Urodynamic Assessments of Bladder Outflow Obstruction Associated with Benign Prostatic Hyperplasia

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Abstract

In order to reliably diagnose bladder outflow obstruction (BOO) in benign prostatic hyperplasia (BPH), we conducted a comparative study in 76 patients with clinically diagnosed BPH, 52 of whom had BOO and 24 who did not. Urodynamic assessment revealed that urethral opening pressure, minimal urethral opening pressure, detrusor pressure at maximal urine flow, and detrusor pressure were significantly higher in patients with BOO than in patients who did not have BOO or whose assessments[1] were normal ($P < 0.01$); no significant difference between patients without BOO and those who were normal were found. Among the 27 patients who underwent prostatectomy, maximal flow rate rate was significantly higher in 23 patients with BOO than in 4 patients without BOO after surgery ($P < 0.01$). Patients with and without BOO had similar I-PSS (International Prostate Symptom Score) and PS (prostatic size) These results suggest that I-PSS, PS, free Qm (maximal flow rate) and RUV (residual urine volume) are not specific markers for BOO diagnosis in BPH patients. Besides, bladder neck pressure, bladder neck length, prostatic urethral pressure, and prostatic urethral length of static urethral pressure profile (SUPP) were significantly higher in BPH patients, compared to normal. However, there was no significant difference in SUPP between patients with and without BOO. We concluded that P/F (prostate and urine flow) study and SUPP together could provide better guide therapy options and prognosis of BPH.

Keywords: benign prostatic hyperplasia, bladder outflow obstruction, urodynamics

Introduction

Benign prostatic hyperplasia (BPH) is caused by gland enlargement and often results in lower urinary tract symptoms (LUTS)[1]. In men aged between 60 and 85 years, 50–90% have been diagnosed with BPH in the US.[2] Thus, treatment of BPH is apparently a substantial burden on families, as well as on the national health care system.

LUTS is commonly caused by bladder outflow obstruction (BOO), which is the main reason of patient's impaired quality of life.[3,4] Unfortunately, an average of 40% of BPH patients have LUTS, confirmed by histology.[5] The symptoms of BPH (for example, hesitancy and voiding frequency) are related to BOO. However, these symptoms are also related to other diseases and are insufficient to be used to diagnose BPH.[6]

In most cases, BOO is the only clinical indication for surgical therapy of BPH. In clinic, physicians currently diagnose BPH by international prostatic symptom scores (I-PSS), residual urine volume (RUV) > 50ml, bigger prostatic size (PS) or/with upper urinary tract enlargement. Resection of the prostate is considered to be an effective way to treat BPH in clinic.[7] But it has been shown that approximately 20% of patients do not get desired outcomes after surgical therapy, and around 17% patients need secondary surgery within eight years.[8]

Further, it has been shown that 25% of BPH patients don't have the symptoms of BOO. Together, a comprehensive diagnosis for a BPH patient is needed in order to determine a suitable therapy. Here we focus on how to precisely diagnose BOO in BPH patients prior to surgical therapy.

In this comparative study, we revalued the specificity and sensitivity of clinical assessments (symptoms, residual urine volume,[9] and prostatic size) and complete urodynamic studies (Free Uroflowmetry, Static Urethral Pressure Profile and Pressure flow studies) in the diagnosis of BOO in BPH patients.

Materials and Methods

Ethics

The IRB (institutional review board) of this research was approved by the Ethics committee of Kunming General Hospital.

Subjects

Group I

Between March 1995 and March 1996, 76 BPH patients with LUTS were evaluated by clinical assessments and urodynamics. The mean age of patients was 65.6 years (47–82 years) and the mean course of disease was 7 months (2 weeks–10 years).

Patients included in the study met the following criteria: 1) a symptom of urination, nocturia, urgency, hesitancy, and weak urine stream; (2 prostatic hyperplasia evaluated by I-PSS, digital rectal examination(DRE), and transrectal ultrasound (TRUS); 3) negative urinalysis; 4) no history or evidence of prostate cancer, urethral stricture, or active urinary tract stone disease; 5) no evidence of neurogenic bladder dysfunction; and 6) no exposure to drugs, such as alpha agonists, anticholinergics, cholinergics, or diuretic agents[10] within one month. Among the 76 patients who met the inclusion criteria, 52 had BOO and 24 patients did not.

Group II

Among the 76 BPH patients, 27 underwent suprapubic prostatectomy. Among these, 23 had BOO and 4 did not. The mean time of follow up is two and one-half months (2 weeks–6 months).

Group III

In addition, 20 normal males were recruited and assigned to Group III. The normal males in Group III meet the following criteria: 1) mean age 65.4 years (50-74 years old); 2) no history of LUTS (I-PSS < 5); 3) mean maximum flow rate of 26.19 ml (20.72–40.82 ml); 4) normal prostatic size determined by digital rectal examination; 5) no evidence of neurogenic bladder dysfunction; 6) no exposure to medication within one month that affects urine.

