Original Article

Does prostate size predict the urodynamic characteristics and clinical outcomes in benign prostate hyperplasia?

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Abstract

Aims: Bladder outlet obstruction (BOO) in large and small prostates is managed in a similar manner despite considerably different pathophysiology, which can result in higher failure rates. We investigate the clinical and urodynamic features and study the outcome of patients with benign prostate hyperplasia (BPH) according to their prostate size.

Subjects and Methods: We prospectively analyzed 100 BPH patients undergoing urodynamic study between January 2015 and August 2016 and divided them into two groups according to their prostate size: small (≤30 mL) and large prostate (>30 mL) groups. We compared the groups regarding age, International Prostate Symptom Score, maximal flow rate (Qmax), postvoided residual, serum prostate-specific antigen (PSA), prostate volume measured by ultrasonography (USG), and urodynamic findings.

Statistical Analysis Used: For testing the hypothesis, we used the Chi-square test, Student's *t*-test, and one-way analysis of variance when comparing between groups and conducted the logistic regression analysis for determining predictive factors of BOO.

Results: Although the total prostate volume significantly correlated with the PSA, patients with a small prostate had lower Qmax (5.27 ± 4.8 mL/s vs. 6.14 ± 6.66 mL/s; $P = 0.74$), higher incidence of abnormal baldder capacity (39.9% vs. 31.25%), lower voiding efficiency ($39.3 \pm 40.5\%$ vs. $40.57 \pm 32.11\%$), low compliance (44.4% vs. 31.3%), higher incidence of indeterminate detrusor contractions (38.9% vs. 37.5%), lower incidence of detrusor underactivity (33.3% vs. 28.1%), lower BOO index (40.9 ± 43.2 vs. 49.10 ± 44.48), lower bladder contractility index (77.8 ± 48.84 vs. 92.09 ± 52.79), and lower PdetQmax (51.44 ± 42.23 vs. 61.38 ± 42.01 cmH$_2$O). Small prostates had higher failed voiding trials postsurgery.

Conclusions: BOO patients with a small prostate showed poor urodynamic parameters and reported higher postoperative complications.

Keywords: Benign prostate hyperplasia, bladder contractility index, bladder outlet obstruction index, compliance, urodynamics

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INTRODUCTION

Benign prostate enlargement (BPE) is the most common cause of bladder outlet obstruction (BOO) in men with benign prostate hyperplasia (BPH) patients constituting the majority.[1] Although the prevalence of histological BPH in autopsy series have been reported to be much higher, the clinical symptoms are reported in 25% men at 55 years and 50% men at 75 years age, respectively.[2]

Although BPH is a commonly reported entity, its pathophysiology is still poorly understood. The enlarged prostate can result in voiding dysfunction either due to static (mechanical) or dynamic (bladder neck and prostate urethra smooth muscles) obstruction.[3] Although the classical literature is controversial regarding the direct relationship between prostate size and voiding dysfunction in BPH patients and its implications on the management as well as outcomes, a few recent studies have highlighted the role of predominant secondary bladder changes in small size prostates including high bladder neck, elevated smooth muscle tone in prostate/bladder neck, and increased prostate urethral angle in contrast to the primary obstructive component in large glands.[4]

Thus, cause of voiding dysfunction in BPH patients must be established before subjecting them to surgery for better patient outcomes as the management strategies differ in BOO due to small and large prostates. However, majority of urologists around the world manage the BOO in large and small prostates in a similar manner, resulting in higher rates of treatment failure, and patient dissatisfaction. Thus, the aim of this study is to add a significant body of evidence on the controversial topic of the effect of prostate size on urodynamic characteristics, factors predicting the BOO in small prostates, and clinical outcomes of BOO due to BPH.

