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Urodynamic effects of α1-blocker tamsulosin on voiding dysfunction in patients with neurogenic bladder

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URODYNAMIC EFFECTS OF α1-BLOCKER TAMSULOSIN ON VOIDING DYSFUNCTION IN PATIENTS WITH NEUROGENIC BLADDER

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Running head: tamsulosin on voiding dysfunction

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Abstract

Backgroud: Therapeutic role of α-blockers in the treatment of voiding disorders due to benign prostatic hyperplasia has been extensively examined. To investigate a possible effect of α_1-blocker on urodynamic voiding parameters in patients with neurogenic bladder, we conducted a clinical trial using tamsulosin. ***Methods:*** Twenty-four patients with neurogenic bladder, 24 to 82 years old (mean age 61), 14 men and 10 women, were analyzed. Urodynamic studies were performed before and after treatment with 0.4 mg tamsulosin daily for 4 weeks. ***Results:*** On uroflowmetry, average flow rate (from 4.6 ± 3.3 to 6.7 ± 3.0 ml/s, p=0.04), maximum flow rate (from 9.4 ± 6.8 to 14.1 ± 7.0 ml/s, p=0.016) and residual urine rate (from 46 ± 29 to 32 ± 21%, p=0.02) improved significantly. In patients with detrusor contraction during voiding, detrusor opening pressure and detrusor pressure at maximum flow decreased significantly from 69.0 ± 36.2 to 49.2 ± 26.4 cmH_2O (p=0.046) and from 66.7 ± 34.6 to 53.6 ± 26.5 cmH_2O (p=0.007), respectively. On the other hand, in those patients with detrusor areflexia, vesical opening pressure (from 78.2 ± 23.4 to 61.6 ± 25.2 cmH_2O), or vesical pressure at maximum flow (from 68.6 ± 23.2 to 62.9 ± 25.2 cmH_2O) did not change significantly after treatment. ***Conclusions:*** Tamsulosin reduces functional urethral resistance during voiding and improves flow rate in patients with neurogenic bladder. It has more beneficial urodynamic effects in patients with detrusor contraction during voiding than in those with detrusor areflexia.

Key Words: tamsulosin, neurogenic bladder, urodynamics, voiding dysfunction, reflex

Introduction

During the last few decades one of the most active fields in research has been the physiology and pharmacology of the lower urinary tract. Since the pioneering work of Caine and his colleagues in 1976,[1] therapeutic role of α-blockers in the treatment of voiding disorders due to benign prostatic hyperplasia (BPH) has been extensively examined. Currently α_1-blockers have been the mainstay of the pharmacological treatment of BPH.[2-5]

The effects of α-blockers on neurogenic bladder have also been examined. Krane and Olsson were the first who reported the effect of α-blocker (phenoxybenzamine) on voiding dysfunction in patients with neurogenic bladder.[6] Since then, numerous clinical studies have been done with the majority in the form of uncontrolled trials. Clinical experience with α-blockers in neurogenic bladder has generally been encouraging, but the efficacy of α-blockers for improving voiding dysfunction may be limited.[7] Nevertheless, recent multicenter placebo-controlled, double-blind trials of an α-blocker urapidil which has been approved for the treatment of BPH have shown that this agent improved voiding dysfunction and decreased urethral resistance in patients with neurogenic bladder.[8,9] Although pressure-flow study was used in those studies, parameters examined were only vesical pressure at maximum flow rate and minimum urethral resistance which was calculated by the formula: minimum urethral resistance = (pressure at maximum flow) / (maximum flow rate) ^{2}.

To further investigate a possible effect of α_1-blocker on urodynamic voiding parameters in patients with neurogenic bladder, we conducted this clinical trial using tamsulosin which has been approved for the treatment of BPH.