Clinical Assessments

All 76 BPH patients were evaluated with I-PSS, OSS (obstructive symptom scores) (questionnaire #3, 5, 6), ISS (irritative symptom scores) (questionnaire #1, 2, 4, 7), RUV, DRE, and TRUS.

Urodynamics

All 76 BPH patients and 20 normal males were evaluated by Free Uroflowmetry, static urethral pressure profile (SUPP), and pressure flow studies (P/F). The urodynamic parameters

were defined according to the International Continence Society (ICS) and Southwestern Hospital; the third military medical university (Chongqing, China), which included detrusor instability (DD); free maximal flow rate (Free Qmax); bladder neck pressure (PN); maximum urethral pressure (Pm); prostatic urethral pressure (Pp, also called verumontanum pressure); bladder neck length (LN); prostatic urethral length (Lp); abdominal pressure (Pabd); detrusor pressure (Pdet); urethral opening pressure (Puo); minimal urethral opening pressure (Pmuo); detrusor pressure at maximal urine flow (Pdet@Qmax), and detrusor pressure (Pdet@ max).

BOO Diagnosis

BOO was diagnosed according to Abrams and Griffiths Nomogram (AG Nomogram; Figure 1), combined with the pressure/flow rate function curve graph (X-Y curve; Figure 2).

Statistical Analysis

All data were analyzed by SPMR medical bioinformatics software, which was designed by the third military medical university (Chongqing, China). The unpaired chi-square and Student's *t*-tests were used to determine whether there was a biological significance ($P < 0.05$ or $P < 0.01$) among the enrolled groups based on urodynamics.

