.8
2001	55.5	625.7	19.3	101.3
2002	54.3	626.8	18.1	97.5
2003	51.4	632.1	17.2	95.1
2004	50.9	646.6	16.4	93.7
2005	49.1	630.6	15.5	88.6
2006	46.4	604.8	15.3	87.0
2007	44.2	594.1	14.6	83.1

FEBUXOSTAT IN GOUT: SERUM URATE RESPONSES IN URIC ACID OVERPRODUCERS VS. UNDEREXCRETORS

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A Phase 2 randomized controlled trial in a hyperuricemic gout population assessed the serum urate (sUA) responses in overproducers and underexcretors of uric acid classified by urinary uric acid (uUA). 153 gout subjects were randomized to daily febuxostat (FEB) 40 mg, 80 mg, or 120 mg or placebo (PBO) for 28 days. Participants were male (136), female (17), 88% Caucasian with a mean age of 54 years. Presented here are data from 110 underexcretors (uUA ≤ 800 mg/day; 80%) and 28 overproducers (uUA >800 mg/day; 20%). 15 subjects were excluded due to missing uUA at BL and/or BL sUA measured out of window.

Subjects who achieved sUA ≤ 6.0 mg/dl at Day 28.				
	PBO n /N(%)	FEB 40 mg n /N (%)	FEB 80 mg n (N) (%)	FEB 120 mg n /N (%)
Overproducers	0/7 (0)	5/7 (71)	6/8 (75)	6/6 (100)
Underexcretors	0/28 (0)	14/27 (52)	21/27 (78)	26/28 (93)
Percent reduction in sUA from baseline values (mean)				
Overproducers	-1.8	-29.6	-43.0	-58.3
Underexcretors	-2.2	-38.4	-46.2	-59.2

Urine uric acid mean percent reductions from BL ranged from 43.6 to 46.5% among all FEB groups vs a mean percent increase from BL of 5.9% in the PBO group. Treatment with FEB showed reduction of sUA at all dose levels in both underexcretors and overproducers. Adverse events were similar across groups.

Subjects with hyperuricemia and gout, regardless of classification as overproducer or underexcretor, responded equally well to FEB at all dose levels. Treatment with FEB does not require measurement of baseline urine uric acid excretion.

THE USE OF INTERNET AS A RESOURCE FOR HEALTH INFORMATION AMONG CKD PATIENTS: A CLINIC BASED STUDY

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The Internet is rapidly changing the way patients access health-related information, learn about their illnesses, and make healthcare-related decisions. There is, however, very limited data regarding the use of Internet by Chronic Kidney Disease patients. This is particularly significant considering that this subset of patients may have different characteristics compared to the general patient population.

We compiled a questionnaire to obtain demographic data, determine patients' use of the Internet as a medical information resource, and to determine their experiences and perceptions of the quality and reliability of the information available online. The survey instrument consisted of 33 multiple-choice questions completed voluntarily and anonymously by patients visiting the outpatient nephrology clinic and dialysis center as well as the internal medicine clinic within a tertiary care center. Statistical analyses were performed using SAS software.

A total of 350 questionnaires were completed by 63 internal medicine and 287 CKD patients. The median age in the CKD group was 64 (18-91) compared to 50 (18-94) in the internal medicine (comparison) group. Over half of the CKD patients (56.55%) were retired and only 29% were in the active workforce, compared to 74.58% active workforce in the comparison group. 48.16% of the CKD group had education beyond high school, compared to 83.87% in the comparison group ($p<.0001$). 95.16% of the internal medicine group had access to Internet, compared to only 61.65% in the CKD group ($p<.0001$). 47.85% of the CKD patients, compared to 84.61% in the other group, had an annual household income of more than \$50,000 ($p<.0001$).

In conclusion, this analysis suggests significant differences between the majority of CKD patients and the general patient population. Several factors such as age, level of education, and socioeconomic status appear to contribute to these difference and limit the use of Internet as a viable resource by CKD patients. This is an area which clearly needs further exploration in an effort to make the Internet a better and more accessible resource for CKD patients.

PRIMARY AMYLOIDOSIS PRESENTING AS ACUTE RENAL FAILURE WITH COAGULOPATHY

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A previously healthy 54-year-old man was admitted to the hospital because of nausea and increasing weakness. On physical examination, blood pressure was 100/60 and he was afebrile. Heart and lung exam were normal and the liver was of normal size. There was no peripheral edema. Laboratory studies showed hemoglobin of 8.1 g/dL, BUN of 169 mg/dL, and creatinine level of 12.2 mg/dL. Liver function tests were normal. Coagulation profile showed a prothrombin time (PT) of 47 s (normal: 13-15) and activated partial thromboplastin time (APTT) of 71 s (normal: 24-35). Both kidneys appeared hyperechoic but no hydronephrosis on ultrasound. He was anuric on admission and was started on intermittent hemodialysis. Renal biopsy was not performed due to coagulopathy. Attempt to correct the coagulation abnormality with Vitamin K and fresh frozen plasma was unsuccessful. Further evaluation of the prolonged PT and APTT showed a factor X deficiency. Bone marrow aspiration revealed 3% kappa clonal plasma cells and amyloid deposits. Hospitalization was complicated with gastrointestinal bleed and hemoperitoneum. Recombinant factor VIIa was given but he continued to have uncontrollable bleeding. His condition deteriorated rapidly and died.

Primary amyloidosis presents in a variety of signs and symptoms. One of the common manifestations is nephrotic syndrome. Acute worsening of renal failure is a rare form of presentation. In our case, the presence of acute renal failure and coagulopathy is suspicious of a systemic disease. The recognition of isolated factor X deficiency suggests that the patient may have amyloidosis. The exact mechanism of the association between Factor X deficiency and primary amyloidosis remains unexplained. The diagnosis of primary amyloidosis strongly depends on histological confirmation. In our case, the presence of coagulopathy delayed the diagnosis. This case illustrates a subtle presentation of amyloidosis with associated coagulopathy, making it a diagnostic challenge. Amyloidosis should be considered in the differential diagnosis of unexplained acute renal failure especially in the presence of an isolated factor X deficiency.

SODIUM THIOSULFATE: A NOVEL AND EFFECTIVE TREATMENT FOR CALCIPHYLAXIS

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Calciphylaxis, also known as calcific uremic arteriolopathy (CUA), is a rare condition with dismal prognosis and high mortality rate that mainly affects patients (pts) with ESRD. CUA is characterized by soft tissue calcification and skin ulceration due to calcium deposition in subcutaneous vessels. Case reports in the literature have described successful treatment of CUA with sodium thiosulfate (ST) but these are subject to reporting bias. ST acts as a chelator of calcium from the vessel walls and as an antioxidant to improve endothelial dysfunction.

To determine whether ST is an effective treatment for calciphylaxis, we identified all pts (n = 14) who received ST for CUA between 4/1/03 to 1/1/08 by pharmacy records, and then performed a retrospective review of electronic medical records and/or information from the primary Nephrologist. The primary end point was the number of pts who responded to ST with improvement of lesion stage graded as 1 to 5 (pain, livido reticularis, erythema, central necrosis, and eschar, respectively). CUA risk factors were also assessed. Our results demonstrated that 14 patients received ST. The patients were 49 ± 12 years old, on dialysis 4.5 ± 4.2 yrs, 4 male and 10 female, 4 White and 10 African American. The average (SD) phosphorus, calcium and PTH at the time of ST were 5.6 ± 2.8 mg/dl, 8.3 ± 0.6 mg/dl, 458 ± 909 pg/ml and 3 were on coumadin. The most advanced stage of lesion at the time of ST was pain in 2, livido in 3, erythema in 5, necrosis in 1, and eschar in 3. Overall, 35% (5/14) improved lesion stage, 14% (2/14) did not improve lesion stage, and 14% (2/14) had a cessation in the progression of lesions. However, the pts who did not improve were advanced in their stage (3 or greater) of disease, were on HD for longer periods of time and received fewer doses of ST. Overall, there was a 53.8% mortality rate (7/13 died, status of one pt unknown), all presenting in stage 3 or greater. The average stage of those who died at the start of ST was 3.7, compared to 2.8 in those who survived. We conclude that ST is an effective treatment for calciphylaxis if given in the early stages of disease and for a consistent period of time. Thus, early clinical detection and aggressive intervention are imperative to prevent mortality from this deadly disease.

HYPERTENSION MANAGEMENT IMPROVED FOR IN-CENTER NOCTURNAL DIALYSIS PATIENTS COMPARED TO CONVENTIONAL DIALYSIS PATIENTS

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Longer dialysis sessions have been shown to improve blood pressure (BP) control and decrease the need for anti-hypertension drugs. To confirm this observation in a large dialysis population, we conducted a retrospective study comparing BP management and anti-hypertensive drug use in 418 patients in the 6 months before and after their switch to in-center nocturnal hemodialysis (NHD). NHD consists of 6-8 hr/treatments, 3 times/wk while conventional, in-center hemodialysis (ICHD) is defined as 3-4 hr/treatments, 3 times/wk. The baseline period was defined as the last 6 months on ICHD prior to starting NHD. The NHD period was defined as months 4 through 9 post-conversion allowing for a clinical stabilization period in the first 3 months of NHD. Results are shown in the table below.

	NHD	ICHD	<i>p</i> -value
Post dialysis systolic BP (mmHg)	150.7 ± 1.1	151.5 ± 1.1	<0.001
Post dialysis diastolic BP (mmHg)	81.8 ± 0.6	82.8 ± 0.6	<0.001
Pre-dialysis wt (kg)	101.6	101.4	NS
Post-dialysis wt (kg)	97.7	97.3	NS
Anti-hypertensive drug utilization (%)	80.2% (76.3, 83.6 CI)	84.6 % (81.1, 87.5 CI)	<0.01

Analysis of this large, longitudinal cohort of NHD patients confirmed that increased dialysis time on NHD lowered post-dialysis BP and reduced the need for anti-hypertensive therapy. These results suggest both clinical and economic benefits may be realized from NHD.

EFFECT OF LOW SERUM CREATININE ON MORTALITY/ MORBIDITY IN HOSPITALIZED ELDERLY POPULATION

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Malnourished elderly patients tend to have lower levels of serum creatinine. A recent retrospective analyses on critically ill patient found independent association between low baseline serum creatinine (LBSC) levels and increased hospital mortality. We hypothesized that LBSC would predict similarly poor outcome in hospitalized elderly population admitted to a regular medical floor.

Retrospective medical record review of all hospitalized elderly (age 65 years or over) patients admitted from June 2008 to June 2009. Abstracted data included baseline serum creatinine, weight, height, body mass index, serum albumin, ICU transfer, in-hospital mortality, disposition, and recurrent admissions to the hospital within that year. Low serum creatinine group (LBSCG) was defined as patients with baseline serum creatinine $<0.8\text{mg/dl}$. Patients with normal baseline serum creatinine {NBSCG} ($0.8\text{--}1.2\text{mg/dl}$) served as the comparison group. Patients with serum creatinine $\geq 1.2\text{mg/dl}$ and patients on dialysis were excluded from the study. The study is ongoing.

Preliminary analyses based on 90 patients, 53% of whom had low serum creatinine. The median age for the whole group was 79 years, 66.7% were women and 72.9% were white. 79.2% of the LBSCG were women compared to 52.4% of the NBSCG ($p=0.007$). There was no statistically significant difference in age (LBSCG 80.2 vrs 78.2, $p=0.3$), race (LBSCG 72.9% white vrs 59.5%, $p=0.1$), serum albumin (LBSCG 3.1 vrs 3.2, $p=0.4$) or BMI (LBSCG 24.1 vrs 26.2, $p=0.1$) between the two groups. The in-hospital mortality rates was 6.3% for the LBSCG and 11.9% for the NBSCG ($p=0.3$). 18.4% of the LBSCG required ICU transfer compared to 11.5% for the NBSCG ($p=0.3$). For those discharged alive, 46.7% of the LBSCG were sent to an ECF rather than home compared to 43.2% for the NBSCG.

Our preliminary analysis fails to find statistically significant association between low serum creatinine and hospital outcomes among non-critically ill patients. This is probably related to the small sample size used for the preliminary analyses. The full analyses, based on all 6000 eligible patients, will clarify these findings.

MICROALBUMINURIA AND LACTATE DEHYDROGENASE (LDH) AS PREDICTORS OF KIDNEY INVOLVEMENT IN A PEDIATRIC SICKLE CELL DISEASE (SCD) POPULATION

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Renal failure due to sickle cell nephropathy is seen in 4-20% of adult SCD patients. Duration of disease, severity of anemia and genetic factors are believed to influence the risk of development of renal disease among patients with SCD. The purpose of this project is to determine the prevalence of renal involvement in a SCD population and identify potential risk factors for renal involvement.

Forty patients with SCD between the ages of 5-19 years that were seen within the past year are identified from UMDNJ-RWJMS Pediatric Hematology and Oncology Clinic. Following IRB approval, a retrospective chart review was performed for age, sex, height, body mass index (BMI), serum creatinine and estimated GFR (eGFR) by Schwartz and MDRD formulas, type of SCD, Hb level (total Hb and % HbF), LDH level, reticulocyte count, blood pressure, history of splenectomy, history of hydroxyurea use and history of transfusions to determine clinical correlates for microalbuminuria and proteinuria. All variables are correlated with microalbuminuria and proteinuria by univariate and multivariate regression analysis.

The mean age of the study population was 12 +/- 4.5 years. The prevalence of microalbuminuria and proteinuria was 32 % and 10 % respectively. Univariate analyses revealed a significant correlation between LDH level and microalbuminuria (Pearson $r=0.47$, $p=0.04$) and proteinuria (Pearson $r=0.48$, $p=0.035$). Multivariate analysis revealed a significant correlation between microalbuminuria and LDH level ($p=0.04$) and microalbuminuria and history of splenectomy ($p=0.05$) when controlled for other variables.

In this pediatric SCD population, LDH is found to strongly correlate with microalbuminuria and proteinuria. Further studies are needed to confirm LDH as an early marker for risk of kidney involvement among SCD patients.

A CASE OF PATHOLOGIC POLYARTERITIS NODOSA (PAN) IN THE SETTING OF CLINICAL CALCIPHYLAXIS

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Calciphylaxis, also known as calcific uremic arteriolopathy is a form of small vessel vasculopathy. It is characterized by deposition of calcium and phosphorus in the subcutaneous arterial vessels. We describe a case of a woman who was diagnosed and treated as calciphylaxis though biopsy revealed medium vessel vasculitis.

A 52-year-old woman with end stage renal disease (ESRD) on peritoneal dialysis (PD), status post aortic valve replacement, and on chronic anticoagulation was admitted to our Burn Unit for management of diffuse desquamative skin lesions on the lower extremities. She developed painful violaceous skin lesions on her thighs which rapidly progressed to bullae formation and ulcerations over the entire lower extremities. During the hospital stay, she developed ischemic gangrene of her toes. Laboratory studies revealed iPTH levels of 2389 ng/l, positive antinuclear antibodies and an ESR of 139mm/hr. Skin biopsy showed leukocytoclastic vasculitis. Patient was treated with I.V. Sodium thiosulfate with some improvement in her symptoms. Her skin lesions, however, continued to worsen. A repeat biopsy was also consistent with medium- vessel leukocytoclastic vasculitis with no evidence of calcium deposition. She was started on steroids without clinical improvement. Her hospital course was complicated by atrial fibrillation, upper GI bleed, pneumonia and septic shock. Given her overall poor prognosis, the family elected to withdraw care and she died after a 2-month hospitalization.

Calciphylaxis is typically seen in ESRD patients with risk factors such as female sex, a high $\text{Ca} \times \text{P}$ product, PD, hypoalbuminemia and warfarin use. Our patient had many of these risk factors. Myriad reports in the literature reveal calciphylaxis as the diagnosis in cases initially misdiagnosed as primary small vessel vasculitides. To our knowledge, this is the first case in the literature with clinical findings of calciphylaxis having occurred concomitantly with histologic findings of medium sized vessels infiltrated by neutrophils and leukocytoclasia; all characteristics of a medium vessel vasculitis.

IGA NEPHROPATHY PRESENTING WITH PULMONARY HEMORRHAGE

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IgA nephropathy is the most common cause of primary glomerulonephritis worldwide. The pathogenesis involves mesangial IgA and C3 deposition. Classically, the disease process is limited to the kidney. There have only been 7 reported cases of IgA nephropathy presenting with pulmonary hemorrhage. We present another case of IgA nephropathy associated with pulmonary hemorrhage mimicking Goodpasture's disease.

A 44y/o man from Bangladesh was admitted to the ICU with coryza symptoms and massive hemoptysis of 3 days duration. On examination, he had stable vital signs and diffuse pulmonary rales. UA revealed 3+ blood and 2+ protein with evidence of UTI; creatinine was 0.9, BUN was 19 and CT chest showed diffuse bilateral patchy ground glass infiltrates. Bronchoscopy with BAL was done and revealed diffuse blood in the airways. Cytology, serologies (ANA, ANCA, Anti-GBM, C3, C4, hepatitis A and B) and all cultures were negative. A CT-guided renal biopsy showed IgA nephropathy. Therapy was started with high dose IV Solumedrol with marked improvement of the hemoptysis. Cyclophosphamide therapy was initiated and the patient received 6 monthly cycles with alternate day steroids. He had a dramatic response with cessation of hemorrhage.

Pulmonary hemorrhage is a rare but possible complication of IgA nephropathy. Recognition of this fact is key to prompt diagnosis and appropriate therapy.

