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Title: URODYNAMIC EFFECTS OF ALPHA1-BLOCKER TAMSULOSIN ON NEUROGENIC VOIDING DYSFUNCTION

Aims of Study:

Since the pioneering work of Caine in 1976, therapeutic role of α-blockers in the treatment of voiding disorders due to benign prostatic hyperplasia (BPH) has been extensively examined. Although α-blocker has been reported to improve voiding dysfunction and decrease urethral resistance in patients with neurogenic bladder [1,2], urodynamic effects of α_1-blocker on neurogenic voiding dysfunction have not been fully evaluated. To further investigate a possible effect of α_1-blocker on urodynamic voiding parameters in patients with neurogenic voiding dysfunction, we conducted a multicenter uncontrolled trial using tamsulosin which has been approved for the treatment of BPH.

Methods:

Twelve hospitals in Japan participated in this study. Twenty-four patients with neurogenic voiding dysfunction, 24 to 82 years old (mean age 61), 14 men and 10 women, were analyzed. The underlying diseases in these 24 patients were brain lesions in 5 (21%), spinal cord diseases in 3 (13%), peripheral nervous system diseases in 5 (21%), and others in the remaining 11 (46%). Free uroflowmetry and pressure-flow study were performed before and after treatment with 0.4 mg tamsulosin daily for 4 weeks.

Results:

Free uroflowmetry:

Free uroflowmetry was performed in 17 patients. Average flow rate (from 4.6 $\pm$ 3.3 to 6.7 $\pm$ 3.1 ml/s), maximum flow rate (from 9.4 $\pm$ 6.8 to 14.1 $\pm$ 7.0 ml/s) and residual urine rate (from 46 $\pm$ 30 to 32 $\pm$ 21%) improved significantly in overall. In 15 of the 17 patients, at least the presence or absence of detrusor contraction during voiding could be evaluated by subsequent pressure-flow study. Thus, free uroflowmetry data were sub-analyzed depending on the presence or absence of detrusor contraction during voiding. In 10 patients with detrusor contraction during voiding, maximum flow rate (from 6.9 $\pm$ 4.6 to 13.1 $\pm$ 6.9 ml/s) but not average flow rate (from 3.7 $\pm$ 3.5 to 6.3 $\pm$ 3.4 ml/s) improved significantly, while in other 5 patients with detrusor areflexia, neither average flow rate (from 6.0 $\pm$ 2.7 to 6.8 $\pm$ 2.7 ml/s) nor maximum flow rate (from 12.9 $\pm$ 9.1 to 15.4 $\pm$ 8.2 ml/s) improved significantly

2. Pressure-flow study

Of 19 patients in whom pressure-flow study was performed, 8 had detrusor areflexia and voided with straining. Thus, 19 patients were subdivided into 2 groups with (n=11) or without (n=8) detrusor contraction during voiding. In patients with detrusor contraction during voiding, detrusor opening pressure, detrusor pressure at maximum flow rate, and maximum detrusor pressure decreased significantly from 69 $\pm$ 36 to 49

$\pm$ 26 cmH_2O, from 67 $\pm$ 35 to 54 $\pm$ 27 cmH_2O, and from 83 $\pm$ 43 to 64 $\pm$ 29 cmH_2O, respectively. On the other hand, in those patients with detrusor areflexia, vesical opening pressure (from 78 $\pm$ 23 to 62 $\pm$ 25 cmH_2O), vesical pressure at maximum flow rate (from 69 $\pm$ 23 to 63 $\pm$ 25 cmH_2O), or maximum vesical pressure (from 90 $\pm$ 38 to 93 $\pm$ 43 cmH_2O) did not change significantly after treatment.

Conclusions:

Although the present study was not a placebo-controlled, double-blind trial and patients numbers were small, it has been shown that α_1-blocker tamsulosin reduced functional urethral resistance during voiding and improved flow rate in patients with neurogenic voiding dysfunction. It has more beneficial urodynamic effects in patients with detrusor contraction during voiding than in those with detrusor areflexia. 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 inhibitory effects on reflexly generated external urethral sphincter activity through the inhibitory action on the urethral smooth muscle [3].

References:

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