Influence of arterial stiffness on microcirculation of the brain and kidney

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Stiffening of the large arteries is related to an increased risk for cardiovascular disease. Recent studies have associated pulse wave velocity (PWV), a measure of arterial stiffness, with earlier subclinical damage of vital organs including the brain and kidney as well as the heart. An interrelationship between asymptomatic cerebral lacunar infarction and albuminuria has also been demonstrated in hypertension and with aging, although the underlying pathogenesis of this cerebro-renal connection remains unknown. It could be hypothesized that both of these cerebral and renal abnormalities are attributed to microvascular damage resulting from similar hemodynamic alterations. In the present study, urinary albumin/creatinine ratio (ACR), PWV, and 24-hour ambulatory blood pressure were measured, and cerebral lacunar lesions assessed on magnetic resonance imaging in a population of 351 general adults aged ≥50 years. Lacunar infarction was present in 86 subjects (25%), and the subjects with lacunae had a higher median urinary ACR than those without (20.6 versus 14.6 mg/g·Cr, P=0.004). The positive association between the presence of lacunae and urinary ACR remained significant in a logistic model that adjusted for covariates including age, gender, and 24-hour blood pressure (P=0.04); the odds ratio for lacunae was 1.32 for each 1SD increase in urinary ACR. However, when PWV was added further to this model, increased PWV but not urinary ACR was associated with lacunae. There was a significant interaction between urinary ACR and PWV with respect to lacunae; only

subjects with a high PWV above the median showed an independent association between urinary ACR and the presence of lacunae (P=0.03). These results indicate that microvascular damage in the brain (lacunar infarction) and kidney (albuminuria) is interrelated in the general population, and large artery stiffening plays a pivotal role in this cerebro-renal connection. This finding may be explicable by the hypothetical hemodynamic mechanism that large artery stiffening increases pulsatile pressure and flow stresses, which extend deeply to the vulnerable microcirculation in vasodilated organs such as the brain and kidneys, and thereby lead to the microvascular damage progression.