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Decreased Expression of Aquaporin-2 Water Channels in the Kidney in Rats Treated with Chlorpropamide

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Background : Whether blood glucose levels may change the regulation of aquaporin (AQP) water channels in the kidney was investigated.

Methods : Male Sprague-Dawley rats were treated with chlorpropamide (40 mg/100 g body weight per day, per oral, for 7 days), and their expression of AQP1-3 and type VI adenylyl cyclase proteins was determined in the kidney.

Results : Following the treatment with chlorpropamide, the blood glucose level was significantly decreased compared with that in the control (64±8 vs 106±7 mg/dL, n=6 each, $p<0.01$). Accordingly, the expression of AQP2 proteins was decreased in the cortex, outer medulla, and inner medulla. The AQP2 targeting was not significantly altered, as evidenced by parallel decreases of its expression in the membrane and the cytoplasmic fractions. No significant changes were observed in the expression of either AQP1 or of AQP3. The protein expression of type VI adenylyl cyclase was not significantly altered.

Conclusion : These results suggest that hypoglycemia attenuates the expression of AQP2 water channels in the kidney.

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Hydronephrosis by an Aberrant Renal Artery

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Ureteropelvic junction obstruction is usually intrinsic and most common in children. Aberrant renal arteries are present in about 30% of individuals. Aberrant renal arteries to the inferior pole cross anterior to the ureter and may cause hydronephrosis.To our best knowledge, there are some papers about aberrant renal arteries producing ureteropelvic junction obstruction, but no report of case in Korea. We describe a 36-year-old woman with right hydronephrosis. Kidney ultrasonogram and excretory urogram revealed right hydronephrosis. Computed tomography angiogram (CTA) and magnetic resonance angiogram (MRA) clearly displayed an aberrant renal artery and hydronephrosis. The patient underwent surgical exploration. For the evaluation of hydronephrosis by an aberrant renal artery, use of CTA and MRA is advocated.