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Changes of URAT-1 in a unilateral ureteral obstruction renal injury model

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Objectives:

Proximal tubular URAT-1 is the most important pathway of uric acid reabsorption, but lack of data in obstructive uropathy. Unilateral ureteral obstruction (UUO) causes progressive renal fibrosis which resembles of chronic kidney disease (tubulopathy) and could compare with non-obstructive kidney. The aim of this study is to find the changes of tubular URAT-1 expression in ureteral obstruction, and to compare GLUT-9 changes via URAT-1 changes after UUO.

Methods:

Male Sprague Dawley rats divided into 4 groups; control, UUO day 1, UUO day 3, UUO day 14. The obstructed ureter was decompressed 24 hours later. 24 hour urine was collected via metabolic cage. Blood samples were obtained during the harvest of kidney (on sacrifice), and Western blot and immunohistochemical stain were performed for measuring tubular URAT-1 and GLUT9 expression.

Results:

24 hr after UUO release showed lowest estimated GFR, urine volume, and body weight and highest urine uric acid and lowest serum uric acid. After 2 wks of UUO release showed largest urine volume and highest serum uric acid and lowest urine uric acid. URAT-1 was highest at UUO then normalized after release 14 days and GLUT-9 was increased at 14 days of release. Non-obstructed kidney showed the opposite feature. Immunohistochemical stain shows similar feature with western blot results. URAT-1 was increased in UUO then decreased by improvement of obstruction, but there was no difference in non-obstructive kidney. GLUT-9 was not changed in UUO rather increased in non-obstructed kidney. Although URAT-1 was increased in UUO, urine uric acid was increased which might be implied that there was compensatory mechanism of GLUT-9 of non-obstructive area.

Conclusions:

URAT-1 is increased in renal tubular injury, and GLUT-9 is increased in the non-obstructed ureter which implies that URAT-1 and GLUT-9 are doing compensatory work in renal tubular injury.