SUBJECTS AND METHODS

This is a prospective observational study done between January 2015 and August 2016, in which 128 patients of clinical BPH were subjected to urodynamic evaluation. Clinical BPH was defined as lower urinary tract symptoms (LUTS), BPE, and/or BOO in men older than 40 years. Men <40 years, urinary tract infection (UTI), lower urinary tract or pelvic surgery, neurological diseases, radiotherapy of the pelvis, interstitial cystitis, bladder cancer, prostatitis, prostate cancer, ureteral stones, and urethral strictures were excluded from the study. Patients already taking alpha blockers/5 alpha reductase inhibitors were asked to stop these drugs for 1 week before inclusion in the study.[5] The various indications of urodynamics (UDS) in these patients were bothersome LUTS refractory to alpha blockers/5 alpha reductase inhibitors, repeated poor flow

rates on uroflowmetry, and before surgery in patients with small prostates. After exclusion, 100 patients were finally assessed and followed up over a period of time after the Institution Ethical Committee approval. Patient's detailed history was taken, and physical examination was done (including digital rectal examination), blood sample for prostate-specific antigen (PSA) measurement was taken and the International Prostate Symptoms Score (IPSS) questionnaire was completed. Then, the patients were subjected to transabdominal ultrasound imaging (USG) of the bladder and kidneys to assess the size of prostate and uroflowmetry was performed. For the analysis of uroflowmetry data, only patients voiding more than 150 mL were considered. Although transrectal US is more accurate measurement of prostate size, transabdominal USG was used in our study as it is less invasive, and enables additional measurement of postvoid residual urine (PVRU) and an evaluation of the upper tract. In addition, previous studies have highlighted the acceptable accuracy of transabdominal USG in prostate size measurement.[6,7] Urine culture was performed and patients with sterile results were subjected to multichannel UDS using Medtronic Logic G/2 model according to the recommendations by the International Incontinence Society (ICS) good UDS practices protocol.[8] Procedure was performed commonly in the sitting position and starts with retrograde filling cystometry at the rate of 10–20 mL/min. An 8 French dual micro-tipped catheter with infusion port is placed with the distal transducer in the bladder. A Foley balloon inflated to 3 mL was placed in the rectum and connected to a transducer to measure the intrabdominal pressure. Patient was placed in the sitting position and urodynamic study is completed. At the end of the study, urethral and rectal catheters are removed. All the urodynamic procedures were carried out under the guidance of a urologist using the same setting and protocols in our institution.

Study design

This was a prospective observational study, in which the clinical and urodynamic characteristics along with the outcomes of management were prospectively evaluated. Patients were divided into two groups based on the prostate size, i.e., <30 cc and >30 cc. This cutoff was taken based on the previous study done in Korea, where 32 cc prostate size cutoff was taken in LUTS/BPH patients with failed therapy according to a receiver operating characteristics curve analysis.[9] These two groups were then compared with regard to age, serum PSA levels, prostate volume, IPSS, maximum flow rate (Qmax), voided volume, PVRU, urodynamic findings, and final outcomes (surgery/conservative). The urodynamic variables analyzed were: maximal cystometric capacity, bladder compliance, detrusor pressure at peak flow (PdetQmax), bladder outlet obstruction index (BOOI), bladder capacity, involuntary detrusor contraction (IDC), bladder contractility

index (BCI), and voiding pattern determined by the provisional ICS nomogram. BOOI was calculated by formula: BOOI = PdetQmax − 2Qmax, and BCI was given by the formula: BCI = PdetQmax + 5Qmax, based on pressure flow studies.[10] BOO was divided into three categories based on BOOI as follows: obstruction (BOOI >40), no obstruction (BOOI <20), and equivocal (20≤ BOOI ≤40).[11] Detrusor underactivity (DUA) was defined as BCI <100. Detrusor overactivity (DO) was defined as any involuntary detrusor contraction during filling phase. Bladder compliance was calculated by dividing change in volume by change in pressure ($\Delta V/\Delta P$) and compliance <12.5 mL/cmH$_2$O was considered low. Voiding efficiency (%) was calculated from the mathematical equation, voided volume/voided volume + PVRU × 100. All these definitions conform to the standard terminology of international continence society.[8] Bladder capacity <300 mL and >550 mL were considered low and high, respectively. The patients were prospectively followed for 6 months with detailed history IPSS, physical examination, and uroflowmetry recorded at 3 months and 6 months, respectively.

