Renal dysfunction caused by aortic stiffening

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Aortic stiffness predicts the progressive decline of the glomerular filtration rate (GFR). However, the underlying pathophysiological mechanism remains to be determined. Recent evidence has shown a close link between aortic stiffness and the bidirectional (systolic forward and early diastolic reverse) flow characteristics. We tested whether the aortic stiffening-induced renal dysfunction is attributable to altered central flow dynamics.

In 222 patients with hypertension, Doppler velocity waveforms were recorded at the proximal descending aorta to calculate the reverse/forward flow ratio. Tonometric waveforms were recorded to measure the carotid-femoral (aortic) and carotid-radial (peripheral) pulse wave velocities, to estimate the aortic pressure from the radial waveforms, and to compute the aortic characteristic impedance. In addition, renal hemodynamics was evaluated by duplex ultrasound.

The estimated GFR was inversely correlated with the aortic pulse wave velocity, reverse/forward flow ratio, pulse pressure, and characteristic impedance, whereas it was not correlated with the peripheral pulse wave velocity or mean arterial pressure. The association between aortic pulse wave velocity and estimated GFR was independent of age, diabetes mellitus, hypercholesterolemia, and antihypertensive medication. However, further adjustment for the aortic reverse/forward flow ratio and pulse pressure substantially weakened this association. After the adjustment, the reverse/forward flow ratio emerged as the strongest determinant of estimated GFR (P=0.001). A higher aortic reverse/forward flow ratio was also associated with lower intrarenal forward flow velocities.

An increase in aortic flow reversal (ie, retrograde flow from the descending thoracic aorta toward the aortic arch), caused by aortic stiffening and impedance mismatch, reduces antegrade flow into the kidney and thereby deteriorates renal function