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THE RELATIONSHIP BETWEEN BENIGN PROSTATIC HYPERPLASIA AND UROLITHIASIS

Ermamatov Muhammadali Dilshodjon o‘g‘li
Central Asian Medical University
First-Year Resident in Urology
muxa0407@icloud.com
+998945561757
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Abstract: Benign prostatic hyperplasia (BPH) and urolithiasis may coexist in older men, but the clinical significance of their association depends on stone location and the presence of functional obstruction. This narrative review examines anatomical, functional, and metabolic mechanisms linking BPH with urinary stone disease, with particular emphasis on bladder calculi. Selected clinical investigations and European Association of Urology guidelines were reviewed. Quantitative evidence included surgical cohorts of 271 and 332 patients, a prospective metabolic comparison, and a randomized treatment study involving 100 men. The findings support an interaction between impaired bladder emptying, intravesical prostatic protrusion, and lithogenic urinary characteristics. Bladder stones were reported in 9.9% of one surgical cohort and in 94 of 332 patients in another. These observations support individualized evaluation of obstruction rather than attributing all urinary stones to prostate enlargement. Management should address stone burden, bladder function, and relevant metabolic susceptibility. The available evidence does not establish BPH as a direct cause of kidney stone formation. Accurate distinction between secondary bladder stones and upper urinary tract calculi is therefore essential for diagnosis, treatment selection, and recurrence prevention.

Keywords: benign prostatic hyperplasia; urolithiasis; bladder calculi; benign prostatic obstruction; urinary stasis; intravesical prostatic protrusion; metabolic evaluation.

Introduction

The relationship between benign prostatic hyperplasia and urinary stone disease presents an important diagnostic and therapeutic problem. When these conditions occur together, it is necessary to determine whether they are causally related, share contributing factors, or represent separate disorders. Their coexistence alone does not establish a single mechanism. A clinically useful assessment must identify the location and probable origin of the stone, evaluate urinary drainage, and determine which abnormalities are likely to remain after treatment. Precise terminology is fundamental. BPH refers to benign proliferation of prostatic tissue, while benign prostatic obstruction (BPO) describes impaired urinary outflow attributable to the prostate. Lower urinary tract symptoms (LUTS) describe the patient’s experience rather than a specific underlying mechanism. Consequently, prostate enlargement, symptoms, and functional obstruction should not be treated as equivalent findings. A patient may have significant symptoms without demonstrable obstruction, making symptom-based assumptions about stone formation unreliable[1,5].

The term urolithiasis includes calculi occurring at different levels of the urinary tract. The association with BPH is most clearly established for bladder stones. European Association of Urology (EAU) guidance identifies bladder outlet obstruction as a predisposing factor in approximately 45–

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79% of adult vesical calculi. This percentage describes the contribution of obstruction among patients with bladder stones; it does not indicate that 45–79% of men with BPH develop calculi. Correct interpretation of the denominator is essential when explaining risk[2].

An additional distinction concerns stone formation versus stone retention. A calculus may originate in the upper urinary tract, enter the bladder, and remain there when emptying is impaired. Subsequent growth may depend on local conditions and urinary composition. This mechanism provides a plausible connection between previous renal stone disease and later bladder calculi without demonstrating that BPH caused the original kidney stone [3,7]. The relationship also requires careful interpretation of research populations. Men undergoing prostate surgery are a selected group whose disease has already prompted intervention. Stone frequency in such patients cannot automatically be applied to the general male population. Similarly, a metabolic comparison investigates different questions from a randomized treatment trial. The former may identify characteristics associated with stone formation, whereas the latter evaluates outcomes after specific interventions.

These distinctions have practical implications. If all stones are attributed to obstruction, metabolic susceptibility may be overlooked. Conversely, if treatment focuses exclusively on the calculus, clinically important impairment of bladder emptying may remain unresolved. An integrated assessment should therefore consider both the immediate reason for treatment and the factors potentially responsible for future complications. The objective of this review is to examine the relationship between BPH and urolithiasis through published patient statistics, anatomical findings, metabolic observations, and treatment outcomes. Particular attention is given to separating association from causation and to interpreting numerical results within their original clinical context [2,3].

Materials and methods

A narrative review was conducted to examine the relationship between benign prostatic hyperplasia and urolithiasis, with particular attention to factors contributing to bladder stone formation. Relevant PubMed-indexed publications, available full-text articles, and European Association of Urology guidelines were reviewed. Search terms included “benign prostatic hyperplasia,” “bladder stones,” “urinary stasis,” “intravesical prostatic protrusion,” and “metabolic abnormalities.”

Four clinical studies by Childs, Kim, Huang, and Hasan and their colleagues were examined in detail. Information on study design, patient numbers, clinical characteristics, diagnostic findings, and treatment outcomes was extracted from each publication. Particular attention was given to prostatic obstruction, impaired bladder emptying, and urinary metabolic changes.

The findings were compared descriptively, taking differences in patient selection, study methods, and follow-up duration into account. Numerical data and reported levels of statistical significance were obtained from the original publications. Percentages calculated from published patient counts were identified in the text. No meta-analysis was performed, and no new patient data were collected. The analysis was based entirely on previously published evidence.

Results and discussion

The reviewed studies support a multifactorial explanation for bladder stone formation in patients with BPH. Anatomical configuration, impaired drainage, and urinary chemistry appear to provide complementary information. However, the evidence does not justify interpreting every significant association as an independent causal mechanism.

