The THAI Journal of SURGERY 2007; 28:65-68.
Official Publication of the Royal College of Surgeons of Thailand

Efficacy of Medical Management of BPH with Lower Urinary Tract Symptoms: Urodynamic Studies at Chiangmai University Hospital

Sumitr Anutrakulchai, MD

Division of Urology, Department of Surgery, Chiangmai University, Chiangmai, Thailand

Abstract

Background: **Nowadays medical treatment of BPH with lower urinary tract symptoms (LUTS) with α_1-adrenoceptor antagonists/α_1-blockers and 5α-reductase inhibitor (Finasteride) is often practiced among urologists and general practitioners. Many patients receiving these medications get some improvement of symptoms but the efficacy of reducing bladder outlet obstruction (BOO) is questionable. The purpose of this study was to study the efficacy of the drugs in relieving BOO by urodynamic studies.**

Materials and Methods: **Urodynamic data of 26 patients with age ranged from 47-88 years (mean 69.52± 8.24 years) who had slight or no improvement of LUTS during medical management (duration 2-96 months, mean 29.52 ± 30.39 months) were collected for analysis. Blaivas's criteria of male voiding dysfunction were used. These patients were divided into 2 groups. Obstructed group consisted of 13 patients (50%), of which 2 patients had previous TURP (inadequate). Of 13 patients, 9 received α_1-blockers, 3 received combination of medications (both α_1-blockers and 5α- reductase inhibitor), 1 received only 5α- reductase. Two of these patients underwent urodynamic studies during on- and off- medication period and were compared. Unobstructed group consisted of 13 patients (50%), 4 had previous TURP. Of these, 11 received α_1-blockers, 2 received combination of medications.**

Results: **The results of the urodynamic study in 26 patients receiving medications were analyzed. In obstructed group, average Pdet.Qmax was 78.69 ± 31.44 cm.H_2O and Qmax was 6.46 ± 5.15 ml/s. Of these 13 patients, 2 stopped medication more than one year and Pdet.Qmax was 76 ± 22.62 cm.H2O, Qmax 6.5 ± 3.5 ml/s; while on medication Pdet.Qmax was 80 ± 35.35 (p = 0.907) and Qmax was 6 ± 2.8 (p = 0.891). In unobstructed group, average Pdet.Qmax was 24.53 ± 9.49 cm.H_2O, Qmax 10.38 ± 6.97 ml/s.**

Conclusions: **The medical management of BPH with lower urinary tract symptoms can improve lower urinary tract symptoms but cannot relieve bladder outlet obstruction. Prescribing these medications is unnecessary for unobstructed patients. Before prescribing, the property and efficacy of each drug should be explained and discussed with the patients.**

Key words: **benign prostatic hyperplasia, medical treatment, urodynamics**

Correspondence address : *Sumitr Anutrakulchai, MD, Division of Urology, Department of Surgery, Chiangmai University, Chiangmai 50200, Thailand. E-mail address: asumitr@yahoo.com*

Introduction

Benign prostatic enlargement (BPE) or benign prostatic hyperplasia (BPH) with lower urinary tract symptoms (LUTS) is very common in men over the age of 50. However, many do not understand that BPH is not a medical problem itself. It is a histological diagnosis and it can cause abnormal symptoms related to micturition.[1] Medical management including α_1-adrenoceptor antagonists (α_1-blockers) and 5α-reductase inhibitor (Finasteride) aims at relieving bladder outlet obstruction (BOO) or to reduce urethral resistance by reducing the size of the prostate. α_1-blockers induce prostate apoptosis and relax prostatic smooth muscle, thus relieve the dynamic component of benign prostatic obstruction (BPO),[2-4] while 5α-reductase inhibitor reduces the size of the prostate gland (16-20%) by inhibiting conversion of testosterone to dihydrotestosterone in prostate gland.[5,6] The aim of this retrospective study was to analyze the efficacy of the medical management of BPE or BPH with LUTS by urodynamic study.