BILATERAL EXTRA-ADRENAL PERIRENAL MYELOLIPOMA MIMICKING RETROPERITONEAL HEMORRHAGE

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Extra-adrenal myelolipomas are rare benign tumors. A few case reports involving extra-adrenal sites of myelolipomas have been published. To our knowledge, there is only one published report of bilateral perirenal myelolipomas. A comprehensive report of imaging characteristics of bilateral perirenal myelolipomas mimicking retroperitoneal hemorrhage is presented. 84 y/o male was admitted for uncontrolled diabetes mellitus. Pt had ARF with a creatinine of 2.4 mg/dl which prompted a renal ultrasound. The sonogram showed bilateral renal cysts with a solid mass adjacent to the right kidney. CT scan without IV contrast confirmed bilateral cysts with expansion to bilateral perinephric spaces. ARF resolved with IV fluid administration. Repeat CT scan with IV contrast revealed bilateral perinephric densities compatible with hemorrhage. Pt was on Coumadin for atrial fibrillation and his INR was supratherapeutic. Triple phase CT scan showed perinephric heterogenous expansion with fat and ill defined soft tissue density. MRI with gadolinium confirmed the heterogenous distribution of fatty tissue without enhancement. After comprehensive review of the radiological findings a diagnosis of myelolipoma was made. In view of the patient's wishes, a decision was made to not proceed with a biopsy.

PERITONEAL INFLAMMATION FROM STERILE SILICONE INTRAPERITONEAL CATHETERS IN RODENTS AND HUMANS

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Research to preserve the dialytic barrier has focused on solution biocompatibility, but we have demonstrated that an intraperitoneal catheter (IPC) promotes inflammatory changes when solution is injected. To test the hypothesis that the catheter alone causes inflammation, we surgically implanted sterile silicone catheters in C57BL mice (IPC, n=28) and observed them for 1-5 weeks. Control mice underwent sham operations without IPC (C, n=12). After 1 to 5 weeks, cultures of peritoneum and IPC confirmed sterility. The catheter adherent cell layer (ACL) was examined with immunocytochemistry (ICC) for cell markers and cytokines. Transport experiments were performed to determine the mass transfer coefficients of mannitol (MTCM), albumin (MTCA), and osmotic filtration flux (JOSM). Immunohistochemistry (IHC) of peritoneal tissue was used to measure the sub-mesothelial thickness, vascular density, and cytokines. Correlation with cells from peritoneal washings of patients receiving IPC was made during the 14-day pre-dialysis period. Mouse tissues were compared with 1-way ANOVA and showed significant differences in thickness (μm : IPC, 103.9 ± 8.1 ; C, 18.8 ± 12.4 ; $p < 2 \times 10^{-6}$), vessel density (#vessels/mm: IPC, 61 ± 7.1 ; C, 10.7 ± 10.8 ; $p < 5 \times 10^{-4}$), MTCM ($\mu\text{l/min/cm}^2$: IPC, 4.0 ± 0.3 ; C, 3.0 ± 0.4 ; $p = .05$), MTCA ($\mu\text{l/min/cm}^2$: IPC, 0.17 ± 0.01 ; C, 0.06 ± 0.02 ; $p = .0002$), and JOSM ($\mu\text{l/min/cm}^2$: IPC, $1.7 \pm .08$; C, 1.3 ± 0.1 ; $p < .01$). Significant differences were also found with the duration of exposure in thickness (μm 1 wk, 46 ± 20 ; 5wk, 132 ± 10 ; $p < .003$), angiogenesis (#vessels/mm: 1wk, 15 ± 0.15 ; 5wk, 90 ± 8 ; $p < .001$), and JOSM ($\mu\text{l/min/cm}^2$: 1wk, 1.9 ± 0.2 ; 5wk, $1.5 \pm .08$; $p < .02$). ICC analysis of ACL revealed macrophages, dendritic cells, lymphocytes, myofibroblasts, and mesothelial cells. IHC demonstrated significant ($p < 0.05$) difference from controls for αSMA , TGF β , FGF, VEGF. ICC studies in humans showed increasing PMNs over the first 7 days, (% PMN, day: 7.8, d 1; 32.9, d3; 50.9, d7; 20, d14) and similar patterns of staining to mouse ACL. We therefore conclude that silicone catheters alone result in sterile inflammation, which increases with duration of exposure.

CT SCANS IN ENCAPSULATING PERITONEAL SCLEROSIS (EPS): COMPARING TWO SCORING METHODS

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EPS is a serious complication of PD. Early diagnosis (dx) is critical. Clinical symptoms/signs are nausea, vomiting, anorexia, abdominal pain, and hemoperitoneum. CT imaging is critical for dx. Two scoring systems: Vjim(PDI 2009; 29; 517-22) and Tarzi(CJASN 2008;3; 1702-10) for evaluating CT scans for the dx of EPS have been published. The purpose of this study is to compare these two scoring methods.

The patients (pts) with the clinical dx of EPS were identified from an IRB approved PD registry from 8/1979 to 8/2009. Electronic patient charts were reviewed for CT scans of the abdomen with IV contrast. Available CT scans were scored by a radiologist co-author (NS) using both scoring systems: Vjim, using a +/- system with 6 parameters (≥ 3 positive parameters consistent with EPS dx.); Tarzi, using 6 parameters each scored 0-3 or 0-4 with 22 possible maximum score (a total score ≥ 4 was selected for EPS dx). For the Tarzi system, we also analyzed each parameter using >0 as + and 0 as negative.

There were 8 pts with EPS dx. 5 were women and time on PD was 17-136 mo. All were diagnosed after transfer to HD/transplant. Seven pts had 19 CT scans available for scoring. Fourteen scans performed after the clinical dx of EPS were + by both methods. Five CT scans were performed before the clinical dx of EPS and 4/5 were (-) by both methods; the 5th had a + score by both methods 15 mo before clinical dx. Contrasting Vjim and Tarzi respectively for + scoring for each parameter: peritoneal (perit) thickening 86 vs 93%, perit. calcifications 71 vs 71%, adhesions/tethering 64 vs 64%, bowel obstruction/dilatation 36 vs 43%, loculation 50% for both, bowel thickening 36 vs 43%. Perit. enhancement was only used by Vjim and was 100% + in EPS. The Tarzi score with clinical EPS was median 6 (4-13) and without clinical EPS was 0 (0-4). The median Vjim score was 4 (3-6) for + EPS and median 0 (0-3) for negative EPS.

Both systems are congruent, easy to use, and accurate in dx EPS. The Vjim system is simpler. We recommend that all CT abdomen scans in PD patients having a Vjim score of ≥ 3 or a Tarzi score ≥ 4 should raise suspicion of EPS in the appropriate setting.

HEMOGLOBIN A1C AND 5-YEAR SURVIVAL IN 2,798 CHRONIC PERITONEAL DIALYSIS PATIENTS WITH DIABETES MELLITUS

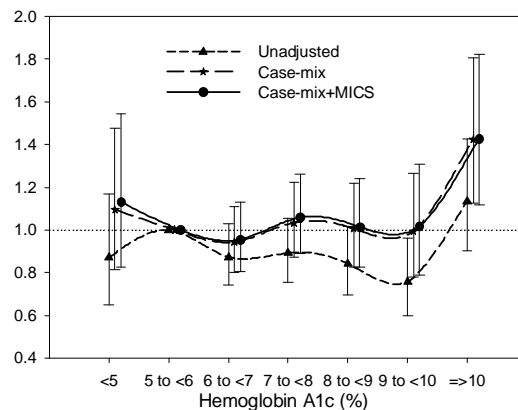
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Background: In chronic peritoneal dialysis (PD) pts, the association of hemoglobin A1c & mortality may be confounded by glucose loading in PD fluid, which may lead to worsened metabolic control in PD.

Methods: We examined a large cohort of all diabetic PD pts who underwent PD treatment for at least 45 days in any Legacy DaVita dialysis clinic over 5 yrs (7/2001-6/2006).

Results: We identified 2,798 diabetic PD pts who had A1c measures during their base calendar quarter; they were 57.4 ± 13.0 yrs old and included 44% women, 20% Blacks & 16% Hispanics. A1c was categorized into 7 groups of $<5\%$, $\geq 10\%$ and 1% increments in-between. A J-shaped trend with significant death hazard ratios (HR) was noted. Taking A1c 5-5.9% as reference, A1c $\geq 10\%$ had a 5-yr death HR (and 95% confidence interval [CI]) of 1.13 (0.90-1.43), 1.43 (1.13-1.81) and 1.43 (1.12-1.82) representing the unadjusted, case-mix and additional malnutrition-inflammation complex syndrome (MICS) adjusted respectively (see figure).



Conclusions: In this large national cohort of diabetic PD patients, a hemoglobin A1c $>10\%$ appears associated with relative risk of death of 1.43 compared to those pts with a A1c of 5-6%. Clinical trials to examine the benefit of tighter glycemic control in PD ps are indicated

THE USE OF INTRAVENOUS SODIUM THIOSULFATE FOR THE TREATMENT OF CALCIPHYLAXIS IN AN ELDERLY PERITONEAL DIALYSIS PATIENT

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Calciphylaxis is a very painful condition which occurs in about 1-4% of patients with end stage renal disease. The use of Sodium Thiosulfate for the treatment of Calciphylaxis in patients undergoing peritoneal dialysis has not been widely studied. Most of the literature focuses on its use in hemodialysis patients.

Case presentation: Mrs. X is an 85 year old lady with end stage renal disease who lives in a rural community in Northern British Columbia. She was started on hemodialysis in May 2006 and switched to continuous cycling peritoneal dialysis (CCPD) 16 months later to limit her travel time. Our patient had an atypical presentation:

- Calcium X Phosphorous product within KDOQI guidelines of <4.4 since starting CCPD
- On no binder therapy
- Low parathyroid hormone
- No diabetes
- BMI = 18.8

Our primary goal for Mrs. X was pain management. Despite the regular use of narcotics, we were unable to achieve adequate pain control. Changing her back to hemodialysis was not an option. Several case reports reviewed the use of Sodium Thiosulfate as a treatment option.

Based on a review of literature and a treatment cost evaluation, it was decided to give 5 g Sodium Thiosulfate IV 3 X/week for an initial 3 months while keeping her on CCPD with one daytime exchange.

After 3 weeks of treatment, she no longer required oxycocet or her fentanyl patch for pain. Her treatment was stopped after 6 months when her wound was healed. Mrs. X's overall quality of life has improved greatly with this treatment.

PERITONEAL DIALYSIS MODALITIES.AND SODIUM REMOVAL. NO DIFFERENCES WITH OPTIMAL USE OF ICODEXTRIN

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Many studies suggest that Continuous Ambulatory Peritoneal Dialysis (CAPD) is more efficient regarding sodium removal than Automated Peritoneal Dialysis (APD), due to longer dwell times and the sodium sieving phenomenon. The aim of the present study was to evaluate the impact of the optimal prescription of these PD modalities (optimal daily ultrafiltrate > 1L/day in anuric patients) on sodium removal.

Forty-six (46) patients aged 52.3 ± 14 years were studied: 26 patients undergoing CAPD by using 7.8 ± 0.5 L daily and 20 patients undergoing APD with a wet (n=13) or dry (n=7) day, by using 18.4 ± 3.9 L daily. Ten (10) patients in each group (all anuric) were using icodextrin solution for the long dwell in order to achieve the adequacy and ultrafiltration (UF) targets.

CAPD patients removed a higher but not statistically significant amount of Na per day (131.7 ± 98.2 mmol) compared with APD patients (79.4 ± 129.2 mmol), although their Kt/V urea was lower (1.48 ± 0.3 vs 2.17 ± 0.33 , $p < 0.05$) and there were no differences on daily UF (1119 ± 533 vs 1005 ± 517 ml) respectively. In both groups, patients using icodextrin for the long dwell presented not statistically different daily sodium removal (CAPD 167.2 ± 136.4 , APD 66 ± 65.5 mmol) compared with patients not using it (CAPD 109.4 ± 59.4 , APD 92.9 ± 174 mmol). In APD, there was no difference in UF between patients using or not using icodextrin, whereas in CAPD, patients using icodextrin had a higher daily UF (1510 ± 395 ml) compared with patients not using it (875 ± 500 ml).

These results indicate that although CAPD is more efficient than APD (with a dry day) regarding sodium removal, optimal prescriptions targeting at high daily UF rates and decent solute clearances (APD with a wet day) can diminish this difference, especially in anuric PD patients. In both modalities icodextrin use as an adjuvant for higher daily UF rates, can increase solute clearances and sodium removal.

SALVAGE OF A TOTALLY OCCLUDED PERITONEAL DIALYSIS CATHETER BY LAPAROSCOPIC SURGERY

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The Peritoneal Dialysis (PD) catheter is the PD patient's lifeline. Dislocation of the tip of the catheter, intraluminal fibrin obstruction, and omental wrapping are the most common aetiologies of PD catheter malfunction. Two-way (outflow/inflow) obstruction of the PD catheter is one of the most serious complications of PD and it is usually observed during the first weeks after catheter implantation.

A 44 year old male was started on hemodialysis (HD) due to uremic symptoms via a right jugular vein HD catheter. One month later he decided to choose PD as maintenance therapy and a PD catheter was successfully inserted by peritoneoscopy. The patient postponed the start of the PD training for almost one month due to some personal issues and the catheter was flushed once weekly with good flows. During the start of a regular weekly flushing, bloody effluent was noticed, whereas the patient had no other symptoms. Heparin was added in the PD solutions and the patient was undergoing daily flushing. On day 4 a two-way catheter obstruction was noted.

Abdominal x rays showed that the tip of the catheter was in the right position in the pelvis without any other abnormalities. Several attempts to restore its patency in the PD unit were unsuccessful. Direct fluoroscopic evaluation and several attempts with the use of guidewires and infusions of urokinase had also no effect.

Laparoscopy revealed a large intraluminal fibrin clot that was occluding the catheter's holes. The clots were dislodged by milking the PD catheter with atraumatic laparoscopic forceps and flushing with a PD solution under pressure. After several attempts, a good patency of the catheter was achieved in the surgical room and the patient had an uncomplicated recovery. He was started on CAPD one week later and he has been on the method for almost one year with no further problem.

Laparoscopic surgery is the ideal method in order to fully diagnose and correct the malfunctioning PD catheter.

**CHEMICAL PERITONITIS FOLLOWING
INTRAPERITONEAL (IP) SODIUM THIOSULPHATE (STS)
FOR TREATMENT OF CALCIPHYLAXIS.**

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We present a case of patient on peritoneal dialysis (PD) who developed calciphylaxis and was subsequently treated with IP STS. She developed chemical peritonitis secondary to STS.

A 82 year-old white female on peritoneal dialysis was admitted with complaints of 1-2 weeks of left lower leg pain with purple discoloration and ulceration. Examination of her leg revealed an ulcerated region suggestive of calciphylaxis. A skin biopsy confirmed the diagnosis of calciphylaxis.

She was treated with IP STS (25 gms/2L of PD fluid). The following day she developed abdominal pain with cloudy dialysate fluid. Analysis of the fluid showed a WCC: 4,500 /mm³, 92.0 % neutrophils, Protein: <0.6 g/dL and LDH: 90 unit/L. Fluid and blood culture were negative. She was diagnosed with chemical peritonitis secondary to STS.

Calciphylaxis has been reported since 1960's¹. Ciccone *et al.*² for the first time described the role of STS in treating this condition. Several case reports since have confirmed the role of STS in treating calciphylaxis. However, all the case reported was with use of intravenous STS. Mataic *et al.*³ for the first time demonstrated the successful use of IP STS. Chemical peritonitis may be a serious side effect which may potentially limit the IP route of STS administration and till further studies are available the role of IP STS remains questionable.

