Diabetic nephropathy induced impaired aortic function is not mediated by mean arterial pressure and its determinants

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Abstract

Introduction: Impaired aortic function is a core mechanism in the development of uremic cardiomyopathy. Herein, we investigated whether mean arterial pressure and its determinants including systemic vascular resistance (SVR) and cardiac output (CO) mediate the impact of diabetic nephropathy (DNP) and hypertensive nephropathy (HNP) on aortic function

Methods: This multi-ethnic study included 67 non-dialysis and 48 dialysis patients. Aortic function measures comprised PP, SBP, central pulse pressure, central systolic blood pressure, proximal aortic stiffness as estimated by the inverse of total arterial compliance (invTAC), carotid-femoral pulse wave velocity, backward wave pressure and forward wave pressure. The calculated power of the study was 0.997 based on α=0.05.

Results: HNP (53.9%), DNP (32.2%), glomerulonephritis (19.1%) and HIV associated nephropathy (7.8%) comprised the major CKD etiologies. Concurrent HNP and DNP was present in 31.1% of the patients. Patients with compared to without concurrent HNP and DNP experienced more frequent cardiovascular disease (43.2% versus 14.9%, p=0.01) and impaired aortic function (p=0.006-0.05 for 5 of the measures). DNP was independently associated with each aortic function measure (p<0.001-0.02). HNP was not directly related to aortic function (p>0.05). Other covariates that were consistently associated with impaired aortic function measures except for invTAC, included MAP (p<0.001-0.01) and its determinants. MAP and CO x SVR did not account for the potential effect of DNP on any aortic function measure (0.02-(-)7.3%). Dialysis status did not impact any of the identified relationships (interaction p>0.05).

Conclusion: This study suggests that reducing MAP by decreasing volume overload and/or SVR through fluid intake restriction, diuretic therapy and antihypertensive agents or vasodilators may improve aortic function in the overall CKD population. However, these interventions are unlikely to reverse impaired aortic function that is induced by DNP. Whether increased arterial medial calcification associated with diabetes and DNP explain our findings merits further study.