Methods

Thirty-two patients with a neurogenic bladder were enrolled in this study that was conducted in 12 different hospitals in Japan. Excluded from this study were patients who could not void at all, and those with an indwelling urethral catheter, prostatic enlargement or urethral stricture. Patients with significant cardiac or cerebrovascular disorders, hepatic disorders, renal insufficiency and orthostatic hypotension were also excluded. Prostatic enlargement or urethral stricture was judged by digital rectal examination, ultrasonography and voiding cystourethrography. Before entry into this study, written informed consent was obtained from each patient, and the study was approved by the local ethics committee. Following baseline observation for 1 week or more, patients were started on 0.4 mg. tamsulosin daily for 4 weeks. α-blockers other than tamsulosin or α-agonists were not used during the study period while other drugs that had been used before entry into this study including β-blockers, β-agonists, anticholinergics, and antidepressants were allowed to be used if the doses were not changed during the study period.

Urodynamic studies, including uroflowmetry and pressure-flow study, were performed before and after treatment. Average and maximum flow rates were measured by uroflowmetry. For pressure-flow study, distilled water or saline was instilled into the bladder with a medium filling rate (50 ml/min or less). Intravesical pressure was monitored using 4 to 7 Fr. transurethral catheter. In each study, the baseline vesical and abdominal pressures were zeroed to atmospheric pressure at the level of the symphysis pubis. Otherwise the methods, definition and units for urodynamic studies conformed to the standards proposed by The International Continence Society.[10] Opening pressure, pressure at maximum flow, and maximum pressure were measured as detrusor pressure (intravesical pressure minus abdominal pressure) as well as intravesical pressure, because some patients with detrusor areflexia voided with straining, which might alter intravesical pressure and, therefore, flow.[8]

Since this study was multicenter trial, the final interpretation of urodynamic data in each patient was made by 3 of us (H.K., K.A. and S.K.) for quality control.

Paired data before and after treatment were analyzed by the paired Student's t test and the Wilcoxon signed rank test. Values of $p < 0.05$ were considered significant.

Results

Four patients withdrew from the study because of side effects in 3 (dizziness in 2 and de novo stress urinary incontinence in 1) and accidental fracture of femoral neck in 1 with spinocerebellar degeneration. Transient mild orthostatic hypotension was noted in another patient without withdrawal from the study. In addition to these 4 patients who withdrew from the study, 4 other patients could not void at urodynamic studies before treatment, and were excluded from the analysis. Thus, we studied the remaining 24 patients (mean age 60.8 years, range 24 to 82), including 14 men and 10 women. The underlying diseases in these 24 patients were brain lesions in 5 (21%, 4 men and 1 women), spinal cord diseases in 3 (13%, 3 men), peripheral nervous system diseases in 5 (21%, 2 men and 3 women), and not determined in the remaining 11 (46%, 5 men and 6 women). Clean intermittent catheterization was performed in 9 patients to eliminate the residual urine after self-voiding. When urodynamic studies were performed before and after treatment with tamsulosin, both uroflowmetry and pressure-flow study were successfully recorded in 12 patients, while uroflowmetry or pressure-flow study alone was recorded in 5 or 7 patients, respectively, because of inability to void or technical errors at either one of the 2 studies. Therefore, free uroflowmetry and pressure-flow study data before and after treatment were collected from 17 and 19 patients, respectively. Detrusor overactivity was noted in 4 of the 19 patients in whom the presence or absence of detrusor overactivity could be evaluated.

Regarding free uroflowmetric parameters in 17 patients, average and maximum flow rates, residual urine rate but not residual urine volume itself improved significantly (Table 1). Mean of maximum flow rate improved 50% from 9.4 to 14.1 ml/sec, and mean of residual urine rate decreased from 45.9 to 32.2% (Table 1). In 15 of the 17 patients, at least the presence or absence of detrusor contraction during voiding could be evaluated by the pressure-flow study. In 10 patients with detrusor contraction during voiding, maximum flow

rate but not average flow rate improved significantly, while in the other 5 patients with detrusor areflexia, none of free uroflowmetric parameters improved significantly (Table 2).