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PERITONEAL DIALYSIS: AN OLD THERAPY WITH SERIOUS COMPLICATIONS

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Background: Continuous ambulatory peritoneal dialysis (CAPD) is a renal replacement therapy modality used in ESRD patients. Like hemodialysis, it has many acute and long-term complications including infections, dialysate leakage, and hernias. Purpose: to report a fatal case of sclerosing encapsulating peritonitis (SEP) presenting as acute intestinal obstruction. Clinical vignette: A 61-year-old man with history of HTN, DM, and ESRD for which he was on CAPD for the last 4 years presented to the ER with acute onset abdominal pain that started few hours prior to presentation. The pain was described as dull, constant, and diffuse. He denied any other associated symptoms. His physical exam was remarkable for a distended abdomen that was tender all over. Abdominal CT scan was performed showing diffuse dilatation of the small bowel, moderate amount of ascites, and thickening of the peritoneum without calcifications, suggestive of sclerosing peritonitis. Sooner, the patient condition started to deteriorate with worsening in his mental status as well as developing intractable vomiting so he was intubated for airway protection. He then underwent diagnostic laparoscopy revealing extensive adhesions in the entire small bowel in addition to firm sclerosis of the entire peritoneum, suggestive of SEP. Several biopsies were taken from the peritoneum and partial lysis of the adhesions was performed. Post operatively, the patient's condition continued to deteriorate and he died on the third day post surgery. Pathologic results returned confirming the diagnosis of SEP. Conclusion: SEP is a rare entity that can be primary or secondary to many conditions including CAPD. It is characterized by encasement of the intestines. The diagnosis is usually made by direct visualization of the sclerotic peritoneum as well as histologic examination. The treatment is surgical aiming for lysis of the adhesions but the prognosis of secondary SEP is generally poor due to the severity and complexity of the adhesions

TUBERCULOUS PERITONITIS FOLLOWING INTRAVESICAL BCG THERAPY IN A PATIENT WITH PERITONEAL DIALYSIS

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Nabil Zaki,¹ Fadi S Rzouq².¹: McLaren Regional Medical Center,
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Background: intravesical Bacillus calmette-Guerin (BCG) therapy is commonly used to treat superficial bladder cancer. Many local and systemic complications have been reported before. Aim: To report a case of TB peritonitis post intravesical BCG administration. Clinical vignette: A 54-years-old man with ESRD who has been maintained on peritoneal dialysis was diagnosed with a low-grade papillary urothelial carcinoma of the bladder. One week after his last course of intravesical BCG therapy, he presented to the ER with abdominal distention and diffuse dull pain associated with nausea and vomiting. Upon presentation, his physical examination was remarkable for extensive ascites with diffuse tenderness. Laboratory work-up was relevant for WBC count of 15700 cells/ml (60% neutrophils, 33% lymphocytes) and serum creatinine of 7.6 mg/dl. Paracentesis was performed showing a WBC count of 1280/mm³ (32% neutrophils, 74% lymphocytes), total protein of 4.5 g/dl, and albumin of 2 g/dl. Based on the ascitic fluid analysis, TB peritonitis was highly suspected. Ascitic fluid and peritoneal catheter were sent for gram stain and culture and anti-TB regimen was started (isoniazid, rifampin, and ethambutol) in addition to IV antibiotic. Three weeks after, all cultures returned back negative except for the catheter tip which grew Mycobacterium Bovis and confirmed the diagnosis of TB peritonitis as a complication of the BCG therapy. The patient received the anti-TB therapy for nine months and was switched to hemodialysis with no further complications. Ascites resolved in 2 months after starting the TB therapy and repeat TB cultures were negative by the end of the therapy. Conclusion: TB peritonitis is a rare complication of BCG therapy but it can be curable with early diagnosis. (Abstract ID # 373)

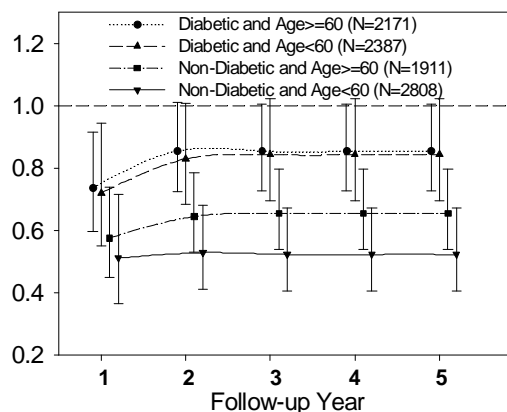
COMPARISON OF MORTALITY OF INCIDENT PERITONEAL DIALYSIS (PD) AND HEMODIALYSIS (HD) PATIENTS BY AGE AND DIABETES IN A NATIONAL COHORT

Rulin Hechter, Kamyar Kalantar-Zadeh, Csaba P Kovesdy, Jennie Jing, Allen R Nissenson, Rajnish Mehrotra. *Harold Simmons Center, Harbor-UCLA, Torrance, CA; VA Salem; DaVita, El Segundo, CA.*

Background: Comparing patient (pt) survival between HD and PD may relate to four statistical interactions including dialysis vintage, age, diabetic status, and co-morbid conditions. We examined a large national cohort that enables these subgroup analyses.

Methods: We compared the death risk of PD vs. HD in DaVita pts over 5 yrs (7/2001-6/2006) after 1:1 matching via “propensity score”, created using logistic regression that included age, gender, race, diabetes, dialysis vintage, calendar quarter, and location (State) to predict the probability that a pt would be assigned to PD vs. HD. We then separately examined survival of pts who were on PD or HD for 3 to 24 mo (n=9,277) by age (≥ 60 vs. < 60 yrs old) and diabetic status.

Results: Cox models, adjusted for case-mix and laboratory measures of “malnutrition-inflammation-cachexia syndrome” (MICS) showed that PD pts had consistent survival superiority compared to HD in all subgroups, although the said survival advantage was somewhat mitigated among the diabetic PD pts (see Figure).



Conclusions: Compared to incident HD pts, incident PD pts, esp. those who were non-diabetic, show a robust and consistent survival advantage up to 5 yrs. Additional studies to identify subgroups that benefit the most from modality selection are indicated.

PERITONEAL TB IN A PERITONEAL DIALYSIS IMMIGRANT PATIENT- A DIAGNOSIS TO CONSIDER

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TB is rare in the USA with an incidence of 4.2 cases per 100,000. It is 9.5 times more common in foreign born than US born persons.

Impaired immune function due to CKD is associated with an increased incidence of TB. Among patients with ESRD, TB carries a 10 to 25 fold greater incident rate than in the general population. Worldwide, peritoneal TB accounts for 37% of tuberculosis cases in patients on CAPD.

Mr. C. is a 66 yr old man from Ecuador with history of DM, HTN, CAD, and on peritoneal dialysis for the past 2 years. Over the last 2 months Mr. C. had presented to the hospital because of recurrent episodes of abdominal pain and diagnosed as culture negative peritonitis. On his last admission PD fluid culture grew *Sphingomonas paucimobilis* and was treated with sulfa-trimethoprim in consultation with infectious disease. There was incomplete resolution of pt's symptoms and dialysate white cell count. CT scan revealed a multi-septated 2.8 x 3 cm lesion in the posterior and inferior lobe of the right liver. A positive PPD was confirmed by the quantiferon-TB gold serum test and the patient had a negative CXR. CT guided biopsy of the liver lesion revealed a necrotizing granuloma, negative for acid fast organisms and fungi. Because of his recurrent symptoms and unwillingness to continue with peritoneal dialysis, hemodialysis was initiated. *Mycobacterium TB* complex was cultured in PD fluid after 2 weeks incubation. There were no acid fast organisms seen on staining of all PD fluid specimens including the specimen on which growth was detected. Mr. C is presently being treated with 4 drug anti-TB regimen.

We use this case report to highlight that despite decreasing incidence of TB in the USA, foreigners on PD are at increased risk of TB.

Clinical presentation is insidious often mimicking uremia. Since the patient's prognosis and survival depends on early diagnosis and prompt treatment, physicians should have a high index of suspicion in foreign-born PD patients presenting with non specific symptoms and culture negative peritonitis.

CHRONIC ABDOMINAL PAIN IN A PATIENT ON MAINTENANCE PERITONEAL DIALYSIS: A CASE OF ENCAPSULATING PERITONEAL SCLEROSIS

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Encapsulating peritoneal sclerosis (EPS) is a rare but life-threatening complication of peritoneal dialysis. A major limiting step to successful therapy is the delay in recognizing the diagnosis.

Here in we present a 57 yrs old Caucasian female with history of ESRD on Peritoneal dialysis (PD) (etiology: vesicoureteral reflux) with 4 episodes of peritonitis over 12 years with progressive increase in dialysate to plasma creatinine ratio. She had symptoms of intermittent nausea, vomiting and diarrhea associated with abdominal pain for over 6 months. All infectious etiologies were excluded including infectious peritonitis on several occasions. She was treated symptomatically with antimotility agents with minimal relief and was referred to our center. On examination she had mild diffuse abdominal tenderness with no guarding or rebound. PD catheter exit site showed no discharge. Contrast CT of abdomen and pelvis showed diffuse wall thickening involving entire colon. She was transitioned to hemodialysis and was started on high dose steroid. Subsequent CT of abdomen and pelvis showed increased peritoneal thickening with calcifications and tethering of the intestine consistent with diagnosis of EPS. Surgical stripping of the encapsulating membrane and enterolysis was not attempted due to high operative mortality. Patient was maintained on total parental nutrition with adequate nutritional support, but rapidly deteriorated and died of septic shock. The incidence of EPS at 5 and 8 years varies from 6.4% and 19.4% in Australia to 2.1% and 5.9% in Japan. The amount of glucose exposure and the occurrence of peritonitis may play a role in EPS but casual relationship has not been established. In the early stages of EPS treatment with steroids was shown to be partially effective. There are anecdotal reports of beneficial use of tamoxifen, a non steroidal antiestrogenic agent. The development of EPS should be considered a potential risk after 3-5 years on PD but no benefit for early screening is recommended using CT scanning. A heightened index of suspicion for EPS is required when

a constellation of clinical findings is present particularly in patient on prolonged PD.

SYSTEMATIC DIFFERENCES AMONG PATIENTS INITIATED ON HOME HEMODIALYSIS AND PERITONEAL DIALYSIS: THE FALLACY OF POTENTIAL COMPETITION?

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Background: The adoption of home-based dialysis therapies is growing internationally. There is a possibility that competition may exist between peritoneal dialysis (PD) and home hemodialysis (HHD) for their respective growth.

Methods: Clinical demographics of patients initiating PD and HHD from 2004 to 2008 in our center were abstracted using institutional electronic records. We compared clinical demographics, laboratory data and process of care to describe potential factors leading to patients choosing specific home-based dialysis renal replacement therapies (RRT).

Results: Between 2004 and 2008, 236 patients initiated home dialysis therapy in our center: 153 patients to PD and 83 patients to HHD. PD and HHD patients differed in age (PD 62 +/- 16 vs HHD 46 +/- 13 years; $p < 0.001$) and gender distribution (PD 57% vs HHD 70% male; $p = 0.05$). A higher proportion of PD patients had diabetes and hypertension as the primary cause of their ESRD. In contrast, there were more patients with glomerulonephritis among the HHD cohort. Cardiovascular and peripheral vascular diseases were more common among patients on PD. HHD patients had higher ESRD vintage (PD 0.34 +/- 0.69 and HHD 4.8 +/- 6.8 years on therapy; $p = 0.002$). The proportion of patients receiving chronic kidney disease care was higher among PD starters (PD 86% vs HHD 65%; $p < 0.001$). Sixteen percent of PD patients and 9% of HHD patients initiated their home-based RRT after an acute hospitalization without prior modality education.

Conclusion: There are systematic differences between patients initiated on PD and HHD. Our data reaffirm that modality selection is a complex process. Patients on the two home therapies differ demographically and arrive through different routes. This finding suggests that the two home-based modalities are not in competition.

HEPCIDIN IN THE CHRONIC KIDNEY DISEASE IN CHILDREN (CKiD) COHORT STUDY

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Higher serum hepcidin (Hep) is associated with decreased iron availability, and has been observed in adult CKD. The objective of this study was to determine serum Hep levels and their relationship to ferritin, hemoglobin (Hb), anemia, and inflammation in the CKiD observational cohort study. Cross-sectional analysis in 124 children enrolled in CKiD was performed. GFR was measured by plasma iothexol disappearance. Anemia was defined as Hb less than the 5th %ile for age and sex OR use of an ESA. Median age was 13 yrs, 60% male, median iGFR 43 ml/min/1.73m². Median Hep level was 64 (36, 157) ng/mL, and median Hb 12.7 (11.7, 13.8) g/dL. There was a significant negative correlation between Hep and iGFR ($r^2=7\%$, $p<0.05$) and a significantly positive correlation between Hep and ferritin (ng/mL) ($r^2=46\%$, $p<0.05$). No correlation between Hep and Hb (g/dL) was seen. Hep was higher among anemic patients (median 93 [42, 203] ng/mL vs. 55 [29, 104] ng/mL, $p=0.02$ by rank sum test). Median Hep did differ by wrCRP; 76.9 ng/mL among pts with wrCRP ≤ 0.3 mg/L, vs. 54.6 ng/mL for wrCRP 0.3-3.0 mg/L and 44.6 ng/mL for wrCRP > 3.0 mg/L (p -value for trend = 0.03). There was no significant correlation between Hep and IL-6. iGFR is an independent predictor of Hep level (each 20% decrease in GFR is associated with 15% [95% CI 3%-29%] increase in Hep, $p=0.01$). Hep is correlated with GFR and ferritin in this cohort. We observed higher serum Hep in anemic children. The correlation between Hep and wrCRP was in the opposite direction expected if Hep were associated with inflammation only. Higher Hep in pediatric CKD may be related to decreased GFR and less driven by inflammation.

ACTIVATION OF PROXIMAL TUBULE SPHINGOSINE 1-PHOSPHATE RECEPTOR 1 PROTECTS KIDNEYS FROM ISCHEMIA REPERFUSION INJURY INDEPENDENT OF LYMPHOCYTES

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Sphingosine 1-phosphate (S1P) receptor agonists reduce kidney ischemia-reperfusion injury (IRI), an effect presumed to be mediated by S1P₁ receptor (S1P₁R)-induced lymphopenia. To test the hypothesis that S1P₁R activation may mediate renal protection from IRI independent of lymphocytes, we investigated the effect of S1P₁R agonists on kidney IRI in Rag-1 KO mice, which lack T and B lymphocytes. Following IRI, less injury was observed in Rag-1 KO mice compared to WT mice as determined by plasma creatinine levels and histology. Administration of FTY720, or the selective S1P₁R agonist, SEW2871, further reduced injury in Rag-1 KO mice as well as WT mice. In mouse proximal tubule cells grown in culture, lipopolysaccharide (LPS) or hypoxia/reoxygenation-induced apoptosis was significantly reduced by SEW2871 supporting a direct protective effect of S1P₁R agonists on proximal tubule cells via MAP kinase and/or Akt pathways. To determine the role of proximal tubule S1P₁Rs *in vivo* in reducing IRI we used *PEPCK-CreSIP₁^{fl/wt}* and *PEPCK-CreSIP₁^{fl/fl}* mice that are deficient in proximal tubule S1P₁Rs. *PEPCK-CreSIP₁^{fl/wt}* and *PEPCK-CreSIP₁^{fl/fl}* mice had a significant rise in plasma creatinine after IRI to a level greater than control (*PEPCK-Cre*) mice. Furthermore, SEW2871 was not protective in the *PEPCK-CreSIP₁^{fl/wt}* and *PEPCK-CreSIP₁^{fl/fl}* mice after IRI. These results suggest that endogenous S1P₁Rs are necessary for stress-induced cell survival and S1P₁R agonists mediate kidney tissue protection at least in part through a direct effect on proximal tubule S1P₁Rs.

ERGOCALCIFEROL INCREASES CIRCULATING LEVELS OF HUMAN CATHELICIDIN (hCAP18)

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Human cathelicidin (hCAP18) is a potent antimicrobial peptide that supports innate immunity. *In vitro* studies show that its expression is regulated by vitamin D. We hypothesized that treatment of vitamin D deficient subjects with ergocalciferol would increase circulating hCAP18. We screened healthy subjects (n=60) without known kidney disease or active infection for vitamin D deficiency. Subjects with 25-hydroxyvitamin D levels below 30 ng/ml were treated with oral ergocalciferol, 50,000 IU every other day for a total 5 doses. Calcium, 25-hydroxyvitamin D, and hCAP18 levels were measured before and after treatment. A total of 25 subjects underwent repletion. Cathelicidin levels were positively associated with 25-hydroxyvitamin D levels in a linear manner ($r=0.28$, $p=0.036$). Following treatment with ergocalciferol, mean 25-hydroxyvitamin D levels rose 25 ng/ml and mean hCAP18 levels rose 26 ng/ml. In multivariable analysis adjusting for age, sex, race, and baseline renal function, increase in 25-hydroxyvitamin D levels were the sole independent predictor of change in hCAP18 ($p=0.02$). This relationship was strongest among patients under 50 years. These findings suggest that circulating levels of hCAP18 in humans are regulated by 25-hydroxyvitamin D and can be increased by vitamin D supplementation.

A NOVEL CENTRAL MECHANISM IN UREMIC BONE DISEASE

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Patients with chronic kidney disease have elevated leptin levels and bone disease. We tested whether leptin signaling, via the hypothalamic melanocortin system, is an important cause of uremic bone disease. We performed nephrectomy (N) or sham operation (S) in c57BL/6J wild-type (WT), leptin-deficient (ob/ob), and melanocortin receptor 4 null (MC4-RKO) mice. Additional WT-N mice were treated with agouti-related (AgRP), an antagonist of MC4-R, or vehicle (V). WT-N mice were fed ad libitum while other mice were pair-fed with WT-N mice. Whole body composition was assessed by DEXA. Blood chemistry was analyzed. Excised left femoral composition was determined by DEXA and X-ray microtomographic scanning and femoral strength was assessed by 3-point failure test. Architecture of right femoral bone was analyzed by μ CT. N mice were uremic. WT-N mice displayed a classic uremic bone phenotype characterized by changes in femoral composition, decreased femoral length, bone mineral content, bone mineral density, and failure load when compared to WT-S mice. Bone architectural parameters such as femoral volume, cortical area and thickness were significantly reduced in WT-N than WT-S mice. Skeletal integrity was maintained despite nephrectomy in mouse strains with impaired leptin or melanocortin signaling (ob/ob-N, MC4-RKO-N and WT-N mice given AgRP). Our results showed that leptin is an important cause of uremic bone disease via signaling through its receptor. These findings may have significant clinical and pharmacotherapeutic implications.

