URINARY SODIUM EXCRETION PREDICTS ARTERIAL STIFFNESS IN NEWLY DIAGNOSED HYPERTENSIVE PATIENTS: A 6-YEAR FOLLOW-UP STUDY

ACC Poster Contributions
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Background: Impaired kidney function and renal sodium handling are crucial in the progression of hypertensive disease. Arterial stiffness has proven predictive value for cardiovascular events. We investigated whether baseline 24 hour urinary sodium excretion is associated with future aortic stiffness in hypertension.

Methods: We prospectively studied for 6 years, 163 white, non-diabetic patients (52±10 years, 98 men, 59 smokers, body mass index=28.8±5.4Kg/m2), with newly diagnosed essential hypertension. Patients underwent 24-hour ambulatory blood pressure (BP) monitoring, and carotid-femoral pulse wave velocity (PWV) measurement (Complior SP device). Metabolic profile, estimated creatinine clearance (Cockfort-Gault formula) (eGFR) and sodium excretion in a 24h urine collection were also assessed at both baseline and last outpatient clinic visit (follow-up: 4.5±0.89 years). Subjects were given optimal antihypertensive therapy supplemented by vasoactive drugs where appropriate. The spectrum of drugs applied in the last 6-month period consisted of: renin-angiotensin system (RAS) inhibitors (49%), diuretics (25%), calcium-channel blockers (20%), beta-blockers (13%), statins (16%) and antiplatelet agents (18%).

Results: In the follow-up period daily sodium excretion and PWV increased by 47±43mmol (113±51 vs.123±46mmol) and 1.83±1.37m/s (7.9±1.5 vs. 9.6±1.5m/s) respectively, while eGFR was decreased by 17±16ml/min. Baseline glucose and low-density lipoprotein levels were 87±14 and 142±36mg/dl respectively, while in the follow-up period 24h systolic and diastolic BP decreased by 12.7±9.1 and 8.2±6.2mmHg (130±12 vs.122±12 and 80±9 vs. 75±8mmHg) respectively. In a stepwise regression model (R2 adjusted=37%) significant predictors of PWV at follow-up were age (beta=0.29, p<0.001), baseline PWV (beta=0.44, p<0.001), baseline 24h-sodium excretion (beta=0.15, p=0.015) and treatment with RAS-inhibitors (beta=0.17, p=0.009).

Conclusions: Increased sodium excretion is associated with future increased aortic stiffening in hypertension and the absence of RAS inhibitors in the antihypertensive regimen further contributes to this phenomenon.