In the pressure-flow study, vesical opening pressure decreased significantly from 89.1 ± 31.2 to 65.7 ± 21.4 cmH_20, while detrusor opening pressure tended to decrease but did not reach statistical significance (p=0.058) (Table 3). Detrusor pressure at maximum flow and maximum detrusor pressure during voiding decreased significantly by 10 cmH_20 or more after treatment (Table 3). Maximum flow rate at pressure-flow study did not increase significantly after treatment (Table 3).

Of 19 patients in whom pressure-flow study was recorded before and after treatment, 8 had detrusor areflexia and voided with straining. Therefore, these patients were subdivided into 2 groups with (n=11, 10 men and 1 woman) or without (n=8, 1 man and 7 women) detrusor contraction during voiding. In patients with detrusor contraction during voiding, detrusor opening pressure, detrusor pressure at maximum flow, and maximum detrusor pressure decreased from 69.0 ± 36.2 to 49.2 ± 26.4 cmH_20, from 66.7 ± 34.6 to 53.6 ± 26.5 cmH_20, and from 83.0 ± 42.8 to 63.6 ± 28.6 cmH_20, respectively, while mean of maximum flow rate increased from 8.4 to 9.6 ml/sec without statistical significance (Table 4). On the other hand, in those patients without detrusor contraction during voiding, vesical opening pressure, vesical pressure at maximum flow, or maximum vesical pressure did not change significantly after treatment (Table 4).

There were sex differences in the overall results of free uroflowmetry, pressure-flow study. At free uroflowmetry, average flow rate and maximum flow rate did not improve significantly in women nor in men, while residual urine rate was significantly improved in men but not in women. At pressure-flow study, vesical opening pressure, detrusor pressure at maximum flow, maximum vesical pressure, and maximum detrusor pressure decreased significantly only in men.

Discussion

The present study has shown that an α_1-blocker tamsulosin improved urodynamic voiding parameters in patients with neurogenic bladder. In 11 patients who had detrusor contraction during voiding, detrusor opening pressure and maximum detrusor pressure significantly decreased by about 20 cmH_20 after treatment with tamsulosin. Detrusor pressure at maximum flow rate also decreased significantly from 66.7 ± 34.6 to 53.6 ± 26.5 cmH_20 after treatment with tamsulosin. These data suggest that tamsulosin can reduce functional outlet obstruction and improve urinary flow rate (confirmed by free uroflowmetry in the present study) in patients with neurogenic voiding dysfunction. Previous multicenter placebo-controlled, double-blind trials have shown that an α-blocker urapidil improved voiding dysfunction and decreased urethral resistance in patients with neurogenic bladder.[8,9] Although the present study was not a placebo-controlled, double-blind trial, results in the present study were consistent with those of previous studies examining the effect of urapidil. Thus, tamsulosin may have a therapeutic role in pharmacological treatment of neurogenic voiding dysfunction.

Theoretically, neurogenic voiding dysfunction is caused by a failure of either sphincter relaxation or detrusor contraction during voiding, or by a combination of both. Because α_1-blockers are not expected to improve detrusor areflexia, the effects of α_1-blockers on neurogenic voiding dysfunction, if any, might be related to improvement of functional outlet obstruction during voiding. Functional outlet obstruction is seen either at the level of the bladder neck (detrusor-bladder neck dyssynergia) or at the level of the external urethral sphincter (detrusor-external sphincter dyssynergia). Because the bladder neck and proximal urethra contain abundant α_1-adrenergic receptors, α-blockers or α_1-blockers are well known to reduce bladder neck obstruction in patients with or without neurogenic bladder.[6,11,12] However, the effects of α-blockers or α_1-blockers on the external urethral sphincter activity