MECHANISMS OF EPITHELIAL CELLS INJURY IN DIABETES: ROLE OF SIGNALING PATHWAYS MEDIATED BY NADPH OXIDASES

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Glomerular epithelial cells or podocytes play a critical role in maintaining the structure and function of the glomerular filtration barrier. In diabetes, podocyte injury contributes to increased urinary albumin excretion (albuminuria), one of the most important prognostic risk factors for kidney disease progression. The aim of this project is to understand the mechanisms by which diabetes causes podocyte injury. We have recently shown (EID et al. Diabetes 2009) that high glucose (HG)/hyperglucemia-induced podocyte loss, together with increased generation of reactive oxygen species (ROS) derived from the sequential activation of 2 family of oxidants-generating enzymes, cytochrome P450 and NADPH oxidases. Inhibition of selected cytochrome P450 isoforms (CYP 4A) prevented podocyte injury and reduced proteinuria in OVE26 mice, a mice model of type 1 diabetes. The mechanism(s) by which glucose activates NADPH oxidases and result in podocyte injury and proteinuria are not known. We are currently investigating the role of the energy sensor AMP-activated protein kinase (AMPK). Our data indicate that exposure of mouse podocytes to HG inactivates AMPK. This is associated with increase in Nox1 and Nox4 mRNA and protein expression and activation of NAD(P)H dependent superoxide anion generation. HG also results in increased expression of p53 and apoptosis. The effects of HG on NADPH oxidase activity, Nox mRNAs and protein expression and apoptosis are blocked by AICAR, an AMPK activator. Nox1 and Nox4 siRNAs mimic the effect of AICAR and significantly reduce p53 expression and podocyte apoptosis. In isolated glomeruli of OVE26 mice, there is an inactivation of AMPK accompanied by an increase in the expression and activity of NOX oxidases and upregulation of p53. These changes are reversed by treatment of OVE26 mice with AICAR. Moreover, Treatment of OVE26 mice with AICAR decreased podocytes loss and ameliorates albuminuria. These data indicate a critical role of AMPK in the regulation of NADPH oxidases and podocyte apoptosis. Our work may identify novel cellular mechanism of kidney injury and set the stage to explore new therapeutic approaches to treat diabetic nephropathy.

ROLE OF COFILIN IN PODOCYTE ACTIN DYNAMICS

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We report that slit diaphragm junctional protein Nephrin recruits and regulates the activity of cofilin1 (Cfl), a highly conserved, ubiquitously expressed protein involved in actin filament severing/de-polymerization and recycling of actin monomers. As podocytes are highly polarized cells and develop unique actin rich processes, both during development and injury high turnovers of actin and polarized actin dynamics is required. We speculated a role for Cfl1 and regulation of its activity by the Nephrin. Activation of Nephrin in cell culture studies lead to dephosphorylation of Cfl1 on its serine 3 residue resulting in activation of Cfl1. Using a previously described cultured podocyte model of Nephrin activation (induced clustering of CD16/7-Nephrin chimeric protein and various mutants) We corroborated that nephrin signals to Cfl1 in podocytes by mapping cofilin pathway signaling intermediaries. Cfl1 dephosphorylation required p13 kinase activity as it was attenuated either with p13K inhibitors or by introducing nephrin mutations at its p85- p13K interaction sites. We were further able to show dephosphorylation of slingshot (SSH1) a phosphatase described to dephosphorylate Cfl1 is responsible for the Nephrin mediated Cfl1 dephosphorylation. To study this further we also developed a podocyte specific conditional Cfl1 null mouse. Surprisingly we did not observe a podocyte developmental abnormality but mice developed proteinuria by 3 months of age and foot process spreading by 8 months.

Overall, these results provide evidence that cofilin is essential for podocyte structure and function. We speculate/propose that nephrin mediated cofilin activation establishes gradients of cofilin activity necessary for polarized actin dynamics required both during podocyte development and injury.

POLYCYSTINS ARE REQUIRED FOR ENDOTHELIAL CELL MORPHOGENESIS

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The purpose of this study is to investigate the role of polycystins during morphogenetic changes of endothelial cells (EC). Autosomal dominant polycystic kidney disease is a multisystemic disorder caused by defects in the genes encoding polycystin-1 (PC1) and polycystin-2 (PC2). Although the disease is characterized by the formation and enlargement of renal and extrarenal cysts, the leading cause of death is due to cardiovascular complications of unknown pathogenesis.

To study the role of polycystins in the endothelium we have first determined the subcellular localization of PC1 and PC2 in HUVEC by immunoconfocal microscopy, subsequently we are expressing PC1 and PC2 RNAi in HUVECs using lentiviral vectors. HUVEC showing low levels of PC1 and PC2 were analyzed *in vitro* vasculogenesis and angiogenesis assay. PC2 concentrates to the centrosome at interphase and during all the phases of the cell cycle. PC2 also localizes to the primary cilium, as we have previously shown for PC1, in 5% of ciliated HUVEC. In addition, PC2 localizes at the cytoplasmic bridge, the mid-body region, during telophase. At the initial phase of the mid-body formation, PC-2 localizes between the two bundles of microtubules derived from the mitotic spindle. Later on during cytokinesis, PC2 mostly overlaps the plus-end extremities of the furrow microtubules. Finally, PC2 no longer concentrates to the mid-body in cells nearing the end of cytokinesis. PC1 also localized to the mid-body but only partially overlaps with PC2. Knockdown of PC1 mediated by PC1-RNAi lentivector inhibits the formation of capillaries *in vitro*. We conclude that PC1 is required for endothelial cell morphogenesis.

THE KLK-1 DNA PROMOTER METHYLATION PILOT STUDY IN ACUTE KIDNEY INJURY

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Subjects with renal complications of transplantation (acute tubular necrosis, or acute rejection) excreted less kallikrein than the subjects without such complications. Kidney repair after injury is a recapitulation of normal morphogenesis. We hypothesized that changes in urine epigenetics could be a biomarker approach during the acute kidney injury. We examined the blood and urine DNA for aberrant methylation of the promoter KLK-1 gene and LINE-1 elements by pyrosequencing from 17 acute kidney injury patients on the day of study entry and 38 healthy controls. Results were compared with clinical data and the urine activity of the kallikrein. Global methylation examined by the LINE-1 was high in both blood and urine DNA (mean: 73.67 ± 0.41 , 69.29 ± 1.05). It was also high in both health and disease (mean: 69.53 ± 1.54 , 69.29 ± 1.05 ; $p = 0.89$). Promotor KLK-1 specific methylation was high in blood (mean: 70.32 ± 2.27). But, it was lower in urine (mean: 40.95 ± 7.06). And it trended lower in the AKI patients than controls (mean: 40.95 ± 7.06 , 30.35 ± 5.88 ; $p = 0.26$). Unexpectedly it turned out that our AKI subjects have very high kallikrein excretion, about 10 times higher than that of controls (mean: $6132.89 \pm 2302\text{mU/L}$, $623.03 \pm 88.15\text{mU/L}$; $p = 0.02$). The promoter KLK-1 DNA methylation study confirmed that this gene is the kidney specific expressed one because it was lower in urine than blood. Its trend to be lower in disease suggests that the KLK-1 has a potentially pathogenic role in the development of the Acute Kidney Injury.

LIPOPOLYSACCHARIDE INDUCED AUTOPHAGY IS CYTOPROTECTIVE IN RENAL TUBULAR EPITHELIAL CELLS

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BACKGROUND: Autophagy often promotes cell survival during conditions of stress. We studied whether LPS induces autophagy in RTEC and whether autophagy protects RTEC from LPS-induced cell death.

METHODS: Autophagy was studied in human RTEC exposed to LPS with and without inhibitors of autophagy *in vitro* and in mice injected with LPS *in vivo*. Autophagy was detected by western blotting for lipidated LC3 (LC3II), visualization of autophagolysosomes by electron microscopy, and immunocytochemistry for LC3.

RESULTS: LPS induced a dose-dependent increase in autophagy in RTEC *in vitro* as demonstrated by the appearance of tertiary lysosomes and LC3-containing puncta. In addition, incubation of RTEC with LPS and injection of mice with LPS induced accumulation of LC3II in cellular and kidney lysates, respectively. Inhibition of autophagy by incubation of RTEC with 3MA sensitized cells to apoptosis as detected by cleavage of PARP-1.

CONCLUSION: LPS induces autophagy in RTEC and inhibition of autophagy increases LPS-induced cytotoxicity. Autophagy is therefore a critical cytoprotective process in RTEC in the setting of LPS exposure and may be an important mechanism of defense against sepsis-induced AKI.

LOWER GENERATION OF UREMIC SOLUTES P-CRESOL SULFATE AND INDOXYL SULFATE WITH A VEGETARIAN DIET

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The uremic solutes p-cresol sulfate (PCS) and indoxyl sulfate (IS) are generated by colon bacteria acting on food components that escape absorption in the small bowel. The production of these potentially important uremic toxins may thus be influenced by the diet. This study compared the excretion rates of PCS and IS in vegetarians and subjects consuming an unrestricted diet.

Urinary excretion of PCS and IS were measured 13 healthy vegetarian subjects and 11 healthy subjects consuming an unrestricted diet during each of two study periods spread one month apart. Subjects recorded food intake over four days and collected urine over the final two days of each period.

Measurements during the first period showed that average PCS excretion was 60% lower and average IS excretion 55% lower in vegetarian subjects compared to unrestricted subjects. Measurements during the second period confirmed the lower PCS and IS production rates in vegetarian subjects and showed that the excretion rates were well correlated between the first and second periods. Food records showed that vegetarians had an average 79% higher intake of dietary fiber, and 30% higher carbohydrate intake, and an average 25% lower intake of protein. PCS and IS excretions correlated inversely with dietary fiber intake.

Among healthy subjects, PCS and IS production rates are markedly lower in vegetarians than those consuming an unrestricted diet. Plasma levels of these potential uremic toxins could be reduced if this dietary effect could be replicated in dialysis patients.

p120 CATENIN REGULATES EPITHELIAL TUBULOGENESIS IN PROXIMAL TUBULES

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In the kidney, defects in the development or maintenance of tubule diameter or length lead to polycystic kidney disease, which is one of the most common genetic disorders in humans. Loss of p120 catenin (p120ctn) in the renal mesenchyme of mice prior to tubule formation leads to shortened, cystic proximal tubules without disrupting normal nephron patterning. In contrast, loss of p120ctn from ureteric bud and its derivatives did not display this phenotype, suggesting a unique role for p120ctn in the mesenchymal to epithelial transition and tubulogenesis of proximal tubules. Additionally, there is an adhesive defect, with luminal shedding of tubular cells and mild cytoskeletal alterations. Classical cadherins, as well as α - and β -catenin, are significantly reduced in level, however, loss of p120ctn had no effect on cadherin switching: the cellular distribution of individual cadherins was maintained. Furthermore, through three-dimensional cell culture data, we find that p120ctn knockdown also results in enlarged cysts with adhesive defects, increased cell height, and reduced cadherin levels. Together these data suggest that p120ctn regulates cadherin levels and that loss of p120ctn and cadherins may underlie some cystic diseases.

NOVEL MARKERS OF ANEMIA RISK IN CHRONIC KIDNEY DISEASE

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Anemia is an early and important complication of Chronic Kidney Disease (CKD). While erythropoietin (EPO) deficiency is believed to be a proximate cause, it is clear that the causation of anemia in CKD is multifactorial. Mitochondria are critical for the proliferative response of erythroid precursors in response to EPO, and play an integral role in heme synthesis. In this pilot study we explored the relationship between mitochondrial DNA (mtDNA) copy number in peripheral blood mononuclear cells and anemia in a population of patients with CKD.

Anemia was characterized in a cohort of patients with CKD stages 1 to 4, untreated with erythropoiesis stimulating agents (ESA). MtDNA relative to total genomic DNA was measured by quantitative PCR. Other markers of chronic disease anemia measured included plasma levels of hepcidin by radioimmunoassay, EPO by ELISA and CRP. Patients were monitored prospectively for progression of anemia - decline in hemoglobin or the need to begin ESA. Healthy control individuals were included for baseline comparisons.

There were a total of 154 patients with CKD and 50 healthy controls. The mean age of the CKD patients was 60 ± 15 years, 59% were male, 86% Caucasian and 26% diabetic. GFR was <60 mL/min/m² in 76% and 44% were anemic by KDOQI criteria. The distribution of mtDNA copy number was log-normal and the transformed variable was used in all analyses. Copy number did not differ significantly between CKD patients and healthy controls; however mtDNA copy number showed significant inverse correlations relationship between GFR ($r = -0.2$; $p = 0.012$), hepcidin ($r = -0.17$; $p = 0.04$) and ferritin ($r = -0.16$; $p = 0.05$). Higher copy number was a significant predictor of Hb ($p = 0.05$) and the relationship was stronger in diabetics (p -value for interaction $= 0.04$). However over a median follow up of 700 days, it did not predict progression of anemia, the strongest predictor of which was the plasma hepcidin level (HR for progression 3.6, $p = 0.001$).

In summary, novel mitochondrial markers and hepcidin may provide more insights into the pathophysiology of anemia in CKD and need to be validated in larger patient populations.

PHYSICAL ACTIVITY AND ALBUMINURIA

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Higher urinary albumin excretion predicts future cardiovascular disease, hypertension, and chronic kidney disease. Physical activity improves endothelial function so may reduce albuminuria. Among diabetics, physical activity decreases albuminuria. In non-diabetics, prior studies have shown no association.

We explored the cross-sectional association between physical activity and albuminuria in 3587 non-diabetic women in two United States cohorts, Nurses' Health Study (NHS) I in 2000 and NHS II in 1997. Physical activity was expressed as metabolic equivalents (METs) per week. The outcome was the top albumin/creatinine ratio (ACR) decile. Multivariate logistic regression was used. Secondary analyses explored the association of ACR with strenuous activity and walking.

The mean age was 58.6 years. Compared with women in the lowest physical activity quintile, the multivariate-adjusted odds ratio for the top ACR decile for those in the highest quintile was 0.65 (95% CI: 0.46,0.93). The multivariate-adjusted odds ratio for the top ACR decile for those with greater than 210 minutes per week of strenuous activity compared with no strenuous activity was 0.61 (95% CI: 0.37,0.99), and for those in the highest quintile of walking compared with the lowest quintile was 0.69 (95% CI: 0.47,1.02).

Higher physical activity is associated with a lower ACR in non-diabetic women.

GD-IGA1: A SCREENING TEST FOR PEDIATRIC IgA Nephropathy?

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As diagnosis of IgA nephropathy (IgAN) requires renal biopsy, development of accurate screening laboratory tests would aid in evaluating patients with persistent hematuria. Increased levels of galactose deficient IgA1 (Gd-IgA1) is a principal pathogenic feature of IgAN. Studies in adults using a highly reproducible ELISA for Gd-IgA1 demonstrate significantly elevated serum Gd-IgA1 levels in patients with IgAN compared to healthy controls. Our objective was to determine whether the levels of Gd-IgA1 could be considered a marker to differentiate the pediatric IgAN patients from the pediatric disease and health controls.

Levels of Gd-IgA1 in sera from pediatric subjects (2-18 years; 16 patients with biopsy-proven IgAN, 83 healthy controls, and 17 patients with non-IgAN glomerular disease were measured. Statistical significance was determined using linear mixed modeling.

Group demographics: healthy controls (mean age 12.5±3.3 yrs; M:F 1.3:1; Black:non-Black 1.3:1), IgAN (mean age 12.7±3.9 yrs; M:F 0.5:1; Black:non-Black 0.3:1), and disease controls (mean age 10.7±3.4 yrs; M:F 0.9:1; Black:non-Black 1.8:1). Median Gd-IgA1 median value of 654 U/dL (241-1517 U/dL) for IgAN patients was significantly greater ($p=0.003$) than that for health controls (289.2 U/dL; 81-1009 U/dL) and for disease controls (351 U/dL; 80-716 U/dL). A Gd-IgA1 level of 446 U/dL (90thtile for Controls), gives a sensitivity of 75% and a specificity of 90% with a positive predictive value of 60% and a negative predictive value of 95%. Repeat Gd-IgA1 measurement at about 6 months after entry demonstrated no significant changed levels in all of the groups.

In summary, serum Gd-IgA1 levels can be used as a reasonable screening test for IgAN in pediatric patients.

PRIMARY CILIA AND FLUID FLOW ESTABLISH THE ORIENTATION OF MITOTIC SPINDLES

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Disruption of renal cilia results in renal failure characterized by tubular dilation and development of cysts. Most of the proteins involved in cystic kidney diseases localize to cilia or basal body (centrosome) at the base of the cilium. However the connection between primary cilia and renal cysts initiation and progression is still unclear. Abnormal mitotic spindle misorientation is a prominent feature of renal cysts. We proposed that cilia and flow coordinately establish the orientation of mitotic spindles and insulting any of these can randomize the spindle orientation that may lead to cyst formation. To test this hypothesis, we have generated conditional cilia mutant collecting duct cell lines from the CAGG-CreERTM, Kif3aflox mice on the ImmortoMouse background. We analyzed mitotic spindle orientation in a series of imaging with parental and cilia mutant cells under linear flow conditions. We show that ciliated control cells orientate the mitotic spindles parallel to direction of flow whereas mutant cells lacking cilia display random orientation of cell divisions. Furthermore, to assess whether cilia loss and cyst development are associated with abnormal mitotic spindle orientation, we analyzed isolated tubules from Hoxb7-Cre IFT88 conditional mutants. We found that mitotic spindle orientation is more random in mutant than control kidneys. Our results suggest that loss of cilia primarily influences mitotic spindle orientation, which appears to be a major cause of cyst development. Intriguingly we found that cilia loss results in stable and posttranslationally modified cytoplasmic microtubules *in vitro* as well as *in vivo*. Furthermore, actin fibers appear to be unorganized in mutant cells. Based on these actin and microtubules phenotypes we propose that abnormal mitotic spindle orientations in cilia mutants could be the result of cytoskeleton defects.

