

**ANTINEUTROPHIL CYTOPLASMIC AUTOANTIBODY  
SPECIFIC FOR PROTEINASE 3 IN A PATIENT WITH SHUNT  
NEPHRITIS INDUCED BY STAPHYLOCOCCUS EPIDERMIDIS.**

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Shunt Nephritis is an immune complex mediated glomerulonephritis caused by chronically infected ventriculoatrial or rarely ventriculoperitoneal shunt inserted for treatment of hydrocephalus.

A 31 year old female with ventriculoatrial shunt for hydrocephalus presented with progressive nausea, vomiting, headache and fever. She reported intermittent fever, proteinuria, microhematuria for the past year with positive blood culture for staphylococcus epidermidis one year ago. Repeated blood cultures (6/6) drawn during current hospitalization were positive for staphylococcus epidermidis. CT Head showed prominent ventricles. Laboratory data showed hematuria, proteinuria (1.6g/ 24hr), anemia, thrombocytopenia, renal dysfunction and hypocomplementemia with positive ANA, C-ANCA and Proteinase 3 antibody (PR3-ANCA). Renal US and TEE normal. Renal biopsy showed basement membrane splitting with subendothelial electron dense deposits consistent with MPGN 1. Patient was started on intravenous antibiotics and was referred for shunt removal.

Glomerulonephritis may develop in 0.7- 2% of the infected VA shunts between 2 months to several years after shunt placement. Common pathogens include Staphylococcus epidermidis (70%) and Staphylococcus aureus. Renal manifestation includes microscopic hematuria (90%), proteinuria with nephrotic syndrome. Serology reveals decreased complement levels, high rheumatoid factor titers, cryoglobulinemia and positive PR3- ANCA. Renal histology reveals MPGN type 1 in 60% and non IgA mesangial proliferative GN in 33% cases. Treatment involves antibiotic therapy and prompt removal of the infected shunt with complete renal recovery in more than 50% of cases.

Only three other cases of shunt nephritis with positive PR 3 antibody (specific for Wegner's granulomatosis) have been reported in the literature. It has been postulated that the PR3 –ANCA is induced by continuous infection with pathogenic bacteria and shunt removal decreases the titer. Failure to recognize shunt nephritis and delay in removal of infected shunt worsens renal outcome.