have been conflicting. There have been controversies regarding the innervation of the external urethral sphincter. The somatic innervation of the external urethral sphincter has unanimously been accepted, whereas the existence of sympathetic and parasympathetic innervation of the external urethral sphincter has been debated. Ultrastructural studies strongly implicated the involvement of sympathetic postganglionic fibers in the innervation of the external urethral sphincter.[13,14] Although it remains an unresolved issue about the exact modes of sympathetic involvement, whether direct or indirect through the central nervous system, in the function of the striated muscle of the external urethral sphincter, our previous studies in spinal cord injury patients revealed that the activity of this unique rhabdosphincter is truly influenced by sympathetic drugs.[15-17] At the membranous urethra, intermingling of muscle fibers between urethral smooth musculature and striated musculature of the external urethral sphincter is noted,[18] which implies that both smooth and striated muscle components of the external urethral sphincter work altogether, not independently. Experimental evidence has shown that urethral smooth muscle contraction or relaxation can modulate the external urethral sphincter activity.[19] Taken together, α_1-blockers seem to have a potential to suppress the external urethral sphincter activity via an inhibitory effect on the urethral smooth muscle. Although electromyographic analysis of the external urethral sphincter was not performed in the present study, significant reduction of voiding detrusor pressure demonstrated in the present study may be derived not only from the direct effects of tamsulosin on the urethral smooth muscle but also from the indirect effects on the external urethral sphincter through the inhibitory action on the urethral smooth muscle.

Following treatment with tamsulosin, significant improvement of maximum flow rate was demonstrated at free uroflowmetry but not at pressure-flow study. This difference in results of maximum flow rate between the 2 studies could be due to some obstructive effects of transurethral catheter at pressure-flow study. Although the size of transurethral catheter used in the present study (4 to 7 Fr.) was not unusual for pressure-flow study, the presence of

transurethral catheter in patients with various degrees of functional outlet obstruction might have substantially jeopardized the effects of tamsulosin, thus preventing the significant improvement of maximum flow rate in spite of reduced voiding detrusor pressure.

Patients with detrusor contraction during voiding can be assessed more accurately with detrusor pressure than with vesical pressure. Basically, in these patients with detrusor contraction, detrusor pressure and vesical pressure correlate well provided that the same level of abdominal pressure is maintained. On the other hand, patients with detrusor areflexia can only be assessed with vesical pressure, because theoretically there can be no change in detrusor pressure in these patients. Thus, in addition to overall results (table 3), pressure-flow data were separately analyzed using changes in detrusor pressure and vesical pressure for those with detrusor contraction during voiding and those with detrusor areflexia, respectively (table 4). The reduction of voiding detrusor pressure following treatment with tamsulosin was clearly documented in patients with detrusor contraction, whereas voiding vesical pressure did not decrease in patients with detrusor areflexia (Table 4). These findings suggest that tamsulosin has more beneficial effects in patients with detrusor contraction than in those with detrusor areflexia. However, the number of patients was very small in this study. A significant improvement in maximum flow rate (table 2) and vesical opening pressure (table 4) may be accomplished if more numbers of patients are recruited. Several parameters in pressure-flow study improved significantly only in men. The most probable reason for these sex differences is the different population of men and women among those with detrusor contraction (10 of 11 men versus 1 of 8 women). The presence of detrusor contraction during voiding indicates the preservation of at least some parts of the voiding reflex arcs. Taken together, tamsulosin is, once again, considered to exert an inhibitory action on reflexly-generated urethral sphincter activity which is often abnormal in the form of detrusor-sphincter dyssynergia in patients with reflex bladder contraction.

α_1-blockers may have different actions on the lower urinary tract function depending on

the selectivity to α_1-adrenoceptor subtypes as well as the capability of passing the blood brain barrier.[20] Regarding central action of α_1-blockers on the neural control of the external urethral sphincter, prazosin that passes the blood brain barrier exerts an inhibitory action on pudendal nerve-dependent urethral constriction.[21] Intrathecal injection of doxazosin in conscious rats with or without bladder outlet obstruction reduced micturition pressure.[22] This experimental evidence endorses the central site of action in the effects of α_1-blockers. Since tamsulosin does not effectively pass the blood brain barrier, the observed reduction of voiding detrusor pressure in the present study is likely to be derived from its peripheral action including indirect effects on the external urethral sphincter through the inhibitory action on the urethral smooth muscle.