POLYCYSTIN-1 INTERACTS WITH ARF4 AND RAB GTPASES TO TRAFFIC TO RENAL PRIMARY CILIA

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The primary cilium plays a central role in kidney development and homeostasis. This specialized organelle sits on the apical surface of renal epithelial cells where it senses the tubular environment and initiates intracellular signaling cascades. Mutant polycystin-1 (PC1) or polycystin-2 (PC2) proteins exhibit defects in ciliary localization and function, which are central to Autosomal Dominant Polycystic Kidney Disease (ADPKD). However, the molecular mechanisms that enable targeting of the polycystins to cilia have not been described. The purpose of this study was to identify the chaperones involved in membrane transport of PC1 to cilia.

We used immunofluorescence and electron microscopy in combination with cell-based and in-vitro biochemical assays to elucidate the ciliary trafficking mechanism of PC1. We show that evolutionarily conserved VxPx motifs within the ciliary targeting domains of PC1 and PC2 form a binding site that is specifically recognized in the Golgi by active Arf4, a GTPase which functions in vesicle coat recruitment. The Arf4-polycystin-1 complex in turn binds a regulatory GTPase activating protein (ASAP1) with membrane curvature inducing activity. PC1 is bound to three further GTPases (Rab6, Rab11 and Rab8) with demonstrated roles in Golgi exocytosis and ciliary delivery. Truncation of the conserved VxPx motif decreases interaction with Arf4, ablates the delivery of PC1 to cilia and causes Golgi retention due to disruption of the trafficking complex binding.

Recently a similar complex involving rhodopsin, Arf4, Rab GTPases and effector proteins was shown to be crucial for ciliary rhodopsin trafficking in retinal cells. Thus, we provide evidence for a conserved multimeric GTPase complex and mechanism of ciliary trafficking that is shared between retinal and kidney epithelial cells. Because alterations in cellular protein localization and function contribute to renal cyst formation, we speculate that a disruption in this complex accounts for the loss of PC1 localization to cilia in ADPKD.

NON-VIRAL GENE TRANSFER FOR RENAL DISEASE AND COMPLICATIONS

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Our laboratory is focused on developing novel non-viral gene transfer strategies for cell and gene therapy of renal disease and its complications. We are developing the *piggyBac* (PB) transposon system for gene transfer to kidney *in vivo* and for development of cell therapy strategies *ex vivo*. PB is a plasmid based integrating gene delivery system capable of achieving long-term gene expression with reduced cost and possibly improved safety when compared to viral vectors. Our long-term goal is to use PB to correct Alport's syndrome in a mouse model of this human disease. We have achieved reporter transgene delivery to kidney and are optimizing strategies to improve gene transfer to kidney *in vivo*. We have also achieved efficient genetic modification (40%) of human T cells with PB and are pursuing using primary human T cells in immunotherapy strategies and cell therapies for complications of renal disease. We have used PB to achieve long-term inducible gene expression in the livers of mice *in vivo* after a single injection of plasmid DNA. PB transposons carrying an inducible human erythropoietin (hEPO) transgene are capable of increasing the hematocrit in mice long-term via regulation of hEPO expression with doxycycline. Future studies will involve using inducible hEPO transgenes in long-lived T cells *in vivo* as a novel cell therapy strategy for gene therapy of anemia of chronic kidney disease. Such a strategy may allow more tight regulation of the hematocrit in patients with ESRD and CKD and may overcome the drawbacks of commonly used recombinant EPO therapy.

NOVEL ROLE OF THE TIM-4 MOLECULE IN ALLOIMMUNITY

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Members of the T cell immunoglobulin mucin domain (TIM) family have been shown to regulate T cell differentiation. Blockade of TIM-1 signaling results in a prolongation of cardiac allograft survival that is dependent on regulatory T cells (Tregs) (JCI 2008). Here, we examine the role of TIM-4, the natural ligand of TIM-1, in alloimmunity.

Administration of RMT4-53, a mAb against TIM-4, slightly but significantly prolonged cardiac allograft survival in B6 WT recipients of BALB/c allografts (MST=9 vs 7d, $p=0.01$). While there was no difference in overall frequency of Tregs or effector/memory T cells, treated mice showed a profound increase in IL-4 producing alloreactive T cells (441 ± 80 treated vs 124 ± 23 control; $p<0.001$).

In a graft-versus-host model, treatment with RMT4-53 led to a decrease in overall CD4⁺ T cell proliferation ($43.8\pm1.0\%$ vs $50.9\pm0.7\%$, $p<0.01$), but amongst CD4⁺ Tregs there was a trend towards increased proliferation ($48.2\pm0.9\%$ vs $43.3\pm2.3\%$, $p=0.06$). Using a unique ABM-FoxP3GFP^{rep} TCR-tg model to track the fate of allospecific cells, CD4⁺GFP⁺FoxP3⁺ tg cells, which express a TCR that recognizes the bm12 antigen, were adoptively transferred into B6 mice who then received bm12 skin allografts. Mice treated with RMT4-53 had a higher percentage of allospecific FoxP3⁺ Tregs as compared to controls ($6.0\pm0.9\%$ vs. $2.9\pm0.3\%$ of CD4⁺ cells, $p<0.05$). In vitro culture of CD4⁺CD25⁻ cells with syngeneic DCs, α CD3, and low-dose TGF β also lead to a greater conversion of CD4⁺ cells into Tregs in the presence of RMT4-53 ($17.2\pm1.6\%$ vs $7.2\pm0.7\%$, $p<0.01$). Further, addition of RMT4-53 to a MLR increased TGF β concentrations (1158 ± 114.0 pg/ml vs. 280.7 ± 1.0 pg/ml control, $p<0.01$).

In summary, TIM-4 blockade leads to prolongation of cardiac allograft survival, perhaps by inhibiting overall T cell proliferation, whilst expanding the allospecific regulatory T cell population by altering the cytokine milieu towards a more tolerogenic Th2 response and/or promoting the production of TGF β .

RENAL AUTOTRANSPLANTATION FOR LOIN PAIN HEMATURIA SYNDROME: A SINGLE CENTER EXPERIENCE

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The objective of this study was to evaluate the effectiveness of renal autotransplantation as a treatment strategy for patients with Loin Pain Hematuria Syndrome (LPHS). We conducted a retrospective review of patients undergoing renal autotransplantation for LPHS during the period 1991 to 2005 in our hospital. Five patients (3 females and 2 males) with 7 autotransplants in total were identified. The average age at presentation was 22.8 years. Four patients presented with left sided flank pain of who 2 eventually developed right sided pain as well, while one patient presented with right sided pain. Typical work up included urinalysis, 24 urine protein collection, IVU, ultrasound scans, renal angiograms and renal biopsies. All the patients reported intermittent hematuria and were on opioid medications for pain relief. None of the patients had significant 24 hour proteinuria. One patient had a duplex collecting system on IVU and another patient showed some evidence of peripheral pruning on renal angiography. Biopsies revealed 2 patients with thin basement membrane nephropathy while another patient showed evidence of IgA nephropathy. One patient was referred following failure of attempted renal denervation surgery. All patients underwent renal autotransplantation with no significant intraoperative problems. Postoperative complications included 1 case each of a perirenal and right iliac fossa hematoma. Both were managed conservatively with no significant complications. 2 patients had recurrence of symptoms on the same side. Of note, 1 of these patients did not have the ureter divided during the first autotransplant and responded well to subsequent denervation and rerouting of the ureter.

Our results are consistent with other studies showing an overall success rate of 71.4% for renal autotransplantation. Compared to other treatment strategies, renal autotransplantation remains the most effective strategy to date of this little understood disorder. Though still considered a therapy of last resort due to its invasive nature, it is relatively safe in well experienced centers.

EVALUATION OF BLOOD PRESSURE AND RENAL FUNCTION BEFORE AND AFTER RENAL TRANSPLANTATION ASSOCIATED WITH CYCLOSPORINE USE

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The prevalence of hypertension in renal transplant recipients is high and has been associated with factors such as immunosuppression, with important role in graft survival. Our aims were to analyze clinical data, blood pressure (BP) and Cyclosporine (CsA) use in renal transplant recipients correlating data before and after renal transplantation.

Methods: We retrospectively analyzed 45 renal transplant recipients between 1996 and 2006. The data were obtained from medical records at Triangulo Mineiro Federal University – Nephropathology Division. We reviewed data that included: BP, urea(U) and creatinine(Cr) levels and medication.

Results: Comparison of the prevalence of hypertension and diabetes before and after transplantation showed a decrease in hypertension and an increase in diabetes. Mean arterial pressure (MAP) was significantly lower after transplantation ($p=0.011$). When analyzing U and Cr levels, we obtained a significantly reduction of both parameters after transplantation ($p=0.01$; $p<0.001$, respectively). We also compared MAP and Cr values and we found a positive but non-significant correlation between the two parameters before transplantation ($p=0,272$); and a negative and non-significant correlation after the transplant ($p=0,823$). Analyzing the use of CsA, 78,4% of the patients had such drug in their immunosuppressive scheme. The correlation between patients on CsA-use and MAP showed a negative but non-significant association ($p=0,271$).

Conclusion: Hypertension is a common complication in post-transplant patients and may contribute to a worse prognosis of the patient. However, we observed a significant decrease of BP values, urea and creatinine, demonstrating that transplantation and effective treatment may have played a significant role.

A CASE OF PRIMARY CNS PTLD IN A RENAL ALLOGRAFT RECIPIENT WITH IgG LAMBDA MONOCLONAL GAMMOPATHY

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PTLD is associated with Epstein Barr virus (EBV) infection of B-Cells. It occurs in 1% of renal transplant patients. The incidence of PTLD isolated to CNS is not known.

We present a 60 year old Haitian female with history of HTN, DM, ESRD s/p renal transplant 13 mths ago, anemia with subsequent workup showing monoclonal gammopathy of unknown significance (IgG lambda) 5 mths ago with bone marrow showing 4% plasma cells, who presented to the hospital with left sided weakness, fever of 100.3F, dizziness and an episode of dysarthria. On physical exam the patient had past pointing, and posterior neck pain. Remainder of the exam was unremarkable. Lab data showed a creatinine of 0.83mg/dL. CT head revealed probable infarct in the left cerebellum extending to the pons and complete opacification of the left maxillary and frontal sinus. The differential included malignancy, metastatic lesion, meningitis and abscess. Subsequently, MRI of the brain with contrast revealed a 3.4 x 2.8cm hyperintense lesion on T2 with central hypointensity in the left cerebellar hemisphere with mass effect on the cerebellar peduncle and shift with compression of the fourth ventricle. As well as moderate peripheral enhancement of the thick rim. Patient was empirically treated with ceftriaxone, vancomycin and ampicillin for meningitis. All diagnostic workup for AFB, fungal cultures, toxoplasmosis, aspergillosis, CMV were sent. The patient then underwent craniotomy with resection of the cerebellar mass. Pathology revealed EBV (+)B-Cell lymphoproliferative disorder consistent with PTLD and favoring diffuse B-Cell lymphoma. Neoplastic cells were positive for LCA, CD39, CD20, p53 and negative for CD10 and CD138. Upon follow up the patient was started on the first cycle of intermediate dose Methotrexate with Rituxan and leucovorin rescue therapy.

This case illustrates that in renal transplant patients, immunosuppression needs to be tailored carefully for a fine balance between the development of allograft dysfunction vs transplant related infections/malignancy. Routine screening of these patients for EBV may also identify patients that are at increased risk for PTLD.

**BK VIRURIA INCIDENCE IN RENAL TRANSPLANT
RECIPIENTS WITH HEPATITIS C AND CMV WHO HAD
DELAYED GRAFT FUNCTION AND RECEIVED
THYMOGLOBULIN**

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Purpose: Delayed Graft Function (DGF) refers to the need for dialysis post renal transplant, and subsequently increases the morbidity and mortality rate. The purpose of our study was to look at renal transplant recipients with hepatitis C and see if having CMV (IgG) and receiving Thymoglobulin for induction or DGF predisposes to having BK polyoma virus.

Method: We retrospectively reviewed records from renal transplant recipients from 1995-2009 at our transplant center who were diagnosed with hepatitis C prior to transplantation. Primary outcome was overall incidence of BK viruria in this cohort in the absence and presence of concomitant CMV (IgG), and the correlation with the use of Thymoglobulin.

Results: 38 patients with Hepatitis C between the ages of 26-78 years were identified. 81.6% were males. 44.7% of the patients received induction with thymoglobulin, 55.3% received Daclizumab, while 9 out of 38 (23.7%) received thymoglobulin post-operatively after receiving initial induction with Daclizumab for DGF or Slow Graft Function (SGF). 31 of 38 (81.6%) were positive for CMV. 9 of 38 (23.7%) had BK virus urine PCR greater than 10,000 copies, while 4 patients were not tested. Of these 9 patients, 8 (88.8%) were on a maintenance regimen of Tacrolimus and one on cyclosporine. 15 of 38 (39.5%) patients had DGF and 6 of 38 (15.8%) had SGF. Of the 15 patients with hepatitis C who had DGF, 80% also had CMV. Meanwhile of the patients with hepatitis C who had DGF only 6.7% had BK viruria. Of those patients with hepatitis C and CMV, 10 had DGF and 5 had SGF. All these patients received thymoglobulin either as induction or for SGF/DGF. Of these 15 patients, only 1 (6.7%) had BK viruria.

Conclusion: This illustrates that there is no increased incidence of BK viruria in patients with hepatitis C and CMV who have received Thymoglobulin for induction or for DGF/SGF. Also shows that a high proportion of patients with hepatitis C are likely to have DGF or SGF.

FATAL ALEMTUZUMAB ASSOCIATED COAGULOPATHY IN A RENAL TRANSPLANT RECIPIENT

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We report a case of life threatening coagulopathy that occurred in a patient who received Alemtuzumab preoperatively for a deceased donor renal transplant. A 51 yr old African American male with history of HTN, DM-2, hypercholesterolemia, CAD with CABG, and end stage renal disease (ESRD) underwent a preemptive deceased donor renal transplant. Baseline Creatinine was 5 to 6 mg/dL. Induction was with Alemtuzumab 30mg and Solumedrol 500mg preoperatively.

There were no operative complications, however post-operatively the patient was hypotensive and agitated requiring continuous mechanical ventilation. During the 20 hour postoperative period the patient's condition continued to deteriorate and required the administration of approximately 11 Units PRBC's, 9 Units FFP, 3 Units Platelets, 20 Units Cryoprecipitate as well as intravenous bicarbonate drip, with levophed, dopamine and vasopressin for further vasopressor support. The postoperative ultrasound of the transplanted kidney showed perinephric fluid collection around the transplant kidney that was believed to represent a hematoma. A concomitant 4 gm drop in hemoglobin triggered re-exploration for acute post operative hemorrhage. A significant amount of blood was found in the retroperitoneal space. The patient continued to be hypotensive and coagulopathic (PT=25.7, INR=2.2, PTT=66) with blood work consistent with disseminated intravascular coagulopathy (fibrinogen=134, D-Dimer > 10.5, platelets=36K). The patient also developed severe anion gap metabolic acidosis with concurrent lactic acidemia. Despite all resuscitative efforts the patient coded and subsequently expired. The cause of death was hemorrhagic shock secondary to coagulopathy, which was confirmed via autopsy.

This case illustrates that a small percentage of patients may be susceptible to uncorrectable coagulopathy secondary to Alemtuzumab. The mechanism and risk factors of this need to be further elucidated.

**PROSPECTIVE BK VIRAL SCREENING IN PEDIATRIC
(Ped) RENAL TRANSPLANT (RTx) RECIPIENTS (Rec)
REDUCES GRAFT DYSFUNCTION AND LOSS
ASSOCIATED WITH BK NEPHROPATHY (BKN)**

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BKN has emerged as a significant cause of allograft dysfunction. It has been reported to cause graft loss in up to 50% once it sets in. The objective of the study was to determine the significance of prospective BK viral screening in Ped RTx Rec.

This is a retrospective study using multivariate analysis to assess risk factors associated with BK infection (viruria, viremia, and BKN) in 27 Ped RTx Rec transplanted and followed up to 25-months (mon) post Tx. RTx Rec were prospectively screened for BK virus in urine and plasma by quantitative PCR at regular intervals.

The mean follow up period was 15.1 (2- 25) mon. The mean Rec age was 12.3 (1.9- 19.8) years, 58% were male, and 85% received deceased-donor Tx. 96% were primary Tx. All pts received induction with a 4-day Methylprednisone taper and either Daclizumab (56%) or r-Thymoglobulin (Thymo) (44%). Maintenance immunosuppression (IS) consisted of Tacrolimus (TAC)/ Mycophenolic acid (MPA) in 88%, TAC/ Prednisone (P) in 8% and TAC/ MPA/ P in 4%. Screening was started 2 weeks post Tx and continued monthly. The frequency of testing was increased once viremia developed or following treatment of acute rejection (AR). 16 (59%) pts developed BK viruria, 7 (26%) viremia and 1 (4%) was found to have BKN on surveillance biopsy. Reduction in IS was the first line therapy in pts who developed viremia.

Thymo use and higher TAC trough level were associated with the development of viremia ($p= 0.0158$ and 0.0269 respectively). Donor and recipient ages and pre Tx IS were not found to be significant risk factors. None of the pts developed AR as a result of reducing IS therapy. No graft loss due to BKN occurred in spite of 26% having viremia. Early screening and reduction in IS appear to be safe and associated with a reduced incidence of BKN. The cost of the screening may be offset by the reduction in cost associated with reduction in IS therapy. The optimal timing, frequency of testing, and the most appropriate therapy still need further study.

**USE OF INTRAVENOUS IMMUNOGLOBULIN (IVIG) IN
THE TREATMENT OF BK VIREMIA (TBKV) AND
NEPHROPATHY IN PEDIATRIC TRANSPLANT (PTX)
RECIPIENTS (REC).**

Anyagbu, E; Al-Akash, S. Kidney Center Driscoll Children's Hospital, Corpus Christi, Texas, USA.