Materials and Methods

Urodynamic studies in 26 patients with an age range of 47-88 years (mean 69.52 ± 8.24 years) who had lower urinary tract symptoms (LUTS) during medical management (duration 2-96 months, mean 29.52 ± 30.39 mos.) were analyzed retrospectively. Of these, only 2 patients underwent urodynamic studies before and during medication. Blaivas's criteria of male voiding dysfunction[7] (Pdet.Qmax >40 cm.H_2O, Qmax <12 ml./s=Obstruction; Pdet.Qmax = 30-40 cm.H_2O, Qmax <12 ml./s =Intermediate; Pdet.Qmax <30 cm.H_2O, Qmax<12 ml./s=Unobstruction or Impaired contractility) were used. In this report, intermediate group was also classified as unobstruction. These data were divided into two groups.

Obstructed group consisted of 13 (50%) patients, 2 of these had previous TURP. 9/13 received α_1-blocker, 3/13 received combination of α_1-blocker and 5α- reductase inhibitor, 1/13 received only 5α-reductase inhibitor. Two cases in this group underwent urodynamic studies during on- and off- medication and were compared.

Unobstructed group consisted of 13 (50%) patients; 4 had previous TURP, 11/13 received α_1-blocker, 2/13 received combination.

Janus V 3.7 Life Tech Inc. was used for videourodynamic studies. For standard deviation of urodynamic variables, SPSS v.2 was used. Urodynamic methods, units, and terminology conformed to the standards recommended by the International Continence Society.[8]

Results

Type, duration of medical management and urodynamic diagnosis in 26 patients were shown in Table 1 and 2.

In obstructed group, 11 of 13 had detrusor overactivity (DO) and BPO, 2 of 13 had BPO. Average PdetQmax in this group was 78.69 ± 31.44 cm.H_2O and Qmax was 6.46 ± 5.15 ml/s. In two patients who had stopped medication for more than one year, PdetQmax was 76 ± 22.62 cm.H2O and Qmax. was 6.5 ± 3.5 ml./s, compared with PdetQmax of 80 ± 35.35 cm.H_2O (p = 0.907) and Qmax. 6 ± 2.8 ml./s (p = 0.891) while taking medication.

In unobstructed group, 6 of 13 had impaired contractility (ImC), 3/13 had DO, 2/13 had DO plus ImC, and 2/13 were normal. Average PdetQmax was 24.53 ± 9.49 cm.H_2O and Qmax was 10.38 ± 6.97

Table 1 Duration and type of medication

Variable	α_1- adrenoceptor antagonists	5α- reductase inhibitor	Combination
Duration of medication (mos.)	2-96 (29.52 ± 30.39)	96	14-24 (18 ± 3.74)
Type of medication			
a. obstructed group (case)	9	1	3
b. unobstructed group (case)	11	-	2

Table 2 Urodynamic diagnosis and variables

Urodynamic diagnosis	PdetQmax ($cm.H_2O$)	Qmax (ml./s)
Obstructed group - DO+BPO 11 (2) cases - BPO 2 cases	78.69 ± 31.44	24.53 ± 9.49
Unobstructed group - ImC 6 (2) cases - DO 3 (1) cases - DO+ImC 2 cases - Normal 2 (1) cases	6.46 ± 5.15	10.38 ± 6.97

DO = detrusor overactivity
BPO = benign prostatic obstruction, ImC = Impaired contractility
PdetQmax = detrusor pressure at maximum flow rate
Qmax = maximum flow rate, ( ) = Post TURP

ml./s. Despite the lack of pre- and during medication studies, randomized studies on medical management of BPH were reviewed.

Discussion

Lower urinary tract symptoms can be improved by less invasive management, i.e. medical therapies which are popular at the present time. There were few reports using Pressure-Flow studies for classifying patients into obstructed and unobstructed group including before and during medication. Most of them used symptom scores and maximum flow rate (Qmax) for evaluation in their studies, which were unable to serve the aim of medical therapy. The patients in this retrospective study still had mild or moderate LUTS.