Conclusions

Although the present study was not a placebo-controlled, double-blind trial and patients numbers were very small, it has been shown that α_1-blocker tamsulosin reduced functional outlet obstruction during voiding and improved flow rate in patients with neurogenic bladder. It has more beneficial urodynamic effects in patients with detrusor contraction during voiding than in those with detrusor areflexia.

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Table 1 Changes in results of free uroflowmetry (n=17)

	Mean ± SD		
	Before Treatment	After Treatment	p Value (t test)
Average flow rate (ml/sec.)	4.6 ± 3.3	6.7 ± 3.1	0.04
Maximum flow rate (ml/sec.)	9.4 ± 6.8	14.1 ± 7.0	0.016
Residual urine volume (ml)	146 ± 133	133 ± 122	Not significant
Residual urine rate (%)	45.9 ± 29.5	32.2 ± 21.2	0.020

Table 2 Changes in results of free uroflowmetry in patients with (n=10) or without (n=5) detrusor contraction during voiding

	Mean ± SD		
	Before Treatment	After Treatment	p Value (t test)
Pts. with detrusor contraction			
Average flow rate (ml/sec.)	3.7 ± 3.5	6.3 ± 3.4	Not significant
Maximum flow rate (ml/sec.)	6.9 ± 4.6	13.1 ± 6.9	0.034
Residual urine volume (ml)	173 ± 128	147 ± 139	Not significant
Residual urine rate (%)	55.6 ± 30.3	33.6 ± 22.9	0.018
Pts. without detrusor contraction			
Average flow rate (ml/sec.)	6.0 ± 2.7	6.8 ± 2.7	Not significant
Maximum flow rate (ml/sec.)	12.9 ± 9.1	15.4 ± 8.2	Not significant
Residual urine volume (ml)	144 ± 153	139 ± 109	Not significant
Residual urine rate (%)	36.5 ± 24.4	30.4 ± 20.6	Not significant

Table 3 Changes in results of pressure-flow study (n=19)

Mean ± SD	Before Treatment	After Treatment	p Value (t test)
Vesical opening pressure (cmH_20)	89.1 ± 31.2	65.7 ± 21.4	0.007
Detrusor opening pressure (cmH_20)	51.2 ± 38.6	38.5 ± 26.7	Not significant
Vesical pressure at maximum flow (cmH_20)	80.6 ± 28.0	74.3 ± 20.6	Not significant
Detrusor pressure at maximum flow (cmH_20)	50.2 ± 36.4	40.2 ± 28.6	0.005
Maximum vesical pressure (cmH_20)	107.7 ± 35.1	97.3 ± 30.2	Not significant
Maximum detrusor pressure (cmH_20)	63.4 ± 45.1	51.1 ± 29.8	0.024
Maximum flow rate (ml/sec.)	10.1 ± 4.7	11.0 ± 4.3	Not significant

Table 4 Urodynamic parameters (pressure-flow study) in patients with (n=11) or without (n=8) detrusor contraction during voiding

Mean ± SD	Before Treatment	After Treatment	p Value (t test)
Pts. with detrusor contraction			
Detrusor opening pressure (cmH_20)	69.0 ± 36.2	49.2 ± 26.4	0.046
Detrusor pressure at maximum flow (cmH_20)	66.7 ± 34.6	53.6 ± 26.5	0.007
Maximum detrusor pressure (cmH_20)	83.0 ± 42.8	63.6 ± 28.6	0.011
Maximum flow rate (ml/sec.)	8.4 ± 3.3	9.6 ± 3.0	Not significant
Pts. without detrusor contraction			
Vesical opening pressure (cmH_20)	78.2 ± 23.4	61.6 ± 25.2	Not significant
Vesical pressure at maximum flow (cmH_20)	68.6 ± 23.2	62.9 ± 25.2	Not significant
Maximum vesical pressure (cmH_20)	89.6 ± 37.5	92.7 ± 43.4	Not significant
Maximum flow rate (ml/sec.)	12.6 ± 5.6	11.6 ± 4.1	Not significant