Risk Factors for Bladder Diverticula in Patients with Benign Prostatic Enlargement: A Retrospective Study

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Purpose: Currently, the risk factors for bladder diverticula in patients with benign prostatic enlargement remain unclear. Therefore, this study aimed to identify independent risk factors and provide a clinical reference for early risk screening of this complication.

Materials and Methods: A total of 310 patients diagnosed with benign prostatic enlargement between January 2021 and December 2024 were included in this retrospective study. General clinical data and relevant clinical indicators were collected, and risk factors were identified through univariate and multivariate logistic regression analyses.

Results: Among the 310 patients with benign prostatic enlargement, 21 had bladder diverticula, representing an incidence of 6.77%. Multivariate logistic regression analysis revealed that total prostate volume (odds ratio [OR] = 1.067, 95% confidence interval [CI]: 1.023–1.113, $P = .002$) and intravesical prostatic protrusion (OR = 1.135, 95% CI: 1.056–1.219, $P < .001$) were independent risk factors for bladder diverticula in patients with benign prostatic enlargement.

Conclusion: Bladder diverticula occurred in 6.77% of patients with benign prostatic enlargement in this cohort. Total prostate volume and intravesical prostatic protrusion are independent risk factors for this complication and may serve as clinical markers for risk evaluation.

Keywords: benign prostatic hyperplasia; urinary bladder diverticulum; risk factors

INTRODUCTION

Benign prostatic enlargement (BPE) is a common urological disorder in middle-aged and elderly men. It is characterized by the nonmalignant proliferation of prostatic stromal tissue and glands, which results in prostate enlargement. This enlargement increases urethral resistance and causes bladder outlet obstruction (BOO), subsequently leading to lower urinary tract symptoms (LUTS) such as dysuria.[1] As the global population ages, the incidence of BPE is increasing annually, making it the primary urological health issue among elderly men in some countries.[2,3] Concurrently, bladder diverticula, which are often clinically silent complications, have attracted increasing clinical attention due to their associated adverse outcomes.[4,5] Epidemiological evidence indicates that bladder diverticula predominantly affect males aged 60 years and older, with an overall prevalence ranging from 1% to 8%. Its prevalence increases with advancing age.[6]

Structurally, a bladder diverticulum is a hernia-like protrusion of the bladder mucosa through weak areas in the muscular layer of the bladder wall.[7] This complex and delicate structure consists of the bladder mucosa, submucosal lamina propria, a thin muscular layer, and outer adventitia. Bladder diverticula are classified into congenital and acquired types, with the latter being more clinically common and often discovered incidentally during the management of conditions such as BPE.[8] Due to the absence of specific clinical manifestations, patients with bladder diverticula often experience delayed diagnosis and treatment, which may progress to severe complications.[9] These complications include recurrent urinary tract infections (UTIs) and acute urinary retention (AUR), as well as more serious conditions such as bladder stones, renal dysfunction, and even malignancies.[10,11] Furthermore, cases of bladder diverticulum rupture causing an acute abdomen have been reported, underscoring the severe health hazards associated with this condition.[12] Therefore, despite being asymptomatic in the early stages, the potential risks of bladder diverticula warrant significant clinical attention.

However, to date, there have been few dedicated studies on the risk factors associated with bladder diverticula in patients with BPE. Therefore, in the present study, we retrospectively analyzed the clinical data of 310 patients with BPE to thoroughly investigate the risk factors for the development of bladder diverticula, aiming to provide a clinical reference for risk screening of this complication.

MATERIALS AND METHODS

Patients

Between January 2021 and December 2024, 310 patients with BPE who had complete clinical data were in-

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Table 1. Univariate analysis of clinical characteristics in patients with benign prostatic enlargement with and without bladder diverticula (N = 310).

