



Conference Abstract

P.21 Albuminuria Intensifies the Relationship Between Urinary Sodium Excretion and Central Pulse Pressure: The Wakuya Study

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Keywords

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ABSTRACT

Objectives: Central pulse pressure (cPP) is responsible for vital organ hemodynamics [1,2], and its monitoring is important for cardiovascular disease prevention [3]. Excess sodium intake and (micro)albuminuria, a manifestation of renal microvascular damage, are also known as strong predictors of cardiovascular disease [4,5]. We sought to investigate the cross-sectional relationships among dietary sodium consumption, albuminuria and cPP in the general population.

Methods: Subjects were 933 apparently healthy adults in Wakuya town, Miyagi, Japan (mean age, 56 ± 10 years). Radial pressure waveforms were recorded with applanation tonometry to estimate mean arterial pressure (MAP), cPP, forward and backward pressure amplitudes, and augmentation index. Urinary sodium/creatinine ratio (UNaCR) and albumin/creatinine ratio (UACR) were measured in spot urine samples.

Results: Median values of UNaCR, UACR and cPP were 139 (interquartile range, 89–205) mEq/g, 5 (4–11) mg/g and 38 (33–45) mmHg, respectively. Both UACR and UNaCR were positively correlated with cPP, even after adjusted for MAP ($p < 0.001$). Moreover, UACR and UNaCR had a synergistic influence on increasing cPP, which was independent of age, sex, estimated glomerular filtration rate, hyperlipidemia and diabetes (interaction $p < 0.05$). A similar synergistic influence between UACR and UNaCR was found on the forward but not backward pressure amplitude or augmentation index. The overall results were not altered on replacement of UACR with the existence of chronic kidney disease.

Conclusions: (Micro)albuminuria strengthens the positive association between urinary sodium excretion and central pulse (and systolic forward) pressure. Excess sodium intake may magnify cardiovascular risk through widening aortic pulsatile pressure, particularly in the presence of concomitant chronic kidney disease.

REFERENCES

- [1] Townsend RR, Chirinos JA, Parsa A, Weir MA, Sozio SM, Lash JP, et al. Central pulse pressure in chronic kidney disease. *Hypertension* 2010;56:518–24.
- [2] Hashimoto J, Ito S. Central pulse pressure and aortic stiffness determine renal hemodynamics. *Hypertension* 2011;58:839–46.
- [3] Vlachopoulos C, Aznaouridis K, O'Rourke MF, Safar ME, Baou K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: a systematic review and meta-analysis. *Eur Heart J* 2010;31:1865–71.
- [4] Matsushita K, van der Velde M, Astor BC, Woodward M, Levey AS, de Jong PE, et al. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: a collaborative meta-analysis. *Lancet* 2010;375:2073–81.
- [5] Hashimoto J, Aikawa T, Imai Y. Large artery stiffening as a link between cerebral lacunar infarction and renal albuminuria. *Am J Hypertens* 2008;21:1304–9.

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