ALTERATION OF ALDOSTERONE RESPONSE TO SALT OVERLOAD THROUGH CHRONIC KIDNEY DISEASE IN PATIENTS WITH HYPERTENSION

ACC Poster Contributions
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Background: Past investigations indicated that salt overload suppresses aldosterone release. Under influence of chronic kidney disease (CKD), however, it is still unclear in hypertensive patients. Therefore, we examined the impact of CKD on response of plasma aldosterone concentration (PAC) to salt intake in hypertensive patients with coronary heart disease (CAD).

Methods: Present population was 48 hypertensive patients (68±13 yo) with treated ischemic heart disease and without taking diuretics such as anti-aldosterone agent. CKD was defined as estimated glomerular filtration rate (eGFR) less than 60ml/ min/1.73m2, and subjects were divided into 2 groups; 28 CKD(-) and 20 CKD(+) patients. PAC was evaluated using venous blood sampling at rest. Daily salt intake (g/day) was calculated using formula of the Japanese society of hypertension.

Results: 1) Between CKD(-) and CKD(+) groups, there is no difference in clinical back grounds including age, sex, number of taking antihypertensive drug, daily salt intake, left ventricular ejection fraction and BP under medications. 2) In CKD(-) group, daily salt intake had inverse relationship with PAC (r=-0.466), but not significant in CKD(+) group. 3) In patients with excess salt intake (≥9g/day), PAC was higher in CKD(+) group than in CKD(-) group.

Conclusions: We concluded that CKD lead to the possibility of coexistence of excess salt overload and high PAC, and induces harmful pathophysiological circumstance in hypertensive patients with CAD.