Variables	Diverticula Group (n = 21)	Non-diverticula Group (n = 289)	*P* Value
Age (years), median (IQR)	73 (67–78)	67 (61–73)	.010[a]
BMI (kg/m2), median (IQR)	24.5 (23.6–25.1)	24.6 (22.8–26.4)	.847[a]
Disease duration (years), median (IQR)	5.3 (3.1–7.8)	5.2 (2.8–7.7)	.908[a]
Hypertension, n (%)			.920[b]
Yes	8 (38.1)	112 (38.8)	
No	13 (61.9)	177 (61.2)	
Diabetes mellitus, n (%)			.750[b]
Yes	5 (23.8)	75 (26.0)	
No	16 (76.2)	214 (74.0)	
Coronary heart disease, n (%)			.950[c]
Yes	4 (19.0)	56 (19.4)	
No	17 (81.0)	233 (80.6)	
UTIs, n (%)			.415[c]
Yes	4 (19.0)	42 (14.5)	
No	17 (81.0)	247 (85.5)	
Bladder stones, n (%)			.402[c]
Yes	2 (9.5)	22 (7.6)	
No	19 (90.5)	267 (92.4)	
Renal insufficiency, n (%)			.781[c]
Yes	1 (4.8)	11 (3.8)	
No	20 (95.2)	278 (96.2)	
Constipation, n (%)			.792[c]
Yes	3 (14.3)	36 (12.5)	
No	18 (85.7)	253 (87.5)	
History of AUR, n (%)			.320[c]
Yes	3 (14.3)	27 (9.3)	
No	18 (85.7)	262 (90.7)	
Smoking, n (%)			.550[c]
Yes	4 (19.0)	46 (15.9)	
No	17 (81.0)	243 (84.1)	
Alcohol consumption, n (%)			.700[c]
Yes	3 (14.3)	37 (12.8)	
No	18 (85.7)	252 (87.2)	
IPSS (score), median (IQR)	22 (17–26)	18 (14–21)	.007[a]
QOL score, median (IQR)	4 (3–4)	4 (3–4)	.648[a]
Qmax (mL/s), median (IQR)	7 (6–9)	10 (8–12)	.002[a]
PVR (mL), median (IQR)	122 (94–148)	76 (55–96)	.001[a]
TPV (mL), median (IQR)	66 (54–76)	55 (47–63)	.004[a]
TZV (mL), median (IQR)	21 (13–28)	20 (12–27)	.876[a]
IPP (mm), median (IQR)	16 (13–19)	11 (9–14)	.001[a]
TPSA (ng/mL), median (IQR)	2.5 (1.7–3.4)	2.4 (1.6–3.2)	.695[a]
FPSA (ng/mL), median (IQR)	0.8 (0.5–1.1)	0.8 (0.5–1.0)	.897[a]

Abbreviations: AUR, acute urinary retention; BMI, body mass index; FPSA, free prostate-specific antigen; IPP, intravesical prostatic protrusion; IPSS, International Prostate Symptom Score; IQR, interquartile range; PVR, post-void residual urine volume; Qmax, maximum urinary flow rate; QOL, quality of life; TPSA, total prostate-specific antigen; TPV, total prostate volume; TZV, transition zone volume; UTIs, urinary tract infections.
a Mann-Whitney *U* test.
b Chi-square test.
c Fisher's exact test.

cluded as study subjects for retrospective analysis. The inclusion criteria for the study were as follows: (1) a clinical diagnosis of BPE confirmed by clinical examination without prior surgical intervention; (2) no history of urethral anatomical abnormalities, including urethral stricture, obstruction, or trauma; and (3) availability of complete clinical and follow-up data. The exclusion criteria for the study were as follows: (1) presence of congenital bladder diverticula; and (2) presence of mental disorders or cognitive dysfunction. At our center, all patients with newly diagnosed BPE routinely underwent bladder imaging as part of the initial evaluation. This study complied with the Declaration of Helsinki and was approved by the Ethics Committee of Affiliated Liyang People's Hospital of Kangda College of Nanjing Medical University (approval number 2025019). The requirement for informed consent was waived by the Ethics Committee because of the retrospective nature of the study.

