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A TEMPORAL DIFFERENCE OBSERVED IN THE EFFECT OF DUTASTERIDE ON REDUCING PROSTATE VOLUME DEPENDS ON THE DEGREE OF INTRAVESICAL PROSTATIC PROTRUSION IN MALE PATIENTS WITH BENIGN PROSTATIC HYPERPLASIA

Hypothesis / aims of study

Recent articles have demonstrated that intravesical prostatic protrusion (IPP) can identify bladder outlet obstruction (BOO) in patients with lower urinary tract symptoms suggestive of benign prostatic enlargement (LUTS/BPE). As IPP increases, the degree of BOO also increase. A cut off value of 10mm of IPP has been reported as a good predictive value to define BOO. Moreover, IPP exceeding 10mm seems to more frequently respond poorly to medical treatment with tamsulosin among patients with LUTS/BPE [1]. The aim of this study is to assess how the effect of dutasteride depends on the degree of IPP in male patients with BPE.

Study design, materials and methods

We retrospectively analyzed 132 patients with BPE who were referred to our hospital with persisting LUTS in spite of receiving alpha-adrenergic antagonist monotherapy. Prior to, six months and one-year after receiving dutasteride (0.5 mg daily) add-on therapy with a preceding alpha-adrenergic antagonist, we evaluated the clinical parameters as described below; prostate volume (PV), IPP, maximum urine flow rate (Qmax), voided volume (VV), postvoid residual urine volume (PVR), international prostate symptom score (IPSS), quality of life (QOL) score, and prostate specific antigen (PSA). PV was more than 30 ml in all patients. IPP, as measured from a sagittal view, was defined by the distance from the tip of the prostatic protrusion into the vesical lumen to the bladder neck. If the PSA level exceeded 4.0 ng/ml, prostate biopsy was routinely recommended to rule out prostate cancer, and patients who were negative for cancer were included in this study.

We divided the patients into two groups according to IPP: Group A ≤ 10 mm (n = 58) and Group B >10 mm (n = 74). All patients received a treatment of 0.5 mg of dutasteride once daily for one year, and none of the study patients received any anticholinergic agents in this period.

All variables are expressed as mean ± SD. The data were analyzed with paired or unpaired *t*-test. Statistical significance was defined as $p < 0.05$.

Results

The mean age of the 132 patients was 73.3 ± 6.9 years. Preceding alpha-adrenergic antagonist was naftopidil in 34 patients, silodosin in 52 patients, and tamsulosin in 46 patients. Alpha-adrenergic antagonists were not changed in this study duration.

The mean PV was 56.6 ± 31.5 ml (group A: 41.5 ± 18.3 ml, group B: 68.3 ± 34.6 ml, $p < 0.001$) and the mean IPP was 1.21 ± 0.80cm (group A: 0.57 ± 0.32 cm, group B: 1.71 ± 0.70 cm, $p < 0.001$). The mean values of IPSS, QOL score, Qmax, VV, PVR and PSA were 13.2 ± 7.3 (group A: 13.5 ± 7.1, group B 13.1 ± 7.5), 3.4 ± 1.2 (group A: 3.5 ± 1.2, group B: 3.3 ± 1.2), 8.8 ± 4.4 ml/s (group A: 9.7 ± 4.9 ml/sec, group B: 8.1 ± 4.0 ml/sec), 154.6 ± 96.9 ml (group A: 176.4 ± 113.7 ml, group B: 137.6 ± 77.9 ml), 78.2 ± 77.5 ml (group A: 71.0 ± 82.5 ml, group B: 83.8 ± 73.5 ml), and 4.8 ± 4.4 ng/ml (group A: 3.7 ± 4.4 ng/ml, group B: 5.7 ± 4.2 ng/ml), respectively. There were no significant differences between the two groups.

Temporal changes of each parameter after dutasteride administration were shown as Table. After six months of dutasteride treatment, the mean PV decreased 20.6% (group A: 24.2%, group B: 17.5%, $p = 0.0391$). And after a one year period, the mean PV decreased 22.9% in both groups.

Interpretation of results

BOO is caused not only by total prostatic volume but also by configuration and/or deformity (mainly IPP) of the prostate [1]. A cut off value of 10 mm of IPP has been reported to offer good sensitivity in defining of BOO [2].

Patient with a severe degree of IPP (> 10 mm) showed a poor response to the treatment with dutasteride over a six-month period, but there were no significant differences between the two groups after a one-year period. Previous report indicated that IPP can predict the clinical progression in patients with BPE [3]. This phenomenon may be a reason that the effect of dutasteride emerges slowly.

Concluding message

A temporal difference observed in the effect of dutasteride on reducing PV depends on the degree of IPP in male patients with benign prostatic hyperplasia.

Table. Temporal changes of each parameter after dutasteride administration.

	IPP − n = 58		*P* value		
	6M	12M	Pre vs. 6M	6M vs. 12M	Pre vs. 12M
IPSS	−2.8 ± 5.5	−2.4 ± 6.1	< 0.001	ns	< 0.01
QOL	−0.6 ± 1.2	−0.7 ± 1.2	< 0.001	ns	< 0.001
Qmax (ml/sec)	+1.7 ± 5.0	+1.1 ± 4.6	< 0.05	ns	ns
VV (ml)	+14.1 ± 147.6	+21.2 ± 126.4	ns	ns	ns
PVR (ml)	−20.8 ± 84.2	−26.5 ± 82.4	ns	ns	< 0.05
PSA (ng/ml)	−1.5 ± 3.1	−1.8 ± 2.5	< 0.001	<0.05	< 0.001
PV (ml)	−9.9 ± 8.5	−9.6 ± 10.1	< 0.001	ns	< 0.001

	IPP + n = 74		*P* value		
	6M	12M	Pre vs. 6M	6M vs. 12M	Pre vs. 12M
IPSS	−2.2 ± 5.1	−2.2 ± 5.1	< 0.001	ns	< 0.001
QOL	−0.5 ± 1.2	−0.5 ± 1.1	< 0.01	ns	< 0.001
Qmax (ml/sec)	+0.7 ± 4.1	+ 1.5 ± 5.7	ns	ns	< 0.05
VV (ml)	−4.4 ± 80.0	+29.2 ± 96.2	ns	< 0.01	< 0.05
PVR (ml)	−28.8 ± 66.2	−18.7 ± 79.0	< 0.001	ns	< 0.05
PSA (ng/ml)	−3.0 ± 2.8	−3.2 ± 2.7	< 0.001	< 0.01	< 0.001
PV (ml)	−13.3 ± 16.6	−17.2 ± 20.1	< 0.001	< 0.01	< 0.001

References

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Disclosures

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