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만성 신부전 동물모델에서 신장의 소디움 및 포타시움 운반체의 적응기전

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Adaptive Mechanism of Sodium and Potassium Transporters in Chronic Renal Failure Rat Kidneys

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Background : In chronic renal failure (CRF), residual nephrons can magnify their excretion of sodium and potassium. However, long- term adaptive mechanism of renal tubular transporters with a diminishing glomerular filtration rate (GFR) has not been fully understood.

Methods : We performed immunoblotting and immunocytochemistry, thus investigating the sequential changes of expression of major renal sodium and potassium transporters in rats from 4 weeks to 12 weeks after 5/6 nephrectomy (Nx).

Results : CRF rats had lower levels of GFR, and higher levels of urinary sodium and potassium excretion throughout the whole periods after 5/6 Nx. At 4 weeks, the density of NKCC2, NCC, ENaC- α , and ENaC- γ in CRF rats significantly increased (477%, 222%, 451%, and 435% of control, respectively) although the expression of NHE3 and SGLT1 did not altered. In contrast, protein of NKCC2 and NCC markedly decreased at 12 weeks (55.4% and 30.8%, respectively), and NHE3 and SGLT1 also decreased at 12 weeks (48.4% and 24.6%, respectively). The activity of ENaC- α significantly increased during the whole period. Urinary potassium excretion rate was well- correlated with urinary urea or sodium excretion rate, although immunoblotting and immunostaining of ROMK and Na- K- ATPase showed a gradual decrease in the late stages of CRF.

Conclusion : We suggest that this sequential decrease of major renal sodium transporters except ENaC may be a main mechanism for increased urinary sodium excretion, and that increased urea excretion rates or distal sodium delivery may be a major explanation for increased urinary potassium excretion in CRF rats.

Key Words : 만성신부전, 소디움, 포타시움
Chronic renal failure, Sodium, Potassium