Statistical analysis

To perform the descriptive analysis for the patient's characteristics and urodynamic findings, we calculated the means with standard deviations or the proportions of events. For testing the hypothesis, we used the Chi-square test, Student's *t*-test, and one-way analysis of variance when comparing between groups. We also conducted the logistic regression analysis for determining predictive factors of BOO. For all statistical tests, two-sided $P < 0.05$ was considered statistically significant. We managed all data using SPSS version 16.0 (SPSS, Inc., Chicago, IL, USA) and GraphPad Prism version 5.0 (GraphPad Software Inc., San Diego, CA, USA).

RESULTS

Table 1 shows the baseline characteristics of BPH patients having bothersome LUTS. The mean prostate volume was 26.38 ± 3.62 cc in small prostate and 51.9 ± 38.3 cc in large prostate group ($P = 0.006$). Patients with small prostate had higher IPSS scores (more storage symptoms), and the difference was statistically significant ($P < 0.046$). Small prostate patients suffered from more issues secondary to BOO, i.e., lower Qmax (5.27 ± 4.8 mL/s vs. 6.14 ± 6.66 mL/s; $P < 0.74$), higher PVRU, and lower voiding efficiency (39.3 ± 40.5% vs. 40.57 ± 32.11%; $P < 0.685$). The urodynamic characteristics of small prostate patients showed lower bladder capacity (358.66 ± 127.89 cc vs. 448.87 ± 129.23 cc), poor compliance (34.32 ± 40.7 vs. 46.23 ± 45.07 mL/cmH$_2$O), higher IDCs (38.9% vs. 37.5%), higher DUA (33.3% vs. 28.1%), higher unobstructed pattern on ICS nomogram (22.2% vs. 9.4%), lower BOOI (40.9 ± 43.2 vs. 49.10 ± 44.48), lower BCI (77.8 ± 48.84 vs. 92.09 ± 52.79), and lower PdetQmax (51.44 ± 42.23 vs. 61.38 ± 42.01) [Table 2 and Figure 1]. Two third patients (66%) underwent surgery and rest 1/3rd cases (34%) preferred to continue on conservative management. Transurethral resection of prostate (TURP) was the most common surgery performed (58%), followed by holmium laser enucleation of prostate (HoLEP) (8%). 24/36 patients in small prostate group underwent surgical intervention, including 8 patients with DUA (out of 12). 4/24 (11.11%) patients did not void on catheter removal (including 4 DUA, 50%) and were managed with prolonged catheterization followed by intermittent self-catheterization. 42 patients (65.6%) in large prostates (>30 g) underwent surgery, including 12 patients with DUA, out of which 2 cases (16.5%) failed to void. Thus, overall about 6% patients had failed voiding trial in 9.09% of total patients undergoing surgery and 20% of all DUA patients having failure to void after surgery. Rest, all patients showed improvement in symptoms (IPSS), Qmax, and PVRU ($P = 0.001$) [Table 3]. Logistic regression analysis was applied to patients with small prostate. In the univariate analysis, both age (odds ratio [OR], 1.061; $P = 0.037$) and serum PSA (OR, 0.001; $P < 0.001$) were found to be the significant predictors of BOO in small prostates. In the

Table 1: Baseline patient characteristics of benign prostate hyperplasia patients with bladder outlet obstruction by prostate volume

Variables	Mean±SD			P
	Total patients (*n*=100)	Small prostate (<30 g) (*n*=36)	Enlarged prostate (>30 g) (*n*=64)	
Age (years)	62.9±8.24	60.61±8.18	64.26±8.10	0.879
IPSS				
Voiding symptoms	11.33±5.53	8.03±3.87	9.02±4.32	0.046
Storage symptoms	7.85±3.70	11.88±5.42	6.76±3.12	0.211
USG				
Prostate volume (cc)	42.53±32.85	26.38±3.62	51.9±38.3	0.006
PVRU (mL)	225.5±164.12	238.7±165.64	201.73±164.28	0.945
Uroflowmetry				
Qmax (mL/s)	5.82±6.01	5.27±4.8	6.14±6.66	0.74*
Voiding efficiency (%)	40.10±35.01	39.3±40.5	40.57±32.11	0.685*