Diagnostic Criteria and Data Collection

The diagnostic criteria for BPE with bladder diverticula were as follows: (1) clinical presentation of male LUTS, comprehensively evaluated by moderate-to-severe LUTS and auxiliary indicators, including an International Prostate Symptom Score (IPSS) $\geq$ 8, a prostate volume $\geq$ 30 mL, a maximum urinary flow rate (Qmax) < 10 mL/s, and a post-void residual urine volume (PVR) > 50 mL (patients did not need to meet all four criteria simultaneously); and (2) confirmation of bladder diverticula on imaging (ultrasonography and computed tomography).

We collected and analyzed the following data: age, body mass index (BMI), underlying diseases (hypertension, coronary heart disease, and diabetes mellitus), disease duration, history of AUR, UTIs, bladder stones, renal insufficiency, constipation, smoking, alcohol consumption, IPSS, Qmax, quality of life (QOL) score, PVR, total prostate volume (TPV), transition zone volume (TZV), intravesical prostatic protrusion (IPP), total prostate-specific antigen (TPSA), and free prostate-specific antigen (FPSA). Data on prior or concurrent medical therapy for BPE (including 5-alpha-reductase inhibitors or alpha-blockers) were not available in the retrospective records.

Table 2. Multivariate logistic regression analysis of risk factors for bladder diverticula in patients with benign prostatic enlargement.

Factors	B	SE	Wald $\chi 2$	*P* Value	OR	95% CI
Age	0.032	0.045	0.501	.479	1.033	0.946–1.128
IPSS	0.028	0.031	0.812	.368	1.029	0.970–1.091
Qmax	-0.065	0.058	1.256	.262	0.937	0.836–1.050
PVR	0.002	0.003	0.444	.505	1.002	0.996–1.008
TPV	0.065	0.021	9.524	.002	1.067	1.023–1.113
IPP	0.127	0.034	12.842	< .001	1.135	1.056–1.219

Abbreviations: CI, confidence interval; IPP, intravesical prostatic protrusion; IPSS, International Prostate Symptom Score; OR, odds ratio; PVR, post-void residual urine volume; SE, standard error; TPV, total prostate volume; Qmax, maximum urinary flow rate.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 20.0; IBM Corp, Armonk, NY, USA). To compare continuous variables, normality was first assessed using the Shapiro-Wilk test. Variables with a normal distribution were compared using Student's *t*-test and are reported as the mean ± standard deviation (SD), whereas variables with a non-normal distribution were compared using the Mann-Whitney U test and are reported as the median (interquartile range, IQR). For categorical variables, the chi-square test was used for intergroup comparisons when the expected frequency of every cell in a contingency table was ≥ 5. Fisher's exact test was used if any cell had an expected frequency < 5 to ensure statistical validity.

Before multivariate logistic regression was performed, the linearity assumption between continuous quantitative predictors and the logit of the outcome was verified via the Box-Tidwell test. Each continuous variable and its natural logarithm interaction terms were entered into a preliminary logistic model; covariates with interaction term $P > .05$ met the linear logit assumption and were included in the regression model. All variables for which P was < .05 in the univariate analysis were entered into the initial full model. The backward stepwise elimination method was adopted for variable screening, with a variable entry criterion of $P = .05$ and an elimination criterion of $P = .10$. After iterative screening, the final multivariate regression model was constructed to identify independent risk factors. All *P* values were two-tailed, and a P value of less than .05 was considered to indicate statistical significance.

Owing to the retrospective nature of this study, a prospective sample size calculation was not performed prior to data collection. This cohort included all eligible patients who met the inclusion and exclusion criteria enrolled between January 2021 and December 2024, totaling 310 individuals with complete data. To assess the adequacy of this sample size in detecting the identified risk factors, a post-hoc power analysis was conducted based on the observed effect sizes from our final multivariate logistic regression model. When a two-tailed α level of .05 was used, the statistical power for detecting TPV as a significant factor (odds ratio [OR] = 1.067) was 82.3%, and that for IPP (OR = 1.135) was 95.1%. These results indicate that the study had adequate statistical power (> 80%) to identify these independent risk factors for bladder diverticula in patients with BPE.

