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Urodynamic investigation of valve bladder syndrome in children

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Abstract *Objective:* To investigate urodynamic manifestations and their relationship with the postoperative experience of children with valve bladder syndrome (VBS).
Methods: Included were 16 children (mean age 3.2 ± 1.8 years) with VBS, who were divided into two groups. The urodynamic study was performed less than 1 year in group 1 (seven boys, aged 1—1.9 years) and more than 1 year in group 2 (nine boys, aged 2.9—6.5 years) after urethral valve fulguration; at the time of operation patients were less than 2 years old. Standards of the International Children's Continence Society were respected, and results were compared between the two groups.
Results: Compared to group 1, group 2 showed a significant decrease in maximum detrusor voiding pressure (Pdet.void.max) and bladder compliance (BC), and an increase in post-voiding residual (PVR) and maximum bladder capacity (MBC) ($p < 0.05$), but the difference in detrusor instability was not significant ($p > 0.05$), Pdet.void.max and PVR were 56.2 ± 14.1 cmH_2O and 96.6 ± 52.4 ml, respectively, in group 2, and there were more intermittent detrusor contractions during voiding in this group.
Conclusion: Patients with VBS frequently present with multiple bladder dysfunctions that can be diagnosed accurately using urodynamics. Even after urethral valve fulguration Pdet.void.max and BC were inclined to decrease, while PVR and MBC increased with the growth of the children.

Introduction

Posterior urethral valves (PUV) are a main cause of lower urinary tract obstruction in boys, which can result in diverse bladder problems and upper urinary tract deterioration even after early valve ablation after birth. Valve bladder syndrome (VBS) is the term used for a group of associated findings identified in patients with PUV and persistent upper tract dilatation following valve ablation, including a non-compliant thick-walled bladder, incontinence and nephrogenic diabetes insipidus. There is continuing debate regarding the etiology and management of the valve bladder [1]. The purpose of the present study was to investigate the urodynamic manifestations of this syndrome and their

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relationship with the postoperative experience of children with VBS.

Materials and methods

Patients

Sixteen children, aged 1—6.5 years (mean 3.2 ± 1.8), with VBS were evaluated in the Pediatric Urodynamic Center of the First Affiliated Hospital of Zhengzhou University, China, from July 2002 to December 2004. Seven boys, aged 1—1.9 years (mean 1.6 ± 0.3), were in group 1 and nine boys, aged 2.9—6.5 years (mean 4.5 ± 1.2), were in group 2. All children underwent successful ablation of PUV before the age of 2 years. The duration between urethral valve fulguration and urodynamic study was less than 1 year in group 1 and more than 1 year in group 2. No VUR and residual valves were found by cystography and cystourethroscope examination in all patients, and no children received anticholinergic drugs prior to the study.

Urodynamic studies

Slow-filling (10% of predicted bladder capacity in millilitre per minute, room temperature saline used as filling medium) cystometry with surface perineal electromyography (EMG) was performed using a Dan Tech Urodynamic Unit. All the children were encouraged to drink water or milk until voiding. Immediately after voiding, a double-lumen catheter (6 Fr) was introduced into the bladder urethrally with no local anesthesia for measuring the post-voiding residual (PVR) and performing a flow/pressure study. Abdominal pressure (Pabd) was measured through a balloon 6-Fr catheter placed in the rectum. Detrusor pressure was calculated by the subtraction of the Pabd from the vesical pressure (Pves). Filling was stopped when void began, urine leakage happened, there was a strong desire to void, or the young child became very anxious. Then the PVR was quickly measured again. Other recorded parameters of cystometry included maximum bladder capacity (MBC), bladder compliance (BC), maximal voiding detrusor pressure (Pdet.void.max), detrusor instability (DI) and voiding pattern. MBC was the sum of voided volume and PVR. The methods, definitions and units conform to the standards proposed by the International Children's Continence Society [2,3]. Cystometry was repeated at least once.

Statistical analysis

Values were expressed as mean $\pm$ SD. The two-samples *t*-test for independent samples was used to compare urodynamic parameters between the two groups. Fisher's exact test of probabilities was used to compare the frequency of DI. The significance level was $P < 0.05$. All calculations were performed using SPSS 10.0 statistical software.

Results

Compared to group 1, group 2 showed a significant decrease in Pdet.void.max and BC, and an increase in PVR and MBC ($P < 0.05$), while the difference in DI was not significant ($P < 0.05$) (Table 1). In group 2, there was more intermittent detrusor contraction during voiding; two boys showed no detrusor contraction, while four showed intermittent detrusor contraction, a special voiding pattern (Fig. 1). Mean Pdet.void.max and PVR were 56.2 ± 14.1 cmH_2O and 96.6 ± 52.4 ml, respectively.

Discussion

The etiology and pathophysiology of VBS are still unclear although this syndrome has been reported for more than 20 years [4]. It is known that PUV develop during pregnancy, and the pathophysiological changes leading to bladder dysfunction and deterioration of the upper urinary tract seem never to stop even in cases of early diagnosis and fulguration. A comparative study of bladder function immediately (within 1 year) and many years after fulguration has not been reported previously, although bladder dysfunction and renal damage are reported in 75% of survivors [5]. The primary results of the present study have confirmed that bladder dysfunction will continue to develop even after removing PUV in the very early stage of disease.

Pathohistological basis of bladder dysfunction in VBS

The human bladder is formed by 21 weeks of gestation. Bladder outlet obstruction and over distention can lead to smooth muscle proliferation, followed by an increase in total collagen and in the ratio of collagen type III-to-type I fibers. Both fiber types are principal elements in providing tension in the bladder. Increase of either causes a decrease in compliance. The bladder wall becomes thicker. Smooth muscle tension, and bladder compliance and storage ability are damaged. It has been reported that, even if treated very soon after birth, bladder histological changes cannot be reversed [4]. This may explain why multiple bladder dysfunction continues to exist in the patients of the present study. Early renal failure in neonates and infants with PUV has been reported, and this is possibly related to renal dysplasia and impaired nephron growth. The late deterioration of renal function,

Table 1 Results compared between two groups

	PVR (ml)	MBC (ml)	Pdet.void.max (cmH_2O)	BC (ml/cmH_2O)	DI (*n*)	No contraction (*n*)
Group 1 ($n = 7$)	42.8 ± 38.9	138.1 ± 20.1	95.1 ± 18.3	52.4 ± 26.9	4	0
Group 2 ($n = 9$)	96.6 ± 52.4*	217.4 ± 61.7**	56.2 ± 14.1**	12.5 ± 7.4**	2*	2

Values are mean $\pm$ SD;*$P < 0.05$, **$P < 0.01$.