Kim and colleagues retrospectively evaluated 271 patients undergoing transurethral resection of the prostate (TURP). Bladder stones were reported in 9.9% of the cohort. Older age ($P = 0.020$),

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greater intravesical prostatic protrusion (IPP; $P < 0.001$), and lower maximum urinary flow rate ($P = 0.019$) were independently associated with stone presence. Their prediction model achieved an area under the receiver operating characteristic curve of 0.850.

These findings suggest that evaluating prostatic configuration may be more informative than considering enlargement alone. Nevertheless, the model's performance within the original dataset does not establish accuracy in other populations. External validation would be required before using it to estimate an individual patient's probability of stone disease. Moreover, an association between IPP and existing stones does not demonstrate that modifying IPP prevents a first stone.

Huang and colleagues examined 332 surgical patients, including 94 with bladder calculi and 238 without calculi. The stone proportion was 28.3%, calculated as 94/332. Mean ages were 68.5 ± 7.6 years and 69.1 ± 7.8 years, respectively ($P = 0.51$). IPP was greater in the stone group ($P < 0.001$), whereas post-void residual volume and peak urinary flow did not differ significantly. In the analysis excluding patients with indwelling catheters, positive urine cultures occurred in 10.7% versus 4.0% ($P = 0.046$).

The difference between the 9.9% and 28.3% proportions should not be interpreted as a temporal increase in stone disease. Different referral patterns, eligibility criteria, and surgical indications may produce substantially different study populations. Neither proportion is a representative estimate for all men with BPH. Similarly, the urine-culture association does not demonstrate that routine antibiotic administration prevents stone formation.

The metabolic study by Childs and colleagues provides a different perspective. The full-text tables identify 30 patients with bladder stones and 27 controls. Suitable 24-hour urine collections were available for 27 stone formers and 21 controls. Median urinary pH was 5.9 versus 6.4 ($P = 0.02$), and uric acid supersaturation was 2.2 versus 0.6 ($P < 0.01$). Supersaturation is reported here as an index, not as a concentration.

The 30% figure represents subsequent prostate surgery, not recurrent bladder stones. These outcomes should remain separate when reporting the study. The result informs counseling about the possibility of later intervention, but the eligibility criteria and follow-up period limit extrapolation to patients with larger calculi or different clinical characteristics.

Taken together, the studies illustrate why a single variable cannot adequately characterize the relationship. An anatomical association, a biochemical difference, and an improvement following surgery each address different parts of the clinical problem. Their findings are compatible with an integrated explanation, but they should not be merged into a universal prediction or treatment rule.

Diagnostic assessment should establish whether the bladder stone accompanies clinically relevant obstruction. EAU guidance recommends evaluation of urinary flow, residual urine, urinary pH, relevant blood chemistry, and stone composition, with culture and upper tract imaging when indicated. A urodynamic study summarized in the guideline found obstruction in only 51% of 46 older men with bladder stones, demonstrating that a calculus alone is insufficient evidence of obstruction.

Treatment selection should incorporate prostate characteristics, comorbidities, anesthetic suitability, patient preferences, and expected benefit. EAU male LUTS guidance identifies bladder stones, recurrent or refractory retention, recurrent infections, and upper tract dilatation attributable to BPO among circumstances in which surgery is usually required. Applying this guidance still requires assessment of the relationship between the complication and the underlying obstruction.

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Recurrence prevention requires a separate consideration of metabolic risk. EAU guidance recommends reliable stone analysis and basic metabolic assessment, with specific investigation for high-risk stone formers. When indicated, two consecutive 24-hour urine collections are obtained. General measures include appropriate hydration, balanced nutrition, normal dietary calcium intake, and avoidance of excessive salt consumption. Fluid recommendations should be adapted to medical restrictions. These principles should not be represented as quantified BPH-specific reductions in recurrence because that evidence remains limited.

Several limitations should guide interpretation. This review used selective searching and did not apply a formal risk-of-bias instrument. The studies involved different populations and cannot provide a pooled estimate. Statistical significance does not necessarily indicate a large clinical effect, and absence of significance does not establish equivalence. Future investigations should distinguish stone location, document functional obstruction, use consistent denominators, and report recurrent stones separately from repeat prostate surgery.

Conclusion

The relationship between benign prostatic hyperplasia and urolithiasis is most clearly established for bladder stones. Available evidence suggests that stone formation results from the interaction of impaired bladder emptying, prostatic anatomy, and metabolic abnormalities. Urinary stasis is an important contributing factor, but it does not fully explain why calculi develop in some patients and remain absent in others with similar obstructive symptoms.

Clinical evaluation should therefore extend beyond the identification of prostate enlargement. Assessment of functional obstruction, residual urine, stone location and composition, and relevant metabolic characteristics can help identify the factors contributing to disease in an individual patient. The coexistence of BPH and kidney stones should not automatically be interpreted as a direct causal relationship.

Treatment should address both the calculus and clinically significant underlying abnormalities. Combined stone removal and surgical correction of prostatic obstruction may benefit appropriately selected patients, while treatment decisions should account for comorbidities, operative risk, and patient preferences. Follow-up should assess urinary function and stone recurrence, with preventive measures tailored to the identified risk factors.

Further prospective studies with standardized diagnostic criteria and longer follow-up are needed to clarify which patients benefit most from combined treatment and how metabolic prevention influences recurrent bladder stone formation.

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