BK virus nephropathy (BKVN) is a significant cause of allograft dysfunction (AD) and loss in ptx rec. No specific therapy is available. Reduction in immunosuppression (IS) has been found to decrease viral load and improve graft function. We report viral clearance and histological resolution of BKVN with IVIg.

Ptx recs were prospectively screened monthly for BK by whole blood or plasma quantitative PCR. Frequency of testing was increased with the detection of BK viremia (BKV) or after the treatment for acute rejection (AR). IS was reduced with BKV until clearance was achieved. Persistent viremia or detection of BKVN histologically prompted IVIg therapy.

Twenty seven ptx recs were screened for BKV. Mean follow-up was 15.11 ± 8.17 months (mon), with 66% completing 12-mon. BKV was detected in 22% at 33-631 (149.17 ± 236.62) days post transplant. PCR ranged from 400-5200 (2049 ± 1747) copies/ml. Three pts (11%) received IVIg (2 gm/kg) due to persistent BKV, while one patient (4%) was diagnosed with BKVN on protocol biopsy, and received IVIg with resolution confirmed by biopsy 27 days later. AR was not seen following TBKV with reduction of IS. AD was not seen in our patients.

Our protocol for BK surveillance might account for our low incidence of BKVN. IVIg seems to be effective for treatment of BKV and BKVN. IVIg therapy may be considered in pts with persistent BKV after IS reduction and pts with BKVN.

CORRELATION BETWEEN IMMUNE CELL FUNCTION (ICF) ASSAY AND RATE OF REJECTION AND INFECTION IN RENAL TRANSPLANT RECIPIENTS

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Background: Immune Cell Function (ICF) assay measures ATP production that occurs in T-lymphocytes as a surrogate marker for functional immunity in solid organ recipients. Few studies have investigated the relation between ICF level and rejection or infection in renal allografts.

Methods: We performed a retrospective analysis of all recipients of kidney allografts between January 2007 and November 2009 using electronic medical records (EMR). All patients who received tacrolimus and mycophenolate mofetil immunosuppression were included. Predictor variables collected included demographic data, ICF level, trough tacrolimus levels, and type of induction therapy (alemtuzumab or antithymocyte globulin). The primary outcome was a rejection diagnosis based on biopsy results. Secondary outcomes were polyomavirus (BK) nephropathy and cytomegalovirus (CMV) infection diagnosed based on clinical and laboratory data.

Results: A total of 93 patients met the inclusion criteria. Of these, 15 patients had clinical rejection (16%). The patients who experienced rejection were more likely to have received cadaveric organs (73 vs. 44%), and induction with thymoglobulin (40 vs. 18%). The ICF level was similar between patients who had rejection vs. those who did not (293 vs. 312; $p=0.6$). There was no statistically significant difference between the ICF level and rate of BK or CMV infection. Average tacrolimus serum level tended to be lower in patients with rejection (7.9 vs. 8.2) albeit not statistically significant.

Conclusion: Our data suggests that there is no significant relationship between the ICF level and the rate of rejection or BK/CMV infections in recipients of kidney allografts. Clinical usage of this test appears to be of limited value in the management of immunosuppression therapy of these patients.

SINGLE-DAY WORK-UP PROTOCOL IS ASSOCIATED WITH A SHORTER TIME TO KIDNEY TRANSPLANT: THE YALE-NEW HAVEN TRANSPLANTATION CENTER EXPERIENCE

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Renal transplantation offers patients with end stage renal failure improved survival and quality of life compared with dialysis. Although more kidney transplants are being performed, the time to transplant is increasing at a faster rate with a resulting average wait time of 4-5 years per patient. Little is known about the role of in-center single day screening programs and their contribution to the waiting time to transplantation.

We conducted a retrospective study at the Yale New Haven Transplantation Center to evaluate the waiting time to kidney transplant between 2 protocols: Protocol 1 (P1, 1996-2006; multiple day screening) versus Protocol 2 (P2, 2007-2009; in-center single day screening).

Three hundred and forty patients were identified in P1 and 161 in P2. The average age 50.1 vs 50.4 ($p=0.7$), gender 62.2% vs 62.7% males ($p=0.9$), distance to transplant center (< 30 miles) 65.4 vs 67.8 ($p=0.06$), African American race 37% vs 32% ($p=0.4$) and adequate functional status (able to perform activities of daily living) 93.3% vs 88.2% ($p=0.3$) were comparable between the two groups. The average time to transplantation listing was 1229 days vs 264 days ($P=0.0001$) for P1 and P2, respectively. The time to transplant was 939 days vs 156.3 days ($p<0.0001$) for P1 and P2 respectively. Deceased donor kidneys constituted 79.6% for P1 compared to 53% for P2 ($p<0.0001$).

We found that an in-center single day screening program for kidney transplant effectively reduced the time to transplant listing. Our data suggests that the time interval between UNOS listing and transplantation may be predicted by the time to transplant listing. Indeed, the statistically significant difference in living vs. deceased donors may explain the majority of this finding. However, we explored the possibility that other potential factors were contributing.

PROGRESSION OF CORONARY ARTERY CALCIFICATION (CAC) IN INCIDENT RENAL REPLACEMENT THERAPY (RRT) PATIENTS BY MODALITIES OF RRT

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Introduction: Cardiovascular disease (CVD) is the leading cause of mortality in end stage renal disease patients. We performed a prospective cohort study to determine the prevalence of CAC, a risk predictor of CVD, at the initiation of RRT and assess impact of mode of RRT on CAC progression. Methods: We used electron beam computed tomography (EBCT) scan to measure CAC in 79 incident dialysis (HD) or renal transplant recipients with no prior coronary history. All patients were followed for at least 2 years and 45 patients had a 2nd EBCT. Results: Participants were equally distributed in gender and race [44.3% male and 49.4% Caucasian]. The prevalence of diabetes and hypertension was 38% and 95%, respectively. The median time to first EBCT after initiation of RRT was 2.5 months.

Variables mean \pm SD	All Patients (n=79)	CAC in baseline EBCT		RRT modality	
		Absent (n=42)	Present (n=37)	HD (n=39)	Transplant (n=40)
Age	51.7 \pm 13.9	44.9 \pm 12.5	59.5 \pm 11.2	55.1 \pm 14.5	48.4 \pm 12.6
CAC score at time of RRT start (Agatston)	144 \pm 267	0 \pm 0	307.3 \pm 320.7	132.5 \pm 253.4	155.1 \pm 282.3
Absolute CAC progression	70.2 \pm 192.7 (n=45)	22.4 \pm 74.9 (n=23)	120.2 \pm 258.5 (n=22)	99.2 \pm 141.5 (n=14)	57.1 \pm 212.7 (n=31)

Discussion: Participants with no CAC were younger than the group with CAC (p <0.001). The mean CAC progression was lower in no-CAC group (p=0.09). HD patients had a higher mean age (p=0.03) but similar baseline CAC. The mean progression of CAC was higher in patients started on HD, but it did not reach statistical significance (p=0.5). Larger studies are warranted.

ISOLATED RENAL ASPERGILLOMA CAUSING OBSTRUCTIVE UROPATHY IN RENAL ALLOGRAFT- CASE REPORT AND UNIQUE APPROACH TO MANAGEMENT

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Isolation of *Aspergillus* in the renal collecting system without dissemination is rare. We present a case of noninvasive renal aspergillosis presenting in the form of a bezoar (collection of fungal hyphae) causing obstructive nephropathy in a renal allograft. Our unique and successful approach to management is presented as an alternative to surgical intervention.

A 51 year old man with a history of diabetes mellitus, hypertension and ESRD secondary to polycystic kidney disease s/p living, unrelated renal transplant who presented ten years later to the emergency department with complaints of fever and decreased urine output for one day. His current immunosuppressive therapy included tacrolimus, mycophenolate mofetil, and prednisone. Physical exam was significant for a febrile male who appeared comfortable. The bladder was not palpable and insertion of a foley catheter yielded no urine. Laboratory studies revealed acute renal failure (creatinine 5.2mg/dL). CT scan revealed moderate hydronephrosis of the allograft without evidence of calculi or mass. Antegrade pyelogram revealed a proximal ureteral filling defect. Insertion of a nephrostomy tube proximal to the obstruction resulted in diuresis. Urine microscopy yielded 50 WBC per high power field, few bacteria and few squamous cells. Three days after collection urine culture and cytology identified *aspergillus* species. Blood cultures remained negative. Radiography with urine culture results diagnosed the obstruction as an aspergilloma, a collection of fungal hyphae obstructing the transplanted kidney's ureter.

Our patient was treated with intravenous voriconazole for seven days without improvement. We adapted a method to monitor intrarenal pressure to prevent further hydrostatic damage using intra cranial manometer during continuous infusion of intrapelvic amphotericin. Clinical response was evident by gradual increase in urine flow via the urethra and defervescence. Six days later a third nephrostogram showed brisk contrast flow through the collecting system and resolution of the obstruction

**CAPILLARY C4d DEPOSITION IN KIDNEY
ALLOGRAFTS BIOPSIES: RELATIONSHIP WITH
DIAGNOSIS, IMMUNOGLOBULINES, RENAL FUNCTION
AND FIBROSIS**

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C4d deposition in peritubular capillaries has been described as a marker of humoral renal graft rejection. The objectives of this study were to characterize the epidemiological and clinical features of kidney transplant patients of the Nephropathology Department of the University of Triangulo Mineiro, and to analyze morphologically and by immunohistochemistry for C4d their biopsies to classify it according to Banff 07.

To analyze the fibrosis the fragments were stained with Picro-sírius. The slides were viewed under polarized light with the 40x objective and calculate the percentage of fibrosis per area of the examined field (% F). For peritubular C4d, IgA, IgG, IgM, C3 and C1q detection was used the technique of immunohistochemistry. The diagnoses were made according to the presence of C4d (C4d0, C4d1, C4d2 and C4d3) and classified according to Banff 07.

The mean age was 41.1 years and had positive correlation with the percentage of fibroses ($r = 0,452$, $p = 0005$). The median of the percentage of fibrosis was 1% in the group C4d0, 1% in C4d1, 2% in C4d2, and 3% in C4d3 and the correlation between the intensity of fibrosis and deposition of C4d was positive. Between the diagnosis, rejection mediated by antibodies (RMA) represented 36.7%, rejection mediated by cells (RMC) 16.7%, borderline lesions (BL) 25% and interstitial fibrosis/tubular atrophy (TF/TA) 18,3%. Comparing according to the type of diagnosis, the mean fibrosis for acute RMA was 2%, acute RMC 1%, chronic RMA 5%, BL 2.5%, TF/TA 4% and others 1%. There was positivity for IgA, IgM and C3 in groups C4d0, 1, 2 and 3, for IgG and fibrinogen in groups C4d2 and 3, and for C1q in groups C4d1, 2 and 3. C4d3 group showed a higher percentage of fibrosis, with elevated serum creatinine levels, and a positive correlation between fibrosis and intensity of C4d expression.

The co-expression of C4d with IgG and C3 could indicate the activation of the classical complement pathway. The relation between C4d, fibrosis and creatinine levels may be indicative of renal function decline and worse prognosis.

COMPARISON OF PROTEINURIA WITH SIROLIMUS AND TACROLIMUS IN RENAL TRANSPLANT RECIPIENTS

Saurabh Goel, Anas Al Rifai, Mohamed El-Ghoury, Detroit, MI, US

Development of proteinuria in patients with and without chronic allograft nephropathy (CAN) has been described after conversion from Calcineurin inhibitor (CNI) to Sirolimus (SRL), as well as in those receiving SRL de novo. The pathogenesis and origin of proteinuria, tubular vs glomerular is being debated. Recent studies reveal no significant difference in proteinuria after conversion from CNI to SRL. Proteinuria is one of the main predictive factors of graft loss. We seek to determine the influence of SRL compared to Tacrolimus (TAC) on proteinuria in our transplant patients.

We compared 178 consecutive patients receiving kidney transplant at our institution. After induction with 4.5mg/kg thymoglobulin and intravenous steroids, all the patients received mycophenolate mofetil (MMF) in a steroid-free maintenance protocol, along with SRL (n93) or Tacrolimus (n85). Both groups were comparable with respect to age, gender and race. The prevalence of abnormal proteinuria (Urine Protein Creatinine ratio (UPCR) > 0.2) at 1 and 3 yrs was 37.3% and 36.4% with SRL vs 44.1% and 50.0% with TAC, respectively (p 0.45 at 1yr, 0.17 at 3 yrs). The prevalence of nephrotic range proteinuria (UPCR>3) was 4.4% with TAC and 0% with SRL at 1 yr (p 0.13), and 10.5% and 3.0%, respectively at 3 yrs (p 0.11). The mean UPCR in the SRL and TAC groups was 0.36 and 0.46 at 1 year (p 0.47) and 0.38 and 0.93 at 3 years (p 0.13), respectively. ACEI and/or ARB usage (27.5% & 40.8% with SRL and 36.8% & 42.5% with TAC at 1 & 3 yrs respectively), serum creatinine and eGFR (Cockcroft Gault) were similar in both groups at both times. The mean systolic & diastolic BP at 1 year were 131 & 78mmHg with SRL and 132 & 77mmHg with Tacrolimus, respectively (p 0.82 for systolic BP, 0.60 for diastolic BP). At 3 yrs, the mean systolic BP was higher in Tacrolimus group at 135mmHg vs SRL group at 129mmHg (p 0.05), while mean diastolic BP was similar.

Our results indicate that, with the doses of SRL used currently with a target trough of 8-12ng/ml, there is no significant difference in the prevalence or severity of proteinuria between TAC and SRL based immunosuppressive regimens at 1 year and 3 years post transplant.

T-CELL LYMPHOMA EARLY AFTER RENAL TRANSPLANT

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The overall incidence of Post Transplantation Lymphoproliferative Disorder (PTLD) is approximately 1%, with a recent trend towards increasing frequency following more potent immunosuppressive regimens. The majority of PTLDs occurring early are EBV associated B-cell non-Hodgkin lymphomas. T-cell PTLDs constitute 14% of all PTLDs and tend to occur late following transplantation after exposure to prolonged immunosuppression (IS), with median time from transplant to diagnosis of 15 yrs. Extranodal involvement is the norm and the prognosis is generally poor. EBV has been inconsistently implicated in T-cell PTLD. We present a case of early T-cell PTLD, after minimal IS in a renal transplant recipient.

A 59 year old African American male with end stage renal failure, received a deceased donor renal transplant with 4.5 mg/kg of Thymoglobulin in 3 divided doses and intravenous steroids followed by steroid-free maintenance IS with cellcept and tacrolimus. He achieved immediate graft function, establishing a baseline creatinine of 1.1 mg/dl and no rejection. Five months post transplant, he presents with a 6-week history of nausea, anorexia, abdominal pain, night sweats and 10 pound-weight loss. A thorough workup including lymph node and bilateral bone marrow biopsies revealed a diagnosis of monomorphic T-cell PTLD stage 3A, with no extra-nodal tissue involvement. In-situ hybridization for EBV demonstrated rare positive cells, while EBV-PCR tested negative in blood. Cellcept was discontinued, tacrolimus reduced by half and oral steroid initiated. He received six cycles of CHOP (cyclophosphamide, doxorubicin, vincristine and prednisone) and achieved complete remission with negative follow-up PET scans at 2 years.

Our case is unique for its early presentation, 5 months post transplantation, and is one of the earliest reported T-cell PTLD with minimal IS. Moreover, there was no extranodal involvement and a rapid remission with chemotherapy and lowering of IS. A low threshold for diagnosis of T-cell PTLD, even early in the post-transplant period may help to improve the outcome through prompt intervention.

RENAL TRANSPLANTATION AFTER SUCCESSFUL TREATMENT OF CALCIPHYLAXIS WITH SODIUM THIOSULFATE

Deepika Jain, Preethi Yerram, Ramesh Khanna

Calciphylaxis or Calcific Uremic Arteriopathy (CUA) is a almost fatal condition characterized by mural calcification of small vessels resulting in severe ischemia and tissue necrosis with skin ulceration, and secondary infection. With the exception of one case report, data on renal transplantation after treatment of CUA, and the outcomes in such patients is lacking. Here in, we report a case of CUA that was successfully treated with intravenous (IV) sodium thiosulfate (STS), and went on to receive a successful cadaveric renal transplantation, (CRT) without recurrence of CUA 2 years post transplantation.

42 year old Caucasian female with CRT in 2002 (ESRD of uncertain etiology), presented with subcutaneous nodules and skin ulcerations on the abdomen, back, and buttocks with severe pain in early 2004. Skin biopsy confirmed the diagnosis of CUA. Pt was treated with IV STS for 5 months along with aggressive wound care with complete resolution of her skin lesions. In the interim, patient was noted to have worsening allograft dysfunction with no clear pathology identified. Pt was evaluated for retransplantation, and received a CRT in October 2007.

After transplantation, patient did well, and has not had any recurrence of CUA since stopping the STS. Pt has had near normal creatinine until November 2008, when she had a sudden increase in her creatinine which was proven to be secondary to nephropathy on renal biopsy, and is currently being treated for the same. At the time of submitting this abstract, no particular etiology for this increase could be found. Patient did not respond to empiric treatment with increased doses of prednisone, ruling down the possibility of acute rejection. A renal biopsy among other things is being planned at this time. Also of note, p

We feel that our case report provides valuable information in an area where data is lacking. This case is unique since the patient not only recovered from CUA after treatment with IV STS, but also went on to receive a second renal transplant, without any evidence of recurrence of CUA after stopping STS therapy. This demonstrates that IV STS successfully treats CUA, and once treated, these patients should potentially be considered for renal transplantation. The above being said, data on long term outcomes in these patients are lacking, and this needs to be studied further.