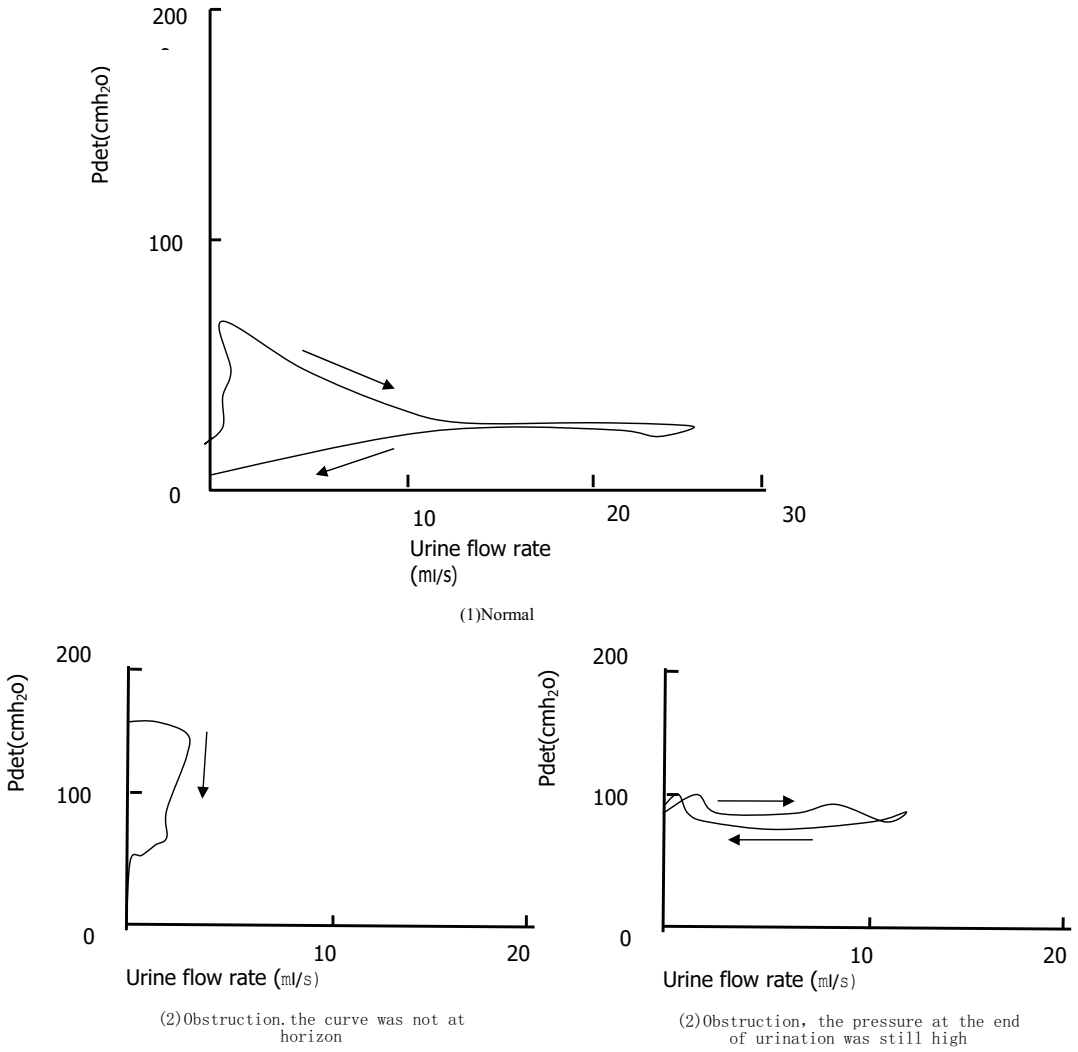

Figure 1 Pressure / flow rate function curve graph (X — Y curve)

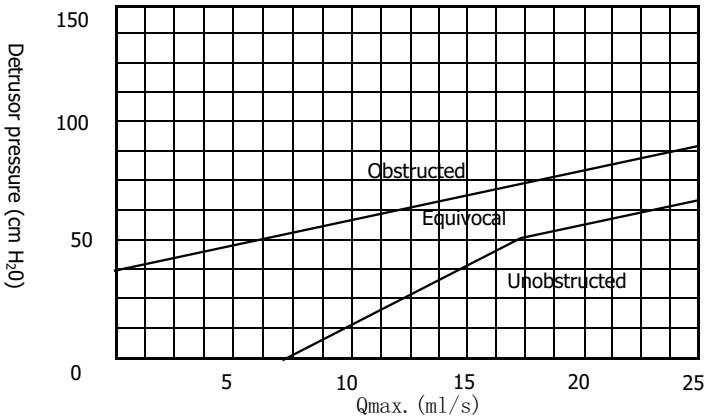

Figure 2 Abrams-Griffiths Nomogram

Results

1. Pressure/Flow Study

Seventy-six BPH patients were categorized into BOO (52) and non-BOO (24) groups by A-G (Figure 1) and X-Y line figure (Figure 2). The results showed the following:

A. PUO, Pmuo, Pdet@Qmax, and Pdet@max were significantly higher in patients with BPH compared to those without BPH normal control subjects ($P < 0.01$), while Qmax was significantly lower in patients with BPH and BOO, compared with those with BPH and no BOO and control subjects ($P < 0.01$). No significant difference was observed in Puo, Pmuo, Pdet.Qmax, and Pdet@max between BPH patients without BOO, compared with control subjects. Qmax was lower in BPH patients without BOO, compared with control subjects, but with a certain overlap (Table 1).

B. Qmax was significantly higher, whereas Puo, Pmuo, Pdet. Qmax, and Pdet@max were significantly lower, in 23 of 27 BPH patients with BOO, compared with 4 of 27 BPH patients without BOO after suprapubic prostatectomy ($P < 0.01$) (Table 2). All the parameters mentioned above showed no difference in 4 of 27 BPH patients without BOO, before and after surgery (Table 3).

2. Clinical assessments and Free Qm

A. There was no difference in I-PSS, OSS, ISS, and TRUS scores between 52 BPH patients with BOO and 24 BPH patients without BOO. There was significantly higher RUV and lower Free Qm in 52 BPH patients with BOO and 24 BPH patients without BOO ($P < 0.05$) (Table 4). Table 5 shows the correlation of Free Qm and P/F study.

B. Of the 27 BPH patients, 23 patients were diagnosed with BOO according to P/F examination before surgery. After suprapubic prostatectomy, the I-PSS, OSS, ISS, Free Qm, and RUV were significantly changed in the 23 BPH patients with BOO ($P < 0.01$). There was also significant difference in I-PSS, OSS, and ISS before and after surgery in 4 BPH patients without BOO ($p < 0.05$) but no difference in terms of Free QM and RUV (Table 6). There was no difference in the removed prostatic weight between the two groups. The mean weight of removed prostatic weight was 24.8g (approximately 3–96g).

C. PN, LN, Pp, and Lp of SUPP were significantly higher in all BPH patients, compared with control subjects (Table 7). However, there was no significant difference in all parameters of SUPP between 52 of 76 BPH patients with BOO and 24 of 76 BPH patients without BOO (Tables 8 and 9). Pm was not changed between 76 BPH patients with and without BOO and control subjects.

Discussion

In general, BOO often happens in patients with BPH. It is well accepted that resection of the prostate is an effective surgical therapy for treating BPH.[7] And BOO is the only surgical indicator for BPH patients. Therefore, sensitivity and specificity of BOO diagnosis in BPH is critical for determining patient's treatment options. This retrospective study aimed to sort out a specific and sensitive approach to diagnose BOO in patients with BPH.

