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OVEREXPRESSION OF AQUAPORIN 2 AND CAVEOLIN 1-3 IN DETRUSOR MUSCLE OF RAT URINARY BLADDER FOLLOWING BLADDER OUTLET OBSTRUCTION

Hypothesis / aims of study
The purposes of this study were to investigate the effect of detrusor overactivity induced by partial bladder outlet obstruction (BOO) on aquaporin 2 (AQP2) and caveolin 1-3 (CAV1-3) in the detrusor muscle and to determine the role of these molecules in the detrusor overactivity.

Study design, materials and methods
Female Sprague-Dawley rats were divided into control (n=30) and experimental (n=30) groups. The BOO group underwent partial BOO. The control group underwent a sham operation. After 4 weeks, an urodynamic study to measure the contraction interval and contraction pressure was conducted. The expression and cellular localization of AQP2 and CAV1-3 were determined by Western blot and immunofluorescent study in rat urinary bladder.

Results
In cystometrograms, the contraction interval (min) was significantly lower in the BOO group (3.4 ± 1.3) than in the control group (6.4 ± 1.6) ($p<0.05$). Conversely, the average contraction pressure (mmHg) was significantly higher in BOO rats (19.4 ± 4.1) than in the control group (12.9 ± 2.3) ($p<0.05$). AQP2 was expressed in the cytoplasm of the detrusor muscle, whereas CAV1-3 was expressed in the cell membrane of the smooth muscle, devoid of cytoplasm of the muscle cell. The protein expression of AQP2 and CAV1-3 was significantly increased in BOO rats ($p<0.05$).

Interpretation of results
Detrusor overactivity induced by BOO causes a significant increase in the expression of AQP2 and CAV1-3, which was differentially expressed in the detrusor muscle. The distinct location of the AQP2 and CAV1-3 might be closely related with the bladder signal activity.

Concluding message
Aquaporin 2 and caveolins 1-3 may be involved in the pathogenesis and also have a functional role in the detrusor overactivity occurring in association with bladder outlet obstruction

References
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Disclosures
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