*Mann-Whitney U-test. IPSS: International Prostate Symptom Scoring, PVRU: Postvoid residual urine, Qmax: Peak flow rate. USG: Ultrasonography, SD: Standard deviation

**Table 2: Urodynamic characteristics of benign prostate hyperplasia patients**

Variables	Mean±SD			P
	Total patients (*n*=100)	Small prostate (<30 g) (*n*=36)	Large prostate (>30 g) (*n*=64)	
Capacity (cc), *n* (%)	415.73±134.7	358.66±127.89	448.87±129.23	
Normal	66 (66)	22 (55.6)	44 (68.8)	0.774
Small	12 (12)	8 (22.2)	4 (6.25)	
Large	22 (22)	6 (16.7)	16 (25)	
Compliance (mL/cmH$_2$O), *n* (%)	41.85±43.47	34.32±40.7	46.23±45.07	
Normal	36 (36)	20 (55.6)	16 (25)	0.147*
Low	64 (64)	16 (44.4)	20 (31.3)	
IDC, *n* (%)				
Negative	62 (62)	22 (61.1)	40 (62.5)	0.99
Positive	38 (38)	14 (38.9)	24 (37.5)	
DUA, *n* (%)				
Negative	70 (70)	24 (66.7)	46 (71.9)	0.578
Positive	30 (30)	12 (33.3)	18 (28.1)	
ICS nomogram, *n* (%)				
Unobstructed	14 (14)	8 (22.2)	6 (9.4)	0.360
Obstructed	56 (56)	18 (50)	38 (59.4)	
Equivocal	16 (16)	4 (11.1)	12 (18.8)	
No void	14 (14)	6 (16.7)	8 (12.5)	
BOOI	46.08±43.75	40.9±43.2	49.10±44.48	0.869
BCI	86.84±51.33	77.8±48.84	92.09±52.79	0.963
PdetQmax (cmH$_2$O)	57.73±41.93	51.44±42.23	61.38±42.01	0.961
PSA (ng/dL)	3.0±3.81	1.50±0.77	3.87±4.5	0.039
HDN, *n* (%)	20 (20)	6 (16.6)	14 (21.8)	0.624
AUR, *n* (%)	42 (42)	16 (44.5)	26 (40.6)	0.866
Failed voiding trial, *n* (%)	56 (50)	20 (55.6)	30 (46.9)	0.632
Comorbidities, *n* (%)	40 (40)	14 (38.9)	26 (40.6)	0.555

*Mann-Whitney U-test. IDC: Involuntary detrusor contractions, DUA: Detrusor underactivity, BOOI: Bladder outlet obstruction index, BCI: Bladder contractility index, PdetQmax: Detrusor pressure at peak flow rate, HDN: Hydronephrosis, AUR: Acute urine retention. SD: Standard deviation, PSA: Prostate-specific antigen

Table 3: Management and follow-up outcomes

	Total patients (*n*=100)	Small prostate (<30 g) (*n*=36)	Enlarged prostate (>30 g) (*n*=64)	P
Outcome, *n* (%)				
Surgery	66 (66)	24 (66.7)	42 (65.6)	0.88
Conservative	34 (34)	12 (33.3)	22 (34.4)	
Type of surgery, *n* (%)				
TURP	48 (48)	12 (50)	36 (56.3)	0.962
TUIP	10 (10)	10 (41.6)	-	
HoLEP	8 (8)	-	8 (12.5)	
Follow-up				
Failure to void, *n* (%)	6 (9.09)	4 (16.66)	2 (4.76)	
IPSS				
Voiding	15.67±5.65	15.66±4.34	14.31±5.78	0.39
Storage	11.98±4.23	11.45±5.1	11.54±5.23	0.95
Qmax (mL/s)	12.34±4.32	12.13±3.65	13.65±4.54	0.23
PVRU (mL)	32.34±12.4	42.16±17.51	24.46±11.63	0.001

