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THE FUNCTION CHANGES IN URETER OF RABBIT WITH NEUROGENIC URINARY TRACT DYSFUNCTION AND THE LVDCC DIFFERENCES EXPRESSION OF URETER

Hypothesis / aims of study

To investigate the functional change of ureter in the early phase of neurogenic urinary tract dysfunction(NUTD) and the expression of L-type voltage-dependent calcium channels (LVDCC) on ureter.

Study design, materials and methods

Thirty Japanese White Rabbits were randomly divided into (NUTD) group (n=10), sham group (n=10) and blank group (n=10). The NUTD group with a spinal cord transaction at the sixth lumbar level and destroyed the sacral cord, Sham group was only bite part of spinous process at the same position, exposed spinal cord but not given a transaction, and blank group without any operation. Video-urodynamic was performed at the second months after operation. After that, taking a ventral incision to expose the left ureter to renal pelvis, recording the Ureteral Peristalsis (UP) of left ureter within 30 minutes. Then, observed morphological variation with HE staining, and detected the expression of LVDCC in ureter among three groups measured by real-time fluorescent quantitative PCR and Western blot Results

The experimental group rabbit showed severe postoperative manifestation of hindlimb paralysis, Video urodynamic study suggests to contractile detrusor(ACD), sham and control group had no significant spinal cord injury performance and urodynamic change, the maximum bladder capacity, bladder compliance and detrusor leak point pressure of NUTD rabbit were statistically significant compared with others (P <0.001). All of the rabbits had no vesicoureteral reflux. The UP of left ureteral in NUTD significantly lower than the experimental and the blank control group. The result of HE staining suggests congestive and inflammatory infiltration in ureter adventitia of NUTD group. Nevertheless, there is a reduced expression of LVDCC mRNA and protein in NUTD ureter compared with sham group and control group, the difference was statistically significant (P <0.001), but the difference between sham group and control group shows no significance meaning.

L-VDCC mediated extracellular Ca2⁺ influx and constituted a major part of depolarized action potential. It is the fundamental to maintain muscle tension and Ureteral peristalsis persists. The reduced expression of L-VDCC will inevitably lead to ureter dysfunction, although it may be a long time.

Concluding message

There is a ureter dysfunction when it had not occurred vesicoureteral reflux and obviously pathological changes in early phase of NUTD, the main reason is the decline of L-VDCC of ureteral smooth muscle cells.

Disclosures

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