RESULTS

Univariate Analysis of BPE Complicated with Bladder Diverticula

Among the 310 patients with BPE, 21 had bladder diverticula, representing a prevalence of 6.77%. All cases were confirmed by a combination of ultrasonography and computed tomography. Seventeen patients had a single diverticulum, whereas 4 presented with multiple diverticula, with the largest measuring 3.2 cm in diameter. The diverticula were located on the lateral wall of the bladder in 18 patients and on the posterolateral wall in 3 patients. Seventeen patients had no obvious diverticulum-related symptoms, whereas 4 presented with recurrent UTIs. All patients subsequently underwent transurethral resection of the prostate (TURP). Univariate analysis revealed that age (median 73 vs. 67 years), IPSS (median 22 vs. 18), Qmax (median 7 vs. 10 mL/s), PVR (median 122 vs. 76 mL), TPV (median 66 vs. 55 mL), and IPP (median 16 vs. 11 mm) were significantly associated with bladder diverticula in patients with BPE (all $P < .05$). No statistically significant differences were observed between the two groups regarding BMI, hypertension, diabetes mellitus, coronary heart disease, UTIs, bladder stones, renal insufficiency, constipation, smoking, alcohol consumption, disease duration, history of AUR, QOL score, TPSA, FPSA, or TZV (all $P > .05$) (**Table 1**).

Multivariate Analysis of BPE Complicated With Bladder Diverticula

Variables demonstrating statistical significance in the univariate analysis were entered into the initial multivariate logistic regression model, followed by backward stepwise elimination to determine independent risk factors. The results revealed that TPV (OR = 1.067, 95% CI: 1.023–1.113, $P = .002$) and IPP (OR = 1.135, 95% CI: 1.056–1.219, $P < .001$) were independent risk factors for bladder diverticula in patients with BPE (**Table 2**).

DISCUSSION

Bladder diverticula, also known as pseudodiverticula, originate from focal defects in the bladder muscular layer and are primarily caused by a prolonged increase in intravesical pressure.[13] Although bladder diverticula are often asymptomatic in early stages, disease progression may trigger multiple complications.[14] As the diverticulum enlarges, space-occupying compression of the surrounding organs becomes apparent, causing unexplained abdominal pain and discomfort, which is easily misdiagnosed as intestinal obstruction or abdominal aortic aneurysm.[15,16] It is also an extremely rare cause of iliac vein compression syndrome.[17] In addition, when the diverticulum is located near the ureteral orifice, it can directly compress the ureter, leading to hydronephrosis, impaired renal function, and even renal failure.[18] Cases of inguinal bladder hernias caused by giant bladder diverticula have been frequently reported.[19] When the diameter of the diverticulum exceeds 5.15 cm, the risk of AUR increases significantly,[10] and large

