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Song Y S[1], Lee H J[2], Doo S W[1], Kim J H[1], Yang W J[1], Lee S J[3], Yun J H[4], Kim S U[2]
1. *Department of Urology, Soonchunhyang University Hospital,* ***2.*** *Medical Research Institute, Chung-Ang University College of Medicine,* ***3.*** *Department of Urology, Kyunghee University Hospital,* ***4.*** *Department of Urology, Soonchunhyang University Gumi Hospital*

ENHANCEMENT OF ANTI-APOPTOSIS GENES EXPRESSION IN VOIDING DYSFUNCTION FROM LONG TERM BLADDER OUTLET OBSTRUCTION

Hypothesis / aims of study
Apoptosis plays a critical role in normal biological processes requiring cell removal. Ischemia induced from bladder outlet obstruction (BOO) causes damaged bladder cells to undergo apoptosis. Apoptotic changes of bladder smooth muscle could be induced by long term ischemia from BOO. Therefore, we designed this study to investigate to obtain an expression profile of apoptosis related genes in voiding dysfunction from long term bladder outlet obstruction.

Study design, materials and methods
Fifteen 6-week old female Sprague-Dawley rats were divided into 3 groups, 5 rats each, group 1: control, group 2: sham operation, group 3: BOO for 8 weeks and bladder dysfunction with the volume of residual urine more than 4 ml. Eight weeks after the onset of BOO, cystometric evaluation was performed, and bladder tissues were processed for PCR array. The first strand cDNA synthesis was performed with 2 μg of total RNA. Genes were considered to be up-regulated or down-regulated.

Results
Group with BOO showed thickened bladder wall and bladder dysfunction compared with control and sham operation group. Five genes were at least 2-fold up-regulated, and one gene was at least 2-fold down-regulated in BOO, compared with sham group. The up-regulated genes (fold change) by BOO belong to Bcl-2 (15.1), Birc5 (5.8), Cd40lg (7.5), Il10 (16.2), Naip2 (13.2), and the down-regulated gene belong to Prlr (-18.1). Anti-apoptosis genes included Bcl2a1d, Birc5, Cd40lg, Il10 and Naip2, Prlr. Caspase inhibitor included Birc5 and negative regulator included Cd40lg. Genes were influenced each other through TNF, STAT3 and TP53.

Interpretation of results
This results demonstrated that anti-apoptosis genes were enhanced in the bladder from long term BOO.

Concluding message
The gene expression profiles could explain changes of apoptosis in voiding dysfunction from long term BOO.

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