

THE TRANSITIONAL PERIOD FROM OAB TO UAB; DECREASED CONTRACTILITY AND INCREASED RESIDUAL URINE IN A RAT MODEL OF CHRONIC ISCHEMIC BLADDER

Hypothesis / aims of study

Epidemiological studies have shown that lower urinary tract symptoms, including overactive bladder (OAB) syndrome, occur commonly in the elderly of both sexes. ¹ Vascular endothelial dysfunction also occurs during human aging process and reported to be an independent risk factor for the atherosclerosis. Atherosclerotic changes in the pelvic vasculature may be an important contributing factor to the urinary tract symptoms. Clinically, detrusor underactivity is diagnosed based on urodynamic exam, defined as a contraction of decreased strength, sometimes with failure to achieve complete bladder emptying. ² It has already been suggested that chronic ischemia due to atherosclerosis is related in the pathophysiology of LUTS and progressive bladder dysfunction. ³ In this report, we introduce the chronic effect of arterial endothelial injury of the common iliac arteries with high cholesterol diet on the rat bladder function, which may clinically reflect the transitional period from OAB to underactive bladder (UAB).

Study design, materials and methods

Adult male Sprague-Dawley rats were divided into arterial endothelial injury (AI) and control groups. The AI group underwent balloon endothelial injury of the iliac arteries and received a 2% cholesterol diet for 16 weeks. The control group received a regular diet. After 16 weeks, urodynamic investigation was performed. Bladders were harvested for pharmacological and immunohistochemical studies for nitric oxide synthase (nNOS).

Results

Iliac arteries from the injury groups showed neointimal formation.

Cystometry showed increased residual volume of urine in the injury groups. (Fig.1) Micturition interval was shorter in the AI group than in the control. There was no difference in maximum pressure (MP) or threshold pressure (TP) between both groups. In the AI group, the bladder compliance was lower than in the control group. Non - voiding contraction was not observed in both of the groups. (Table.1)

Contractile responses of bladder strips from Control (n=10) were higher in KCl stimulation, carbachol concentration-response than in AI groups. The contractile responses to electrically field stimulation (EFS) were lower in the AI-group than in controls. Immunohistochemistry revealed that the expression of nNOS were almost absent in the AI group, when compared with the controls.

Interpretation of results

The present results suggest that a well-established rat model of chronic bladder ischemia shows increased residual urine and decreased contractility in longer period. Therefore, this model of chronic bladder ischemia reflects UAB in human.

Concluding message

Pelvic arterial occlusive disease may cause the decreased strength of contraction of the bladder and increased residual urine in a longer period. When translated clinically, those results might be associated with the symptom of UAB such as difficulty in emptying bladder. Our well-established model of the bladder chronic ischemia for 16 weeks may be the transitional period between OAB and UAB.

Fig.1

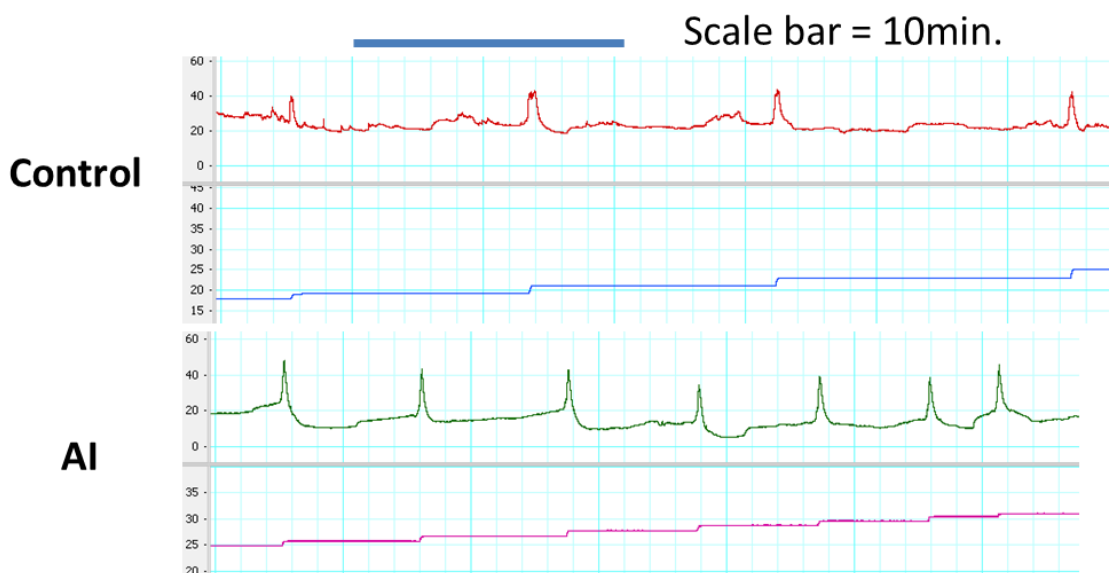


Table 1.

	control	AI
Micturition intervals (min)	9.45±1.57	4.21±0.39*
Bladder capacity (ml)	1.58±0.24	0.70±0.07*
Micturition volume (ml)	1.82±0.31	0.73±0.07*
Post void residual volume (ml)	0.00±0.00	0.02±0.01 *
Baseline pressure (cmH2O)	9.80±2.85	6.96±1.56
Threshold pressure (cmH2O)	20.03±3.23	19.33±2.34
Maximum pressure (cmH2O)	39.48±3.59	44.93±6.56
Compliance (ml/cmH2O)	0.17±0.03	0.05±0.01 *

Data were shown Mean ± SEM.

* p<0.01 vs control

† p<0.05 vs AI

References

1. Andersson KE, de Groat WC, McVary KT, et al. Tadalafil for the treatment of lower urinary tract symptoms secondary to benign prostatic hyperplasia: pathophysiology and mechanism(s) of action. *Neurourol Urodyn.* 2011;30: 292-301
2. Nomiya M, Yamaguchi O, Akaiha H, et al. Progressive vascular damage may lead to bladder underactivity in rats. *J Urol.* 2014;191: 1462-9.
3. Yamaguchi O, Nomiya M, Andersson KE. Functional consequences of chronic bladder ischemia. *Neurourol Urodyn.* 2014;33: 54-8.

Disclosures

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