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Expression of Renal Tubular Transporters in Urinary Exosomes from Patients with Acute and Chronic Hypokalemia

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Objectives: Urinary exosomes has been used as an index of renal tubular transporter expression in primary aldosteronism or renal tubular disorders. Urinary exosome analysis of renal sodium (Na^+) and potassium (K^+) associated transporters in hypokalemia patients has not been studied. Our purpose to evaluate the expression of renal Na^+ and K^+ associated transporters in patients with acute and chronic hypokalemia.

Methods: We have collected timely spot urine from thirty-one hypokalemia patients. Urinary exosomes were isolated by ultracentrifugation method. Membrane transporters abundance including NaCl cotransporter (NCC), phospharylated NaCl cotransporter (pNCC), Na^+-hydrogen exchanger 3 (NHE3), Na/K/2Cl cotransporter (NKCC2), epithelial Na^+ channel β (ENaCβ), and renal outer medullary K1 channel (ROMK) were analyzed by immunoblotting.

Results: In Gitelman syndrome (n=11) patients, immunoblotting of NCC and pNCC abundance significantly decreased corresponding to NCC mutation compared to healthy control. Na^+ associated transporters abundance of NHE3 and ENaCβ significantly increased while K^+ associated transporters abundance of ROMK significantly increased. Expression of transporters in urinary exosomes was consistent with immunofluorescence staining of kidney biopsy. In thyrotoxic periodic paralysis (n=9) patients, there were no significant change of transporter abundance in acute hypokalemia phase compared to recovery phase. Patients with Sjogren syndrome with distal renal tubular acidosis (distal RTA, n=4), aldosterone producing adenoma (APA, n=3), and gastrointestinal disorders (n=4) all exhibited significantly increased abundance of NHE3, ENaCβ and ROMK. Of note, there were also significant increased NCC abundance in APA patients and increased NKCC2 abundance in distal RTA patients compared to healthy control.

Conclusions: Urine exosomics could be used to evaluate the renal Na^+ and K^+ associated transporters expression in hypokalemia. Acute hypokalemia could not affect expression in Na^+ or K^+ associated transporters. Chronic hypokalemia could activate both upstream NHE3 and downstream ENaCβ with concomitant increased expression of ROMK in response to flow.