Increased Expression of Aquaporin Water Channels in Hypothyroid Rat Kidney

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A decreased urinary water excretion is frequently observed in hypothyroidism both in humans and experimental animals. The decrease has been related with various factors including inappropriately elevated arginine vasopressin (AVP), a diminished glomerular filtration rate, and a decreased salt reabsorption in the ascending limb of Henle's loop. However, the detailed mechanisms by which hypothyroidism induces water retention has not been completely understood.

Recent discovery of aquaporin (AQP) water channels has advanced our understanding of water transport and AVP action in the kidney. A series of studies have underscored crucial roles of AQP in the regulation of renal water reabsorption and hence body water balance. For instance, the expression of AQP2 is increased in such conditions association with water retention as in severe congestive heart failure, pregnancy, syndrome of inappropriate antidiuretic hormone, and glucocorticoid-deficiency.

The present study was aimed to determine the molecular mechanisms underlying the water retention associated with hypothyroidism. Male Sprague-Dawley rats (200-220 g) were experimentally induced of hypothyroidism by treatment with methimazole in drinking water (0.04%) for 8 weeks.

In the experimental group, serum concentrations of thyroxine and triiodothyronine were significantly decreased. The expression of AQP2 was significantly increased in the cortex, outer medulla, and inner medulla of the kidney. The expression of AQP1 as well as that of AQP3 was significantly increased in the cortex, though not in the medulla.

The adenylyl cyclase activity provoked by arginine vasopressin, sodium fluoride, or forskolin was blunted in the hypothyroid kidney, while plasma levels of arginine vasopressin were not significantly changed. The increased expression of AQP1-3 channels may in part account for the water retention in hypothyroidism.