TURP: Transurethral resection of prostate, HoLEP: Holmium laser enucleation, TUIP: Transurethral incision of prostate, IPSS: International Prostate Symptom Scoring, Qmax: Maximum flow rate, PVRU: Postvoid residual urine
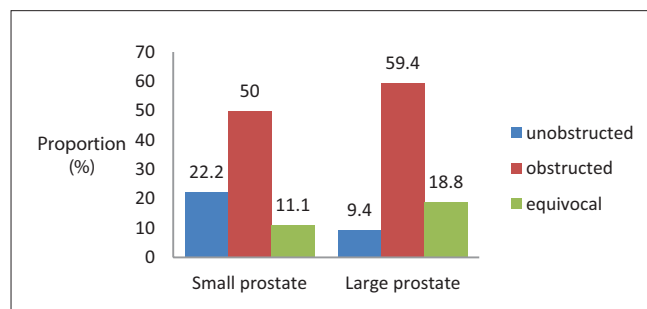
Figure 1: Comparison of the voiding patterns according to the International Incontinence Society nomogram between the prostate size groups

multivariate analysis, only serum PSA (OR, 1.944; $P=0.001$) was found to be the significant predictor of BOO in small prostate patients [Table 4].

DISCUSSION

LUTS in BPH are traditionally managed depending on their degree of botherness, irrespective of the size of prostate, although this issue is controversial. Recently, few studies have highlighted the exaggeration of voiding symptoms with increase in the size of prostate. Recent emphasis on the detailed

Table 4: Logistic regression analysis according to large and small prostates

Predictors	Unadjusted odds ratio (univariate)	CI (95%)	P	Adjusted OR (multivariate)	CI (95%)	P
Age	1.061	1.003–1.121	0.037*	1.032	0.968–1.099	0.333
PVRU	1.001	0.999–1.004	0.319	-	-	-
PSA	2.012	1.374–2.946	<0.001*	1.944	1.320–2.864	0.001*
Qmax	1.026	0.954–1.105	0.489	-	-	-
Voiding efficiency (%)	1.001	0.989–1.013	0.861	-	-	-
AUR	0.903	0.394–2.068	0.809	-	-	-
Comorbidities	1.444	0.613–3.404	0.400	-	-	-

*Significant predictor of bladder outlet obstruction. PVRU: Postvoid residual urine, PSA: Prostate-specific antigen, Qmax; Peak flow rate, AUR: Acute urine retention, SD: Standard deviation, OR: Odds ratio

pathophysiology of BPH has put some important insights into the difference in mechanism of BOO in cases of small and large prostates.[4] Despite this fact, all patients irrespective of their prostate size are managed in a similar manner resulting in higher treatment failure and dissatisfaction rate among the patients.

BPH presents with BOO in only 50%–80% patients.[12,13] Our study showed the presence of BOO in 50% patients of the small prostate group and 59.4% cases in the large prostate group. Kang *et al.*[4] reported 16.4% BOO in small prostates and 32.8% BOO in large prostates. The pathophysiology of BOO differs among small and large prostates. The storage symptoms (predominant in small prostates) could be due to the DO, myogenic/neurogenic failure, or behavioral problems (increased fluid intake and decreased vasopressin production), and obstructive/voiding dysfunction (predominates in larger prostates), occurs primarily due to obstruction.[14] Thus, we planned this study to put some light on the always controversial issue of role of prostate size with respect to BOO with respect to clinical and urodynamic bladder characteristics.

BPH patients with BOO complaint of initial increased detrusor contractility in the compensatory phase with normal bladder emptying.[15,16] Prolonged obstruction to urine outflow results in detrusor hypertrophy and bladder wall thickening, ultimately culminating in DUA and emptying failure.[17,18] This condition can become worst in small prostates, thereby resulting in the management dilemma. If surgery is offered at this stage, higher treatment failure, and need for long-term clean intermittent catheterization (CIC) are the possible associated risks that should be kept in mind. In this study, patients with small prostates had more bothersome LUTS (high IPSS), lower peak flow rate, and voiding efficiency compared to large prostates similar to the findings reported by Kang *et al.*[4] in their retrospective study on 659 men with BPH.