A 57 YEAR OLD KIDNEY TRANSPLANT RECIPIENT WITH SKIN LESIONS, POLYNEUROPATHY AND DIFFICULTY WALKING

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Multiple nutritional deficiencies with skin lesions and neurologic manifestation is a rare disorder.

A 57 year old African American male who underwent deceased donor kidney transplant 10 years ago, presented with diffuse skin lesions, polyneuropathy, difficulty walking, memory deficits, weight loss and diarrhea of 4 month duration. His graft function so far has remained stable while maintained on cyclosporine, mycophenolate and prednisone. His exam was significant for exfoliative dermatitis of the hands and finger tips in addition to a psoriasiform dermatitis of the trunk and bilateral lower extremities.



He was diagnosed with acute on chronic pancreatitis. Skin biopsy was consistent with nutritional deficiencies vs. necrolytic migratory erythema. Work for neuroendocrine tumors was negative. However, he was found to have multiple vitamin and mineral deficiencies: vit A <0.06mg/l(0.3-1.2), vit B1 66 nmol/l (70-180), vit B6=1.4 ng/ml (5-30), 25 hydroxy vitD <6 ng/ml (25-80), and zinc 35 mcg/dl (60-150). He was subsequently placed on vit A, B1, B6, vit D and zinc in addition to multivitamins, vit C and pancreatic enzymes. He was able to walk again, and 4 months later, there was a dramatic improvement of his skin lesions with complete resolution.

Multivitamin deficiencies causing neurological deficits has been commonly described in the medical literature but it is uncommon to encounter extensive skin manifestations of vitamin deficiency combined with neurological symptoms in a transplant patient. Early recognition and prompt treatment may prevent the progression to potentially irreversible neurological and skin disorders.

HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN A RENAL TRANSPLANT PATIENT

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Hemophagocytic lymphohistiocytosis (HLH) is a rare condition that develops due to inappropriate and dysregulated activation of immune system associated with familial immune disturbances, several infectious, neoplastic and autoimmune diseases, and immune deficiency states such as post kidney or liver transplantation.

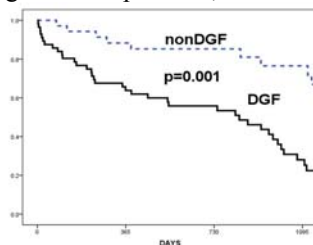
We report a 55 year old female 15 years post renal transplant for focal segmental glomerulonephritis who was started on Sirolimus after discontinuation of Cyclosporine 2 months prior to hospital admission. She presented with sudden onset high grade fever of 39.6 degree centigrade and splenomegaly, devoid of other localizing symptoms or signs. Cultures and serologic evaluation failed to reveal any obvious source of fever. She was leukopenic with WBC of 3300/ cu. mm. Lipid panel demonstrated triglyceride of 490 mg/dl. Buffy coat antigenemia was negative for Cytomegalovirus while Epstein Barr virus (EBV) antibodies were elevated with IgG EBV of 3420 IU/ml; however, the IgM antibody was only 10 IU/ml. Renal biopsy showed chronic allograft nephropathy. The bone marrow biopsy showed an increased proportion of hemophagocytic macrophages with normal lymphocyte subsets without evidence of infection. The patient became afebrile 48 hours after treatment with Prednisone 40 mg daily.

Our patient had high fever, sudden onset cytopenia, splenomegaly, hypertriglyceridemia, and bone marrow morphology suggestive of HLH. The central pathophysiologic abnormality in HLH is cytokine dysfunction resulting in uncontrolled accumulation of activated T-lymphocytes and activated histiocytes (macrophages) in various organs. The recent change from Cyclosporine to Sirolimus may have unmasked HLH in her as Cyclosporine is considered a component of treatment for HLH. She was converted from Rapamycin back to Cyclosporine. Our patient has done well with this strategy for 2 years and recently received her second kidney transplant.

DELAYED GRAFT FUNCTION (DGF) IN HIV+ KIDNEY TRANSPLANT RECIPIENTS PREDICTS WORSE LONG-TERM RENAL FUNCTION

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In general, DGF has been associated with worse renal function and allograft survival compared with non-DGF renal transplant recipients. However, this may not hold true in certain subsets of patients. DGF has not yet been explored in HIV+ renal transplant recipients. We conducted a retrospective review of 91 consecutive HIV+ renal transplants performed at our university hospital center for incidence of DGF and the effect on renal function, graft survival, and patient survival at 1 and 3 years. Of the 91 patients, 35 did not have DGF and 56 had DGF. Demographics and HAART therapy were comparable between the two groups except that the DGF group was statistically older than the non-DGF group. Donor risk factors for DGF, including ECD, ARF, donor terminal creatinine, and number of pressors were also comparable between the two groups. One exception was the cold ischemia time (CIT), a known risk factor for DGF, which was statistically longer in the DGF than in the non-DGF group (mean 16.9 vs. 12.6, $p=0.009$). We found that renal function estimated by MDRD at all time points calculated over a 3-year period post-transplant was worse in the patients with DGF with a statistical significance of $p\leq 0.001$. Long-term graft survival was also significantly worse in the group with DGF than in the group without DGF at 3 years (log rank significance $p=0.001$, dotted line=non-DGF, solid line=DGF).



VITAMIN D DEFICIENCY AND BONE DISEASE IN RENAL TRANSPLANT PATIENTS: A MISSED OPPORTUNITY

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Vitamin D deficiency may aggravate secondary hyperparathyroidism and can cause significant decrease in bone mineralization. Kidney transplantation causes a reduction in parathyroid hormone (PTH) concentration by restoration of kidney function, correction of hyperphosphatemia, and production of 1, 25 dihydroxyvitamin D₃. In spite of this, persistent hyperparathyroidism is common after transplantation and renal osteodystrophy still remains an ongoing problem. This study aimed to determine the vitamin D status after renal transplantation and examine its relationship with PTH and bone mineral density (BMD).

We conducted a retrospective study on all adult renal transplant patients attending the renal transplant clinic from January 1999 to July 2009 who had 25-hydroxyvitamin D, PTH and BMD measured. Vitamin D level was defined according to NKF/KDOQI guidelines.

Of the 419 renal transplant recipients, only 33 had a vitamin D level measured. In these patients, all had low levels with a mean of 17 (+/-9) ng/mL. Vitamin D insufficiency was present in 54%, deficiency in 45%, and severe deficiency in 1%. Seventeen of the 33 patients also had a PTH level measured. Of these, 82% had a degree of hyperparathyroidism. Ten of the 33 patients had a BMD measured. Three had osteoporosis and 4 had osteopenia.

In this study, we identify a missed opportunity in monitoring bone disease in our patients. Of the few patients who had vitamin D levels measured, all had low serum 25-hydroxyvitamin D. This may aggravate secondary hyperparathyroidism and may have an impact on BMD. In order to increase the recognition of the previously neglected bone complication after kidney transplant, a comprehensive approach is currently being developed to address this opportunity in our center.

MANAGEMENT OF PERSISTENT HYPERPARATHYROIDISM WITH CINACALCET FOLLOWING KIDNEY TRANSPLANTATION: THIRTY SIX MONTH FOLLOW UP

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We evaluated the use of cinacalcet to optimize persistent hyperparathyroidism (HPT) over a period of 36 months in patients with successful kidney transplantation. Thirty-seven patients with persistent HPT 3 months following transplantation with stable graft function were treated with cinacalcet 30 mg/day and titrated to a maximum of 120 mg/day. Thirty five of the 37 patients underwent induction therapy with alemtuzumab and methylprednisolone at the time of engraftment, followed by rapid steroid withdrawal (500/250/125 mg/day on days 0/1/2 respectively). Maintenance immunotherapy comprised of standard suppression regimens with 33/37 (89%) of the patients receiving a combination of calcineurin inhibitors and mycophenolate mofetil. Serial serum iPTH, calcium, phosphorus and alkaline phosphatase (AP) levels were measured over 36 months. The data is presented as mean value \pm standard deviation. Serum iPTH decreased from 230 ± 139 pg/mL at the start of therapy to 128 ± 68 pg/mL at 36 months ($p < 0.001$). Serum calcium decreased from 10.26 ± 0.71 mg/dL to 9.58 ± 0.91 mg/dL ($p < 0.001$) and serum phosphorus concomitantly increased from 2.55 ± 0.74 mg/dL to 3.26 ± 0.62 mg/dL ($p < 0.001$). Serum AP levels decreased from 110 ± 38.7 U/L to 84.9 ± 23.8 U/L ($p < 0.001$). Twenty-one of the 37 patients remained on cinacalcet therapy for the duration of 36 months with a mean dosage of 50 ± 30 mg/day. Factors contributing to discontinuing treatment included achieving appropriate iPTH level for their stage of chronic kidney disease, gastro-intestinal side effects, and financial constraints. Renal function remained stable with a mean serum creatinine and eGFR (MDRD equation) of 1.55 ± 0.94 mg/dL and 53.3 ± 22.6 ml/min at baseline and 1.42 ± 0.43 mg/dL and 53.3 ± 19.6 ml/min at 36 months (NS). Serum albumin levels remained unchanged and no interactions were observed with the immunosuppressive agents used. **Conclusions:** Cinacalcet is an effective agent in the long term management of persistent post renal transplant hyperparathyroidism and has no deleterious impact on renal function in this population.

SCHISTOSOMIASIS AND CMV COLITIS IN A RENAL TRANSPLANT PATIENT

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A 51 year old Hispanic male with ESRD secondary to polycystic kidney disease s/p cadaveric kidney transplant in April 2008 was admitted with fever and diarrhea of three days duration. CMV status +ve donor to -ve recipient. Past history was positive for CMV viremia. His current regimen includes Tacrolimus 10mg twice daily and prednisone 10mg daily.

On clinical exam he was found to have a temperature of 38.5⁰ C, hypotensive and WBC of 3.1 with 88% neutrophils. The chest was clear with good air entry bilaterally. Heart sounds S1 and S2 were heard with no murmurs, rubs or gallops. The abdomen was diffusely tender to palpation with no organomegaly or rebound tenderness. The clinical picture was consistent with septic shock and he required pressor support for his blood pressure and broad spectrum antibiotics. The CMV PCR titers were >250000 copies/ml and was treated with gancyclovir 2.5 mg/kg IV bid.

Given the high suspicion for CMV colitis he had a colonoscopy which showed erythematous friable colonic mucosa which was biopsied. The biopsy results came back positive for *Schistosoma mansoni* and cytomegalovirus colitis. On reviewing the history the patient had been to Porto Rico recently where Schistosomiasis is known to occur. He is currently being treated with Praziquantel 1600mg PO BID and Gancyclovir.

Diagnosis- Schistosomiasis and Cytomegalovirus colitis.

INFLUENCE OF HEPATITIS C (HCV) CO-INFECTION ON C4D STAINING AND PRESENCE OF DSA IN HIV POSITIVE RECIPIENTS

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To determine the incidence of C4D deposition in renal biopsy specimen of HIV+ and HIV- transplant recipients with or without Hepatitis C infection

We studied 405 surveillance and indication biopsies and Panel reactive antibody titers performed between March 2007 and November 2009 on 194 renal transplant recipients. This included 27 HIV+ recipients and 156 HIV- recipients.

Table: Comparison of HIV positive and HIV negative recipients

	HIV+/HCV+	HIV+/HCV-	HIV-/HCV+	HIV-/HCV-
No of patients	4(2%)	23(12%)	11(6%)	156(80%)
ATP level	150±71	251±116	375±179	283±134
T cell PRA	9.25±14.7	16.5±31.1	17.5±32.8	4.9±14.4
B cell PRA	10.5±19.6	18.8±24	9.4±28.2	4±14.1
Antibodies				
1)DSA positive	2(50%)	9(39%)	2(13%)	16(9%)
2)Non specific	2(50%)	12(52%)	4(36%)	32(18%)
3)Negative	0(0%)	2(9%)	3(27%)	106(68%)
C4D1 positive	0	6(26%)	3(27%)	34(22%)
C4D2 positive	0	1(4%)	0(0)	7(5%)
C4D3 positive	2(50%)	6(26%)	1(6.5%)	9(6%)
C4D negative	2(50%)	10(43%)	7(30%)	106(68%)

HIV positive recipients have a significantly higher incidence of C4D positivity in renal biopsies ($p\text{-value} = 0.001$) and higher incidence of DSA positivity compared to non HIV patients ($p\text{-value} < 0.001$). In addition, HIV and HCV positivity independently appear to be associated with higher panel reactive antibody titers compared to non HIV and non Hepatitis C positive recipients.

Higher incidence of intense C4D staining in HIV positive transplant biopsy specimen. HCV does not seem to influence the intensity of C4D staining in HIV + recipients.

MINORITIES HAVE SURVIVAL ADVANTAGE AMONG RAPAMYCIN-TREATED RENAL TRANSPLANT PATIENTS

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Ethnic differences in mortality among recipients treated with Rapamycin are not well understood. We did retrospective cohort study assessing ethnic differences in mortality among first renal allograft recipients transplanted between 1993 and 2003 and followed until 03/2009. Mortality was compared for Whites, Blacks and Hispanics using Cox proportional hazards model, adjusting for demographic factors, comorbid conditions, donor variables, and acute rejections.

411 of 977 patients (42%) received primary immunosuppression with Rapamycin. 380 of those (92%) with complete data were included. There were 44% Whites, 29% Blacks and 26% Hispanics. Blacks had higher prevalence of hypertension, a longer time on dialysis prior to transplant, a higher incidence of humoral rejections, an increased graft loss, and a higher BMI. A total of 106 (28%) patients died during follow-up. The results of analysis are shown below.

Race/Ethnicity	RR	95% CI	p
Unadjusted Model			
Black (vs White)	0.74	0.46-1.19	0.21
Hispanic (vs White)	0.62	0.38-1.01	0.06
Demographic Model			
Black (vs White)	0.81	0.51-1.30	0.39
Hispanic (vs White)	0.75	0.46-1.22	0.25
Age at transplant RR-1.06/ yr, male sex RR-1.61 were significant			
Full Model			
Black (vs White)	0.54	0.31-0.91	0.02
Hispanic (vs White)	0.61	0.36-1.04	0.07
Male sex (RR 1.73), age at transplant (RR 1.05/yr), diabetic ESRD (RR 3.80), time on dialysis pre-transplant (RR 1.01/mth), PRA >20% (RR 1.98), and graft loss (RR 2.02) were significant.			

Blacks had a survival advantage despite a higher incidence of humoral rejection and a greater graft loss. Hispanics showed a trend towards an improved survival despite an adverse comorbid burden. The reasons for these discrepancies needs further study.

MICROANGIOPATHIC HEMOLYTIC ANEMIA COMPLICATING RENAL TRANSPLANT

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Development of thrombotic microangiopathy (TMA) syndromes has been documented in organ transplant recipients, the majority of which are presumed to be due to calcineurin inhibitors (CNI) that typically are administered to prevent rejection. However the diagnosis of this entity is not straightforward, as rejection may present with TMA and requires active management.

We are presenting a case of a 41 year old white female seen 2 weeks postoperative following kidney transplant (DCD-reason for transplant CKD of unknown origin) with PRA of 40% -with complaint of copious wound drainage, acute rise in creatinine (2.4 to 4.7), emesis and incisional pain. During the first day of hospitalization, she developed anuria, hemolytic anemia and progressive thrombocytopenia, requiring multiple PRBC and platelet transfusions and hemodialysis was initiated. Hemolytic uremic syndrome (HUS) was initially suspected, because in addition to hematological changes the graft function deteriorated. Unexpectedly, the results of the direct antiglobulin test became positive which is not normally observed in the HUS. In the meanwhile her post-transplant flow crossmatch results showed presence of donor-specific antibodies, consistent with diagnosis of antibody-mediated rejection. Plasmapheresis and IVIG treatments were initiated, and her Tacrolimus was gradually phased by Cyclosporine. The kidney biopsy that was performed during the exploration showed no evidence of cellular rejection, but was consistent with AMR (antibody mediated rejection) and TMA.

Whether the thrombocytopenia was also due to an immune process was not clear, although some evidence favors this hypothesis. Patient received multiple transfusions with subsequent reexploration of the perigraft collection and died due to acute respiratory failure.

The diagnosis of post transplant recurrent HUS is difficult; because the histological features could be similar to those seen in acute humoral allograft rejection and HUS induced by cyclosporine or FK-506 nephrotoxicity.

The presence of TMA should prompt a diligent search for AMR. CNI induced TMA is a diagnosis of exclusion.