diverticula themselves may lead to incomplete emptying, UTIs, diverticulitis, bladder calculi, and malignant diverticular tumors.[20] Chronic inflammation or stone irritation within the diverticular mucosa can cause dysplasia, squamous metaplasia, and leukoplakia, substantially increasing the risk of malignant transformation.[21] Moreover, the absence of an intact muscular layer in diverticular walls facilitates early transmural tumor invasion.[22] The overall incidence of malignant tumors arising in bladder diverticula is 0.8% to 14.3%,[14] accounting for approximately 1% of all bladder cancers,[23] with the predominant histological types being urothelial carcinoma, squamous cell carcinoma, adenocarcinoma, small cell carcinoma, and sarcoma.[24-26] The presence of a bladder diverticulum even poses a risk of intraoperative bladder explosion during transurethral prostatectomy.[27] Thus, the health threats associated with bladder diverticula in patients with BPE cannot be ignored.[28] Analyzing their risk factors is crucial for early clinical prevention and therapeutic management. BPE complicated with bladder diverticula is closely related to persistent elevation of intravesical pressure, mainly resulting from BOO caused by increased prostate volume.[29,30] In this study, after rigorous screening against predefined inclusion and exclusion criteria, 310 patients with BPE were enrolled, among whom 21 patients were diagnosed with bladder diverticula, representing an incidence of 6.77%, which is consistent with previously published population-level estimates.[7] Furthermore, univariate analysis in this study revealed that age, IPSS, Qmax, PVR, TPV, and IPP were associated with bladder diverticula in patients with BPE, whereas multivariate logistic regression analysis confirmed that TPV and IPP were independent risk factors for this complication. The core pathological change of BPE is hyperplasia of prostatic glandular and stromal elements, leading to increased prostate volume, which compresses the urethra and elevates bladder emptying resistance.[31] Long-term BOO causes compensatory detrusor hypertrophy, with thickening and disordered arrangement of bladder wall muscle bundles, eventually leading to the formation of localized weak areas in the muscular layer.[32] Sustained elevation of intravesical pressure forces the bladder mucosa to herniate outward through these weak areas, forming acquired bladder diverticula. As a direct anatomical indicator of BPE, TPV is positively correlated with urethral resistance and thus represents a key driver of increased intravesical pressure.[33] Our results are consistent with those of previous studies, such as those by Hossain et al. and Lim et al.[34,35] who reported a strong correlation between TPV and BOO. IPP quantifies the degree of prostate protrusion into the bladder lumen. Increased IPP elevates the bladder neck, creating a "ball-valve" mechanism that further exacerbates functional voiding resistance.[36] Simultaneously, the protruding prostate tissue may directly compress the adjacent bladder base, causing localized ischemia and detrusor muscle fiber atrophy, thereby promoting diverticulum formation. Our study revealed that the risk of developing a bladder diverticulum increases with increasing severity of IPP, a finding consistent with that reported by Al Rashed et al.[37] However, the study by Eze et al. did not observe an increased probability of bladder diverticula in patients with BPE and greater IPP.[38] This discrepancy may be attributable to differences in study populations, ethnicity, or sample sizes. Notably, although age, IPSS, Qmax, and PVR were associated with diverticula in the univariate analysis, they did not retain statistical significance in the multivariate model. These findings suggest that structural anatomical parameters (TPV and IPP) should be prioritized over purely functional urodynamic parameters when assessing the risk of diverticular complications in patients with BPE.

Study Limitations

However, this study has several limitations. First, only 21 patients had bladder diverticula, resulting in a low event count for multivariate logistic regression. The full model contained six predictors and failed to satisfy the events per variable (EPV) $\geq$ 10 criterion, which may have caused overfitting, unstable regression coefficients, and biased odds ratios. Second, the single-center retrospective design may have introduced selection bias. Third, the lack of a detailed medication history for BPE precludes adjustment for potential confounding effects of pharmacotherapies (such as 5-alpha-reductase inhibitors or alpha-blockers) on bladder function and diverticulum formation. To more comprehensively validate these conclusions, prospective multicenter studies with larger sample sizes and prespecified statistical analysis plans are warranted.

CONCLUSIONS

In this cohort of 310 patients with BPE, bladder diverticula were identified in 21 patients, corresponding to an observed prevalence of 6.77%. Multivariate logistic regression analysis revealed that TPV and IPP were independent risk factors associated with the presence of bladder diverticula, and may serve as clinical indicators for evaluating the risk of this complication.