Review of literature reports IDCs in 20%–40% BPH patients clearly pointing to the fact that higher rate of IDCs is present in patients without BOO. Patients in our study

reported IDCs in 38.9% of cases of small prostates. Hirayama *et al.*[19] reported IDCs in 23.8% of the men with BOO and in 79.6% of the unobstructed group. Similarly, Kang *et al.*[4] reported IDCs in 30.4% small prostates and Gomes *et al.*[14] had IDCs in 48.8% small prostates with higher proportion in patients without BOO. Low compliance was reported in 44.4% patients with small prostates in our study. Kang *et al.*[4] reported similar findings while Oelke *et al.*[15] reported low compliance in small prostates with DO ± BOO. This higher rate of IDCs may be responsible for higher storage symptoms in small prostates.

The overall incidence of DUA in BPH population is unknown. DUA (33.3%) was higher in small prostates in this study, and this component was assessed by measuring the BCI which is a simpler numeric parameter and derivation of the Schafer's nomogram. The mean BCI was lower in small prostate. Oelke *et al.*[15] reported lower BCI in small prostates without BOO and with DO. Kang *et al.*[4] reported findings similar to our study. Thus, this study truly represents the higher proportion of secondary bladder abnormalities in small prostates as oppose to the BOO as the primary determinant in larger glands. Patients with small prostates with DUA represents the decompensated stage of BOO, especially in the presence of reduced Qmax and voiding efficiency along with elevated PVRU. These patients are at increased risk of postsurgical failure and higher patient dissatisfaction rate. Hence, they should be carefully evaluated and thoroughly counseled regarding the possible future complications.

This study clearly points to the fact that prolonged obstruction to the urine outflow results in detrusor hypertrophy (bladder wall thickening) which ultimately leads to detrusor hypocontractility, further emptying failure, and secondary bladder complications such as recurrent UTI and bladder calculi. Thus, low Qmax, high PVRU, and low voiding efficiency can result in bladder decompensation in BPH patients with small prostate.

All the patients in our study were prospectively followed up for 1 year at 3 monthly intervals. About 1/3rd patients in our study preferred not to undergo any surgical intervention and continued on medical management with uroflowmetry and

PVRU at 3 monthly intervals. Rest of the cases underwent surgical intervention to relieve their bothersome symptoms. TURP was the most common surgery performed followed by HoLEP. Patients with DUA (especially small glands) were explained regarding possible long-term CIC along with the long term need of anticholinergics in associated DOA after surgery.

Although TURP is still considered the gold standard in the surgical management of BPH, 5%–35% patients have persistent symptoms post-TURP possibly attributed to DUA.[20] Considering overall patients with DUA in our study, 20% patients of hypocontractile bladder had surgical failure. This is similar to 5%–35% failure rate reported in literature.[4] Rest all the patients showed positive response to surgery with improvements in IPSS, Qmax, and PVRU.

Age and serum PSA in univariate analysis and serum PSA on multivariate analysis were the significant predictors on BOO in the small prostate in our study. Kang *et al.*[4] reported serum PSA and Qmax as significant predictors of BOO in small prostates. Thus, these results indicate that the risk of BOO increases by 32% per one unit elevation in the serum PSA levels. These parameters can prove to be effective in the preoperative surgical planning in small prostate patients.

This is the first study of its kind which prospectively evaluated the urodynamic findings and outcomes of management in BPH patients based on the prostate size. The patients were selected using strict inclusion criteria, and hence, selection bias was eliminated. Although few similar retrospective studies are present in Western literature, no such study has been reported from the Indian subcontinent. Thus, our study provides a significant body of additional information on this important issue.

Limitations of this study

Small sample size and nonrandomized nature were the limitations of this study. BOOI and BCI were calculated using numerical equations which are simpler but nonvalidated. This may not be the best method, and hence, some difference from real values can be expected.

CONCLUSIONS

The detailed pathophysiology must be kept in mind before managing every case of symptomatic BPH. The patients with small prostate with BOO have higher component of obstruction related to secondary bladder changes. UDS should be performed in the symptomatic small prostate with refractory symptoms and high PVRU. The findings of UDS in such patients can predict the outcomes of management.

Conflicts of interest
There are no conflicts of interest.

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