INTRAVENOUS IMMUNE GLOBULIN ASSOCIATED NEPHROPATHY IN KIDNEY TRANSPLANTATION

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IVIG is increasingly used in kidney Tx recipients for desensitization and in the treatment of antibody mediated rejection. Several IVIG preparations are available with stabilizing agents including sucrose and maltose. We present a case of acute kidney allograft injury from IVIG in a renal transplant recipient. A 69 yr old male with history of kidney Tx was admitted with persistent diarrhea. Stool testing revealed C-diff infection and he received metronidazole and oral vancomycin. Lack of timely improvement in diarrhea led to testing for immunoglobulin levels, which came back at low. Patient received sucrose containing IVIG, total of 1.2 gm/kg in divided doses. Serum creatinine started rising within 24-48 hrs after IVIG from 2.2 to 7.4. Allograft biopsy showed severe arteriolar hyalinosis and severe epithelial cell vacuolization. Patient was not on calcineurin inhibitors (CNIs) and a diagnosis of osmotic nephropathy from the sucrose containing IVIG was made. He was treated symptomatically and did not require dialysis, allograft function returned to baseline values over the next 2 weeks. It has been suggested that nephrotoxicity of IVIG is related to osmotic insult and proximal tubular damage caused by the sucrose or other sugar stabilizing agents of some IVIG forms. It is characterized by marked proximal tubular cell swelling and vacuolization and lumen narrowing and occlusion. The pathological changes of extensive vacuolization and cellular swelling in Tx recipients can mimic CNI nephrotoxicity; in our case the patient was not on such medication. With increasing use of IVIG in recipients of kidney transplantation, physicians should be aware of such a complication and the use of IVIG formulations that do not contain sucrose or other sugar stabilizers, although more expensive, may be safer.

RENAL DISEASE IN FAMILIAL DYSAUTONOMIA: TWO CASE REPORTS OF SUCCESSFUL TRANSPLANTS

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Familial dysautonomia (FD), a rare autosomal recessive disorder nearly exclusive to individuals of Ashkenazi Jewish heritage, affects the development and survival of sensory and autonomic neurons and leads to a spectrum of systemic manifestations. Renal disease, a common but underexplored complication of FD, likely occurs in the context of cardiovascular lability and chronic volume depletion. Forty percent of patients with FD reach at least stage 3 CKD by age 20, and >75% progress to stage 3 CKD or beyond by age 35. Renal replacement therapy with conventional HD is often complicated by intradialytic hypotension; the average time spent on dialysis is only 9 months. To date, there have been 3 successful kidney transplants in patients with FD. We present our experience with 2 of these patients.

Both patients had FD diagnosed at birth and progressed to ESRD. They underwent kidney transplantation at ages 27 and 16, after 3 months of intermittent hemodialysis (IHD) and 1 month of continuous veno-venous hemodialysis (CVVHD), respectively. Issues of blood pressure lability in the perioperative period, namely orthostatic hypotension and supine hypertension, were prominent but manageable. Both patients have done well since transplant, with maintenance of good graft function at 14 and 18 months post-transplantation, respectively. Symptomatic and functional improvements have included lower baseline blood pressures and increased sensitivity to antihypertensive agents.

As survival rates are improving for the FD population as a whole, and more patients are now reaching adulthood, renal disease is becoming increasingly recognized. We believe that our experience reflects the potential for interventions such as kidney transplantation to improve outcomes and quality of life in FD-related chronic kidney disease. We also conclude that in light of the apparent increase in mortality on dialysis for FD patients, transplantation provides a better alternative.

AFRICAN AMERICAN RACE & MALE SEX AS RISK FACTORS FOR SIROLIMUS ADVERSE EFFECTS IN RENAL TRANSPLANT RECIPIENTS

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We discuss one transplant center's experience with SRL and high rate of discontinuation in renal transplant recipients due to various adverse effects; in particular pulmonary complications.

We performed retrospective chart review of patients (pts) receiving SRL & reviewed pts age, gender, race, serum creatinine, reasons for discontinuation, dosage & trough levels at time of discontinuation & duration for adverse effects to occur.

Of 34 pts receiving SRL, 14 (41%) had to be discontinued. Average age of pts was 49 years (34 –72). All were > 1 year post transplant. 11 pts were switched to SRL due to chronic transplant rejection or CNI toxicity while 3 were started de novo on SRL. Average dose of SRL at onset of adverse reaction was 3.8 mg & average trough level was 11.2 ng/ml (within therapeutic range). 8/14 (57%) were males, 8/14 (57%) were African-Americans (AA), 12/14 (85%) were DD & 2/14 were LD recipients. Mean serum creatinine at onset of side effects was 2.68 mg/dl. Mean treatment time was 12 months. Most common adverse event was pulmonary toxicity. 5/14 (35%), all males of which 3 were AA (60%), had pulmonary complications including 3 pts with bilateral pulmonary infiltrates, 1 alveolar hemorrhage & 1 BOOP. 3/14 (21%) had hyperlipidemia, 2 (14%) impaired wound healing, 1 liver enzyme elevation, 1 lymphocele, 1 discontinued due to failed transplant & 1 stopped due to financial limitations. Adverse events rapidly resolved with SRL discontinuation with exception of patient with BOOP.

Temporal relationship between SRL exposure and onset of patient's symptoms suggest that SRL was cause of adverse effects more commonly among DD recipients. These results not only reinforce that male sex continues to be a major risk factor for pulmonary complications of SRL but AA race is also a risk factor. High discontinuation rate (41%) of SRL in our patients indicates that clinicians must remain vigilant to its potential complications especially in males and AA patients.

SEGMENTAL PARASIS, A RARE PRESENTATION OF VARICELLA ZOSTER IN A KIDNEY TRANSPLANT PATIENT

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Disseminated Varicella Zoster Virus (VZV) infection is a rare but potentially life threatening complication in renal transplant patients. Segmental limb paresis typically occurs in 2 to 3 weeks after the herpetic rash development and has been described in 2-5% of patients with VZV infection. However Herpes Zoster sciatica with paresis preceding the skin lesion is extremely rare. We report a case of disseminated VZV in a renal transplant recipient that presented with segmental limb paresis preceding skin rash development.

A 67 year old kidney transplant patient was admitted with right lower limb pain accompanied by right foot weakness of two weeks duration. On physical examination movements of the right hip were limited and painful. He exhibited 2/5 motor weakness in his right foot. Results of routine laboratory studies were normal. An MRI of the lumbar spine showed enhancement of the right spinal L5 nerve root with right L5-S1 foraminal protrusion and surrounding enhancement. EMG corroborated the MRI findings and showed involvement of the right L5 nerve root. Two days later he developed disseminated 1-3mm purpuric papules with overlying vesicles on his torso, upper and lower limbs. Tzanck smear from the skin lesions and VZV PCR of the spinal fluid confirmed the diagnosis of disseminated VZV. He was treated with intravenous and oral acyclovir. The patient showed marked functional recovery during his three months follow up visit.

Segmental paresis is a rare complication of VZV infection. Its pathogenesis is unclear. An inflammatory response triggered by the viral spread may be the cause. Diagnosis depends upon including it in the differential when evaluating a patient with acute painful muscle weakness. EMG is useful for the correct diagnosis. The treatment includes pain medications, physical therapy and antiviral treatment

Recognition of segmental paresis as a presentation of VZV infection in kidney transplant patients is important to avoid unnecessary testing and to initiate therapy promptly. To our knowledge this is the first case report of disseminated VZV infection in kidney transplant patients that presented with Zoster paresis preceding the skin lesion.

POSTERIOR REVERSIBLE ENCEPHALOPATHY (PRES) RELATED TO TACROLIMUS (TAC): A CASE REPORT

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43 year old white female post heart transplant in 2001 secondary to postpartum cardiomyopathy on immunosuppression with TAC and history of calcineurin inhibitor induced End Stage Renal disease presented with sudden onset of agitation and generalized tonic-clonic seizures occurring within 1 hour after dialysis. Review of systems positive for high fever, vomiting and recent admission for line sepsis. Past history is also significant for insulin-dependent diabetes and hypertension controlled with Labetalol, Clonidine.

Physical examination revealed temp 103, BP 170/90mm Hg and HR 117/mt. Patient appeared somnolent, confused and hence intubated. Air entry reduced bilaterally and reflexes were brisk. Left internal jugular permacath was intact with clean exit site. Labs showed WBC 11.1, Hb 10.1, platelet count 121, sodium 141, K 4.1, bicarbonate 21, BUN 17, Cr 3.4, prograf level 6.3. Treatment initiated with loading dose of dilantin, broad spectrum antibiotics and antivirals. Permacath was removed suspecting line sepsis. CSF studies, CT head, Echo and USS liver were unremarkable. Patient continued to remain somnolent, febrile and markedly hypertensive for next 2 days requiring labetalol drip, but was seizure free. Blood and CSF cultures were negative. Due to absence of clinical improvement, non-contrast MRI brain was done that demonstrated subcortical white matter lesions in right posterior lobe and pons suggesting the diagnosis of PRES. Intravenous TAC was immediately discontinued and cyclosporine started, to which a remarkable response was noticed. Patient became more awake and alert within few hours and was extubated the next day.

This case aims to draw attention to consider PRES as differential diagnosis in solid organ transplant recipient on TAC or cyclosporine presenting with following symptoms: headache, mental status changes, focal neurological deficits, and/or hypertension. PRES can occur with therapeutic levels of these drugs. Mostly, PRES is reversible by reducing the dosage or withholding the drug for a few days. Early recognition is of paramount importance for prompt control of blood pressure or removal of precipitating factors and treatment of seizures.

SEVERE REVERSIBLE RENAL ALLOGRAFT FAILURE FROM ANTI-MICA ANTIBODIES

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Presence of allo-antibodies against minor antigens like MHC class I related chain A (MICA) may cause reduced allograft survival. However acute allograft rejection from Anti-MICA allo-antibodies is rare.

We describe a case of a 66 year old man with a T cell PRA of 0%, B cell PRA 73% and no donor specific antibody. Patient received a renal transplant from a 49 year old deceased donor. Prior to transplant, the patient serum was examined by flow cytometric crossmatch and was found to be compatible to the donor. The patient was treated with standard immunosuppression including anti-lymphocyte globulin, tacrolimus, mycophenolic acid, and a steroid taper. Post operatively patient had immediate graft function. Late on day 2, patient had abrupt decrease in urine output and he became anuric. Renal ultrasound and nuclear renal scan showed preserved blood flow and no evidence of either hydronephrosis or urinary leak. Patient was treated with urgent plasmapheresis and a biopsy performed showing minimal pathological changes other than peritubular C4D staining. Patient was treated with a combination of high dose IVIG, plasmapheresis, and Rituximab. On day 9 the urine output started to increase and hemodialysis was stopped. Repeat biopsy showed worsened peritubular C4D staining but no significant fibrosis. By day 11 his urine output normalized and by day 15 serum creatinine improved to 1.2 mg/dl, 2 months later his creatinine remains at a baseline of 1.0. Patient's serum was found to demonstrate anti-MICA antibodies by Luminex beads assay.

We conclude that non-HLA antigens should be considered as a source of humoral rejection in crossmatch-compatible renal transplants with graft dysfunction. Once identified, early treatment may allow for significant recovery of allograft function even after a prolonged period of anuria.

DE NOVO IMMUNOTACTOID GLOMERULOPATHY (ITG) IN A RENAL ALLOGRAFT: A CASE REPORT.

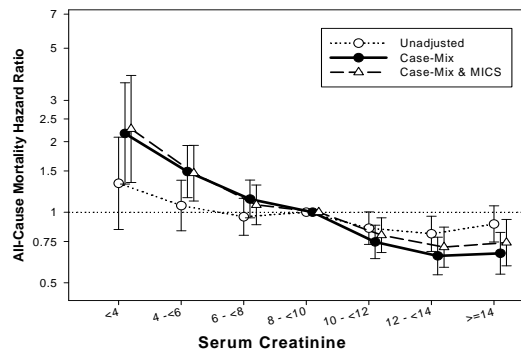
Salman Singapuri and Sudha Tata, Emory University School of Medicine, Division of Hypertension and Nephrology, Atlanta, Ga. Recurrent glomerular diseases are key causes of graft dysfunction after renal transplantation. We report an interesting case of a patient with focal segmental glomerulosclerosis (FSGS) who underwent a living donor renal transplant and subsequently developed de novo ITG. Case: A 50 yo patient presented to our clinic after having an uncomplicated course of a renal allograft 13 years ago. He was in good health when he was noted to have subnephrotic range proteinuria (1225mg/day) and a rapid rise in his S Cr to 2.9mg/dl (baseline S Cr 1.6 mg/dl) after suffering pneumonia. A biopsy of the renal allograft was warranted which interestingly revealed de novo ITG consisting of numerous subepithelial and mesangial microtubular paraprotein deposits. Additional battery of tests including a SPEP and UPEP revealed several abnormal protein bands detected in the gamma globulin region and a faint monoclonal free kappa light chain respectively. The patient was referred to an oncologist and a bone marrow biopsy was performed, showing myeloblasts (6%) consistent with myelodysplastic syndrome (MDS). In the interim, subsequent testing revealed transaminitis and a positive hepatitis B e antigen, likely from reactivation. Fulminant hepatic failure gradually ensued and the patient succumbed to his illness, without having an opportunity to be treated for ITG and MDS with chemotherapy. To the best of our knowledge and after extensive literature review, de novo ITG after renal allograft has only been reported with cytomegalovirus but not with FSGS.

ASSOCIATION OF PRE-TRANSPLANT SERUM CREATININE AS A POTENTIAL MUSCLE MASS SURROGATE AND 5-YEAR PATIENT AND GRAFT SURVIVAL IN 10,090 HEMODIALYSIS PATIENTS

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Background: Larger lean body & muscle mass may be associated with greater survival in hemodialysis (HD) patients (pts) but its association with post-transplant outcomes is not known. We hypothesized that a higher pre-dialysis serum creatinine, a surrogate of muscle mass, in the months prior to transplant is associated with better post-transplant outcomes. **Methods:** After merging the “Scientific Registry of Transplant Recipients” database with DaVita national database of HD pts over 5 yrs (7/01-6/06), we identified 10,090 renal transplant recipients (RTR), in whom time to death or graft failure was calculated. **Results:** Pts were 49 ± 13 yrs old and included 49% women, 45% diabetics & 27% Blacks. The 3-mo averaged creatinine prior to transplant was $10.6 \pm 3/2$ mg/dL. Cox models adjusted for case-mix & “malnutrition-inflammation-cachexia syndrome” (MICS) showed a linear and incremental association with the composite pt & graft survival (see Figure).

Conclusions: If average pre-dialysis serum creatinine is a surrogate of muscle mass in HD pts, larger muscle mass appears associated with better post-transplant outcomes. Trials to examine intervention to improve sarcopenia are indicated in transplant wait-listed pts.



HYPOMAGNESEMIA CAUSING RAPID NEW ONSET TYPE II DIABETES POST TRANSPLANT (NODAT)

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Hypomagnesemia is a frequent complication in the early post transplantation period and is particularly associated with the use of calcineurin inhibitors (CNI). Mg depletion has shown some link in contributing to a post-receptor insulin resistance. We did a study to look at the relationship of hypomagnesemia and development of rapid NODAT(in the first 3 months).

Single-center chart analysis was conducted to determine the onset of DM II in the first 3 months of renal transplant. Eligibility criteria included patients who underwent living transplants at our center and able to collect relevant data. Exclusion criteria are patients with prior history of DMII, multiple transplants, Hepatitis C patients & DDRTx. Of the 38 transplants done total at our center in the last 2 years, 16 patients met the above criteria. All patients were on triple (mycophenolate sodium, tacrolimus and prednisone) drug protocol. 3 patients developed new onset of DM II in the first 3 months compared 13 patients who had no evidence of DMII. We compared HgA1C, Mg levels, Ca, CNIs, BMI and age in both groups. Table below shows the significant values.

Means	Controls	Cohorts	P values
HgA1C	5.1	8.1	<0.0001
1 month trough tacrolimus(ng/ml)	10	13.4	<0.1128
3 month trough tacrolimus(ng/ml)	9.9	15.7	<0.0087
1 month Mg(mg/dl)	1.7	1.3	<0.0065
2 month Mg(mg/dl)	1.7	1.4	<0.0286
3 month Mg(mg/dl)	2.1	1.3	<0.0338

Along with CNI, and other known risk factors, we conclude that hypomagnesaemia is potential risk factor of rapid NODAT in patients evaluated within the first 3 months of the transplant. Based on our data, it might be prudent to maintain Mg levels >1.5mg/dl.

BONE DISEASE IN LONG TERM POST RENAL TRANSPLANT PATIENTS: SINGLE CENTER EXPERIENCE

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Bone disease after renal transplant is a cause of significant morbidity in this population. In this report we present the histomorphometric pattern of bone disease in 14 patients at our institution.

Bone biopsies were performed on patients because of abnormal DEXA scan. After double tetracycline labeling, bone biopsies were done. Patients were long term post transplant (2.8-30 years). 28% had diabetes and 71% were females. All were on maintenance steroids, 4 received FK506 and cellcept whereas 5 were on cyclosporine. Cumulative steroid dosage was 43 +/- 44 grams. 36% of patients were on either bisphosphonates or Vitamin D and 57% on phosphate binders. 5 patients (35.7%) had documented fractures since transplant. All patients had low bone turnover on histomorphometric analysis.

Histomorphometric Data	Patients	Normal
Bone volume/tissue volume (%)	17.50 ± 2.48	16.8-22.9
Trabecular thickness (µm)	82.56 ± 9.36	99-142
Osteoid volume/bone volume (%)	3.68 ± 0.64	0.57-6.0
Osteoblast surface/bone surface (%)	0.29 ± 0.06	0.2-3.5
Erosion surface/bone surface (%)	0.97 ± 0.21	0.1-5.69
Mineralizing surface/bone surface (%)	2.22 ± 0.53	4.3-12.0
Bone formation rate/bone surface (mm ³ /cm ² /yr)	0.87 ± 0.18	1.80-3.8
Mineralization lag time (days)	86.6 ± 16.23	<50
Activation frequency (yr ⁻¹)	0.18 ± 0.03	0.49-0.72

We demonstrate increased prevalence of low turn over bone disease in long term post transplant recipients.