SUMMARY

In men with an enlarged prostate, a larger prostate volume and greater growth into the bladder increase the risk of bladder outpouchings (diverticula). Checking these features helps identify patients at higher risk.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

REFERENCES

1. Manov JJ, Mohan PP, Kava B, Bhatia S. Benign Prostatic Hyperplasia: A Brief Overview of Pathogenesis, Diagnosis, and Current State of Therapy. Tech Vasc Interv Radiol. 2020;23:100687.
2. GBD 2019 Benign Prostatic Hyperplasia Collaborators. The global, regional, and national burden of benign prostatic hyperplasia

in 204 countries and territories from 2000 to 2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Healthy Longev. 2022;3:e754-e776.
3. Launer BM, McVary KT, Ricke WA, Lloyd GL. The rising worldwide impact of benign prostatic hyperplasia. BJU Int. 2021;127:722-728.
4. Choi JB, Min SK. Complicated urinary tract infection in patients with benign prostatic hyperplasia. J Infect Chemother. 2021;27:1284-1287.
5. Tortorelli AP, Rosa F, Papa V, Alfieri S, Doglietto GB. Giant bladder diverticulum. Updates Surg. 2011;63:63-66.
6. Iscaife A, Dos Anjos G, Barbosa C Neto, Nahas WC, Srougi M, Antunes AA. The role of bladder diverticula in the prevalence of acute urinary retention in patients with BPH who are candidates to surgery. Int Braz J Urol. 2018;44:765-770.
7. Chawla S, Bhatt S, Tandon A, Meena G, Dangwal S. Bilateral Hutch diverticula in an elderly male: Revelation of an unknown past. SA J Radiol. 2020;24:1963.
8. Lahham S, Gutierrez S. Diagnosis of Bladder Diverticula with Point-of-Care Ultrasound. Clin Pract Cases Emerg Med. 2021;5:466-467.
9. Ibrahimi A, Kallat A, Ziani I, El Sayegh H, Benslimane L, Nouini Y. Spontaneous Intraperitoneal Rupture of Bladder Diverticulum: A Rare Cause of Peritonitis. Case Rep Urol. 2020;2020:8880748.
10. Favorito LA. Bladder diverticula with more than 5 cm increases the risk of acute urinary retention in BPH. Int Braz J Urol. 2018;44:662-663.
11. Matković A, Ferenc T, Jurjević N, et al. Urothelial carcinoma in a urinary bladder diverticulum: A case report and review of the literature. Radiol Case Rep. 2023;18:1169-1174.
12. Coco D, Leanza S. Rupture of urinary bladder diverticulum. Pan Afr Med J. 2021;39:171.
13. Gazzah W, Ben Taher S, Masmoudi S, Hamza M, Naouar S, Salem B. Management of multiple giant bladder diverticula: a comprehensive approach - a case report. Ann Med Surg (Lond). 2024;86:4187-4190.
14. Zhu GG, Siegel CL, Rogers DM, et al. Urinary bladder diverticula: imaging features and complications. Abdom Radiol (NY). 2025;50:3584-3598.
15. Han Z, Sun X, Ma X, Li X, Zhang L, Shi H. Bladder diverticulum accompanied by abdominal pain: a case description. Quant Imaging Med Surg. 2024;14:1215-1218.
16. Shaked G, Czeiger D. Distended urinary bladder and diverticulum-a rare cause of large-bowel obstruction. Am J Surg. 2009;197:e23-e24.
17. Wang X, Chen X, Gao X, Feng H. Iliac vein compression syndrome caused by a large bladder diverticulum: Case report and literature review. J Int Med Res. 2022;50:3000605221123670.
18. Ananthapadmanabhan S, Williams Z, Wang H, Jeffery N, Mehan N. Large bladder diverticulum causing direct extrinsic compression of the left ureter. Urol Case Rep. 2024;55:102779.
19. Hinojosa D, Joseph UA, Wan DQ, Barron BJ. Inguinal herniation of a bladder diverticulum on PET/CT and associated complications. Clin Imaging. 2008;32:483-486.
20. Janardanan S, Nigam A, Moschonas D, Perry M, Patil K. Urinary Bladder Diverticulum: A Single-Center Experience in the Management of Refractory Lower Urinary Symptoms Using a Robotic Platform. Cureus. 2023;15:e42354.
21. DeWitt-Foy ME, Anele UA, Accioly JPE, Sharpe MG, Michael P, Angermeier KW. Cancer risk in bladder diverticula: a large institutional analysis of risk and management. Int Urol Nephrol. 2023;55:541-546.
22. Fang CW, Hsieh VC, Huang SK, Tsai IJ, Muo CH, Wu SC. A population-based cohort study examining the association of documented bladder diverticulum and bladder cancer risk in urology patients. PLoS One. 2019;14:e0222875.
23. Walker NF, Gan C, Olsburgh J, Khan MS. Diagnosis and management of intradiverticular bladder tumours. Nat Rev Urol. 2014;11:383-390.
24. Moussa M, Abou Chakra M. Urothelial carcinoma arising from a bladder diverticulum containing multiple stones: A case report. Urol Case Rep. 2018;20:80-82.
25. Dong WX, Ping YX, Liang WC, Jian LZ, Lin ZJ. Small cell carcinoma of the urinary bladder diverticulum: a case report and review of the literature. J Cancer Res Ther. 2013;9:151-153.
26. Omeroglu A, Paner GP, Wojcik EM, Siziopikou K. A carcinosarcoma/sarcomatoid carcinoma arising in a urinary bladder diverticulum. Arch Pathol Lab Med. 2002;126:853-855.
27. Vincent DP. Bladder explosion during transurethral resection of prostate: Bladder diverticula as an additional risk factor. Urol Ann. 2017;9:68-70.
28. Aijaz P, Farooqi Baloch K, Faiz H, et al. Clinical Presentation, Tumor Characteristics, and Management of Intradiverticular Transitional Cell Carcinoma of the Urinary Bladder: A Systematic Review. Cureus. 2024;16:e62974.
29. Agarwal DK, Krambeck AE. Holmium laser enucleation of the prostate is an effective treatment in patients with concomitant bladder diverticula and outlet obstruction. World J Urol. 2018;36:87-90.
30. Li X, Liu T, Wang L. Diagnosis of bladder diverticulum: Retrograde cystography CT more valuable. J Xray Sci Technol. 2015;23:481-487.
31. Aaron L, Franco OE, Hayward SW. Review of Prostate Anatomy and Embryology and the Etiology of Benign Prostatic Hyperplasia. Urol Clin North Am. 2016;43:279-288.
32. Speakman MJ, Cheng X. Management of the complications of BPH/BOO. Indian J Urol.