In the obstructed group, there were still high PdetQmax (>40 $cm.H_2O$) and low Qmax (<12 ml./s) (Table 2). These showed that medications (single or combination) were unable to reduce urethral resistance to normal or unobstructed level even in long-term treatment. There were 2 cases that underwent urodynamic studies during on- and off- medication period which showed no improvement of PdetQmax ($p = 0.907$) and Qmax ($p = 0.891$). Tammela and Kontturi[9] reported long-term effects of Finasteride in 36 patients with BOO. There was some decrease in PdetQmax but it was still high (>40 $cm.H_2O$) and with low flow (<12 ml./s), of which 15 patients were classified as equivocal, only 2 cases could be classified as unobstruction. Rossi et al. performed urodynamic studies pre- and 6-months of medication in 163 BOO patients treated with 3 types of α_1-blockers and reported that α_1-blockers were effective for treating symptoms suggestive of BOO but not for treating the obstruction regardless of the α_1-blockers used,[10] compared with results of TURP in previous publication[11] in which high PdetQmax and low Qmax returned to low or normal PdetQmax and normal Qmax respectively.

In unobstructed group, there was no improvement of Qmax to normal level because of no increased urethral resistance. Decreased flow rate might be caused by ageing bladder, so these treatments were unnecessary. Of these, 2 were normal (Qmax. >12 ml./s). They received α_1-blockers for treating their LUTS (frequency urgency and poor stream) which also showed no improvement. Although this was not a randomized, controlled study it could be a guideline or a warning before prescribing the medications.

Conclusions

Medical management of BPE or BPH with LUTS can improve some degree of symptoms but cannot relieve bladder outlet obstruction or can only partially decrease outlet resistance. In case of unobstruction, medication is unnecessary in many cases.

REFERENCES

1. Burnett AL, Wein AJ. Benign prostatic hyperplasia in primary care: what you need to know. BJU 2006; 175: S19-S24.
2. Chon JK, Borkowski AW, Partin AW, et al. α_1- adrenoceptor antagonists Terazosin and Doxazosin induce prostate apoptosis without affecting cell proliferation in patients with benign prostatic hyperplasia. J Urol 1999; 161: 2002-8.
3. Caine M. Alpha-adrenergic blockers for the treatment of benign prostatic hyperplasia. Urol Clin North Am 1990; 17: 641-9.
4. Abrams P, Speakman M, Stott M, et al. A dose-ranging study of the efficacy and safety of tamsulosin, the first prostate-selective α_{1A}- adrenoceptor antagonist, in patients with benign prostatic obstruction (symptomatic benign prostatic hyperplasia). BJU 1997; 80: 587-96.
5. Finasteride (MK-906) in the treatment of benign prostatic hyperplasia. The Finasteride study group. Prostate 1993; 22: 291-9.

6. Frankel S. Analyzing finasteride data. Neurourol Urodyn 1995; 14: 619-24.
7. Blaivas JG. Obstruction uropathy in the male. Urol Clin North Am 1996; 23: 373-4.
8. Abrams P, Cardozo L, Fall M, et al. The standardization of terminology of lower urinary tract function: report from the standardization sub-committee of the International Continence Society. Neurourol Urodyn 2002; 21: 167-178.
9. Tammela TLJ, Kontturi MJ. Long-term effects of finasteride on invasive urodynamics and symptoms in the treatment of patients with bladder outflow obstruction due to benign prostatic hyperplasia. J Urol 1995; 154: 1466-9.
10. Rossi C, Kortmann BBM, Sonke GS, et al. α_1-blockade improves symptoms suggestive of bladder outlet obstruction but fails to relieve it. J Urol 2001; 165: 38-41.
11. Anutrakulchai S. Detrusor response after TURP in benign prostatic enlargement; an immediate result of urodynamic study. Thai J Urol 2004; 25: 1-14.