The simplest way to diagnose outlet obstruction is to test flow rate.[11] However, decreased Qmax is unable to differentiate the etiology of a weak stream: BOO, impaired detrusor function, or both.[12] It has been extensively studied that only the P/F study can confirm the pathology of BOO.[13–16] However, P/F study is an invasive evaluation, and it could cause urinary tract damage, bleeding, infection, and other complications, in addition to being relatively expensive, compared to other urodynamic studies. In this study, first we proved that P/F study is well correlated with the indications of BOO in 76 patients with BPH (Table 2), which is consistent with others. [17]

The accuracy of DRE for assessing PS is unfavorable.[9] TRUS is the imaging modality used most often to assess prostate volume, and it is more accurate than DRE.[18] We also evaluated

Table 1 P/F parameters among BPH patients (with and without BOO) and control

	Number	Age (Yr)	Qmax (ml/s)	PUo (cmH_2O)	PmU0 (cmH_2O)	Pdet@ Qmax (cmH_2O)	Pdet@ max (cmH2O)
BOO	52	66.1	8. 4	68. 9	52. 9	91. 1	99.2
		±8. 5	±6. 8**	±18. 7**	±15.5**	±18. 9**	±21. 0**
Non-BOO	24	65.0	16.7Δ.	30. 2	25.4	41. 6	50. 5
		±6.7	±5. 8	±9.2	±7. 3	±9.6	±18.5
Control	20	65. 4	23. 3	25.5	23. 5	36.2	44.0
		±6. 9	±6.7	±10.6	±10. 1	±13.2	±12.6
** : *P* <0. 01 BPH-BOO vs. Non-BOO or normal control; Δ: *P* <0. 05 BPH vs. normal control							

Table 2 P/F parameters in 23 BPH patients with BOO before and after surgery (X±s)

Group	Qmax (ml/s)	PUo (cmH_2O)	PmU0 (cmH_2O)	Pdet@Qmax (cmH_2O)	Pdet@max (cmH2O)
Before surgery	9. 2±5. 9**	70. 7±21. 5**	64. 7±14.5**	88. 1±23.3**	95. 0±22. 4**
After surgery	20. 2±7. 2	41. 8±9. 2	35.9±12.8	48.4±8. 1	55. 3±17.8
** : *P* <0.01					

Table 7 Urethra pressure of 76 BPH patients (with and without BOO) and control

Group	Case	PN (cmH_2O)	LN (cm)	Pp (cmH_2O)	Lp (cm)	Pm (cmH2O)
BOO	52	36.4±12.9 **	2.2±0.9**	38.1±10.3 **	4.0±1.3**	78.3±20.2
Non-BOO	24	35.3±10.9 **	2.1±0.9**	36.4±6.4**	3.9±1.1**	76.9±16.1
Control	20	20.6±4.1	1.2±0.7	19.1±4.2	2.9±0.6	64.9±9.6
** *P* <0.01 BPH patients vs. Control						

Table 8 Urethra pressure in 23 BPH patients with BOO before and after surgery (X±s)

Group	PN (cmH_2O)	LN (cm)	Pp (cmH_2O)	Lp (cm)	Pm (cmH2O)
Before surgery	38.8±14. 9**	2.3±0.9**	41. 3±10.4**	4.1 ±1.3**	81.6±21.6
After surgery	16.1±7. 5	1.0±O. 1	19. 1±5.6	2.0±O. 6	72. 9±13.2
**: P <0. 01					

Table 9 Urethra pressure of 4 BPH patients without BOO before and after surgery (X±s)

Group	PN (cmH_2O)	LN (cm)	Pp (cmH_2O)	Lp (cm)
Before surgery	35.8±9.4**	2.1±0.7**	37.6±8.4**	3.8±0.9**
After surgery	14.7±8.2	0.8±0.2	16.2±7.3	2.1±0.7
**: *P* <0. 01				

PS by DRE and TRUS. We presented that I-PSS, PS, free uroflowmetry (free Qm), and RUV did show the difference between BPH patients with and without BOO, but shoed no biological significance (Table 4). These results indicate that these four parameters are neither specific nor sensitive markers for BOO diagnosis in BPH patients, which is in line with others' observations.[19–21] Lastly, we demonstrated that SUPP could determine the degree and location of prostatic hyperplasia, but SUPP alone is not a specific marker to represent BOO (Tables 4 and 5).

Although P/F study could confirm the presence and status of BOO, it is unable to determine the degree and location of BOO.[6] On the other hand, SUPP could determine the degree and location of BOO but not the presence and status of BOO. Therefore, we suggest that a combination analysis of P/F study and SUPP will provide better specific and sensitive means to diagnose the BOO in BHP patients in clinical practice.

In summary, concomitant analysis of P/F study and SUPP could greatly improve the sensitivity and specificity of BOO diagnosis in patients with BPH, which will lead to earlier medical/surgical treatment and better evaluation of the efficacy of treatment.

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