

AORTIC STIFFNESS IN GLOMERULONEPHRITIS CHRONIC KIDNEY DISEASE COMPARED TO OTHER CKD ETIOLOGIES

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Aortic stiffness, estimated by augmentation index (AI), increases with increasing severity of CKD. Little is known about the effect of different CKD etiologies on aortic stiffness. We sought to compare the AI in glomerulonephritis (GN) CKD to the non-GN causes of CKD.

We retrospectively reviewed the medical records of 283 patients who had AI measured with applanation tonometry in our renal clinic. They are subgrouped into: No CKD (n=110), GN(glomerulonephritis)-CKD (n=62) and non-GN-CKD (n=111). The non-GN-CKD group included 22 diabetic nephropathy, 30 hypertensive nephrosclerosis and 59 other causes of CKD. We compared continuous variables by gender using Wilcoxon rank sum test, and categorical variables using Chi-square tests. Linear regression analysis was used to compare the AI between patients with GN-CKD and those with non-GN-CKD while adjusting for age, gender, history of diabetes, hypertension and smoking.

AI was not significantly different between patients with GN-CKD and non-GN-CKD while adjusting for patient characteristics (average effect of GN on AI is 0.3 units increase, 95% confidence intervals [CI] -3 to 3.5, $P=0.87$). When including all 283 patients, females had significantly higher AI compared to males (median AI of 28 [quartiles 22, 34] vs. 18 [12, 26], $p<0.001$) despite having significantly higher GFR than males (median GFR of 67.2 [quartiles 38, 88] vs. 56.2 [29, 76], $p=0.016$). Both female and male patients were similar in their clinical characteristics (including peripheral blood pressure [BP]) except females were significantly shorter (median of 160 cm [quartiles 155-165] vs. 175 cm [170-180], $p<0.001$).

Our study showed no difference in AI between GN-CKD and non-GN-CKD patients. The higher AI in our female population compared to male population despite better GFR and similar peripheral BP needs further evaluation in terms of optimal BP targets, impact of different pharmacologic agents and methods of assessing 'true' BP control.