2014;30:208-213.
33. van Venrooij GE, van Melick HH, Eckhardt MD, Boon TA. Diagnostic and predictive value of voiding diary data versus prostate volume, maximal free urinary flow rate, and Abrams-Griffiths number in men with lower urinary tract symptoms suggestive of benign prostatic hyperplasia. Urology. 2008;71:469-474.
34. Hossain AK, Alam AK, Habib AK, et al. Comparison between prostate volume and intravesical prostatic protrusion in detecting bladder outlet obstruction due to benign prostatic hyperplasia. Bangladesh Med Res Counc Bull. 2012;38:14-17.
35. Lim KB, Ho H, Foo KT, Wong MY, Fook-Chong S. Comparison of intravesical prostatic protrusion, prostate volume and serum prostatic-specific antigen in the evaluation of bladder outlet obstruction. Int J Urol. 2006;13:1509-1513.
36. Gharbieh S, Reeves F, Challacombe B. The prostatic middle lobe: clinical significance, presentation and management. Nat Rev Urol. 2023;20:645-653.
37. Al Rashed AA, Isa QM, Mahdi A, et al. Clinical Outcomes of Intravesical Prostatic Protrusion in Patients With Benign Prostatic Hyperplasia. Cureus. 2024;16:e52541.
38. Eze BU, Ani CO, Mbaeri TU. Is intravesical prostatic protrusion associated with more complications in benign prostatic hyperplasia patients?. Low Urin Tract Symptoms. 2021;13:468-474.