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Urine Chloride level and Progression of Chronic Kidney Disease

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Objectives: Urine chloride is regulated by the kidney transport channels and its low level can be caused by tubular defects. In addition, tubuloglomerular feedback is the physiologic mechanism to regulate glomerular filtration rate, which is mainly driven by chloride level in the distal tubules. However, little attention has been paid to urine chloride as a biomarker of clinical outcome. Here, we studied the relationship between urinary chloride level and CKD progression.

Methods: Data were retrieved from the KoreaN Cohort Study for Outcome in Patients With Chronic Kidney Disease cohort. Among 2,238 participants, a total of 2,086 patients were eligible for the analysis after excluding patients with missing data for baseline urinary chloride level (n=93) and follow up (n=59). Patients were categorized into 3 groups according to baseline tertiles of urinary chloride excretion using the first voided samples: < 77, 77-116, and ≥ 117 mEq/L. The study endpoint was a composite of a ≥50% decrease in eGFR from baseline values, or ESRD.

Results: At baseline, urinary chloride concentration positively correlated with eGFR and inversely with proteinuria. During a median follow-up of 3.3 years (7452 person-year), 565 (27.1%) participants reached the renal endpoint. CKD progression events occurred in 281 (40.2%), 216 (30.7%), and 68 (9.9%) patients in the lowest, middle, and highest tertile groups (P<0.001), respectively. Compared to the lowest tertile, the highest tertile was associated with significantly lower risk of adverse renal outcome in multivariable models after adjustment of confounders (HR, 0.73; 95% CI, 0.49-0.89; P = 0.007). When urine chloride was treated as a continuous variable, a 10 mEq/L increase in urine chloride was associated with a 4.6% lower risk of CKD progression.

Conclusions: This hypothesis generating study suggests that urine chloride may be used as a biomarker of CKD progression.