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Altered Expression of Aquaporin Water Channels and Sodium Transporters in Two-kidney, One Clip Hypertension in Rats

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Background : The present study was aimed at determining whether the regulation of aquaporin (AQP) water channels and sodium transporters is altered in the kidney in two-kidney, one-clip (2K1C) hypertension.

Methods : Adult male Sprague-Dawley rats were made 2K1C hypertensive for 1 week. The protein expression of AQPs (AQP1, AQP2, AQP3) and sodium transporters (NHE3, NKCC2, NCC, Na,K-ATPase) was determined in the clipped and contralateral kidneys. The activity of adenylyl cyclase was determined by stimulated generation of cAMP.

Results : The blood pressure was increased significantly in 2K1C rats. The urine volume increased along with impaired urinary concentration, and fractional and total excretion of sodium increased. The protein expression of AQP1-3 was decreased in the cortex/outer stripe of the outer medulla, inner stripe of the outer medulla, and inner medulla in the clipped kidney, while remained unchanged in the contralateral kidney. The expression of NKCC2 and NHE3 was decreased, while that of NCC remained unchanged in the clipped kidney. On the contrary, the expression of these transporters remained unchanged in the contralateral kidney. The adenylyl cyclase activity provoked by arginine vasopressin, sodium fluoride or forskolin was blunted in the clipped kidney, while remaind unaltered in the contralateral kidney. The expression of adenylyl cyclase VI was decreased in the clipped kidney, but not in the contralateral kidney. The protein expression of a1 subunit of Na,K-ATPase was decreased in the clipped kidney, while rather increased in the contralateral kidney.

Conclusion : The downregulation of AQPs and sodium transporters in the clipped kidney may contribute to the salt wasting and urinary concentration defect in 2K1C hypertension.