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CORRELATION BETWEEN PROSTATE VOLUME AND SEVERITY OF LOWER URINARY TRACT SYMPTOMS AMONG DIABETIC PATIENTS WITH BENIGN PROSTATIC HYPERPLASIA

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Abstract

Background: Benign prostatic hyperplasia (BPH) and lower urinary tract symptoms (LUTS) are prevalent urological conditions in aging men. Diabetes mellitus (DM) further impacts urological health through neuromuscular effects on the bladder. This study investigates the correlation between prostate volume (PV) and LUTS severity in diabetic men to strengthen evidence-based clinical management. Objectives: To evaluate the correlation between prostate volume and LUTS severity, assessed via the International Prostate Symptom Score (IPSS), among diabetic patients with BPH. Material and Methods: A prospective cross-sectional study was conducted on 138 diabetic BPH patients (aged ≥40 years) presenting with LUTS for abdominopelvic ultrasound. Demographic and clinical data (age, PV, diabetes duration) were collected via a structured questionnaire. LUTS severity was assessed using the IPSS (mild: 0–7, moderate: 8–19, severe: 20–35) along with a Quality of Life (QoL) single-item score (1–7). PV was measured via transabdominal ultrasonography using the standard ellipsoid formula. Statistical analyses included Pearson's correlation coefficient, Chi-Square, and One-Way ANOVA. Results: A significant moderate positive correlation was observed between PV and total IPSS ($r = 0.513$, $p < 0.01$). Furthermore, longer duration of diabetes was significantly associated with greater PV ($p < 0.01$) and heightened LUTS severity ($p < 0.01$). Conclusion: Prostatic enlargement significantly influences LUTS severity in diabetic BPH patients ($r = 0.513$). Prolonged diabetes duration exacerbates both prostate enlargement and symptom severity, emphasizing the need for concurrent glycemic and urological evaluation.

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INTRODUCTION

Background and Anatomy of the Prostate Gland

The prostate gland is a walnut-shaped biological organ positioned at the lower portion of the urinary bladder within the male reproductive system. It produces and releases alkaline fluid into the seminal vesicles, forming an essential component of seminal fluid (1). Anatomically, the gland wraps around the prostatic urethra within the pelvic cavity. The lateral and inferior surfaces rest on the fascia of the levator ani muscle superior to the urogenital diaphragm, with its apex gently pressing against the diaphragm and its base positioned at the bladder neck. The gland comprises three distinct anatomical lobes: two lateral lobes and one median lobe (1).

Histologically and structurally, the prostate is composed of three primary glandular zones: the central zone (CZ), surrounding the ejaculatory ducts; the transition zone (TZ), encompassing the prostatic urethra; and the peripheral zone (PZ), which forms the outer posterior aspect (2). While prostatic carcinoma (PCa) predominantly originates within the PZ, benign prostatic hyperplasia (BPH) develops almost exclusively within the TZ (2). In young males, the TZ constitutes only 5–10% of total glandular volume; however, progressive hyperplastic transformation with aging causes substantial enlargement, making it the dominant zone in older men. The CZ accounts for approximately 25%, while the PZ constitutes 70% of normal glandular tissue. A young adult prostate typically measures 4 cm in width, 3 cm in length, and 2 cm in depth, with an average weight of 20 grams. Though anatomical position remains stable, prostate mass and volume undergo significant age-dependent fluctuations (3).

At the cellular level, the prostate epithelium consists primarily of basal and luminal epithelial cells (4). Basal cells provide structural attachment between the basement membrane and luminal layers and possess somatic stem cell regenerative capabilities. Luminal cells are secretory in nature, releasing prostatic fluids essential for sperm viability and fertility. Luminal cell survival is dependent on continuous androgen receptor (AR) transcriptional activity, whereas basal cells can survive in androgen-deprived environments (4). The functional growth and tissue homeostasis of the prostate are regulated by complex endocrine mechanisms involving circulating steroids, including androgens, estrogens, and glucocorticoids (5). Androgens—primarily dihydrotestosterone (DHT) synthesized locally from testosterone—act as the primary drivers of prostatic cellular proliferation.

Benign Prostatic Hyperplasia (BPH) and Lower Urinary Tract Symptoms (LUTS)

Benign prostatic hyperplasia is defined as non-cancerous proliferation of stromal and epithelial cells within the transition zone of the prostate gland (8, 9). This hyperplastic growth compresses the prostatic urethra, producing bladder outlet obstruction (BOO) and presenting clinically as lower urinary tract symptoms (LUTS) (8). Globally, BPH represents an escalating health burden. Worldwide cases increased from 51.1 million in 2000 to 94.0 million in 2019—a 70.5% surge driven primarily by population expansion and global aging (10, 11). BPH affects nearly 50% of men in their sixties and up to 90% of individuals past the age of 80 (8, 12).

The etiology of BPH is multifactorial, involving intrinsic hormonal shifts (declining serum testosterone, altered estrogen-to-androgen ratios, elevated DHT) and extrinsic factors, including microflora, diet, and chronic tissue inflammation (13, 14). Local insulin-like growth factor-1 (IGF-1), epidermal growth factor, and inflammatory cytokines further stimulate cellular growth (14, 15).

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Pathophysiologically, BOO comprises a static component (physical enlargement of the gland) and a dynamic component (increased smooth muscle tone within the prostatic stroma and bladder neck, mediated by sympathetic nervous activity) (17).

LUTS encompass storage symptoms (increased daytime frequency, urgency, nocturia), voiding symptoms (weak urinary stream, hesitancy, intermittency, straining), and post-micturition symptoms (incomplete emptying) (18, 19). Unmanaged severe LUTS can lead to secondary urological complications, including acute urinary retention, recurrent urinary tract infections (UTIs), and bladder calculus formation (18). Assessment of LUTS severity relies heavily on the International Prostate Symptom Score (IPSS)—an eight-item standardized questionnaire evaluating seven symptom domains (scored 0–5) and one single-item Quality of Life (QoL) measure (scored 1–7). Total IPSS scores are stratified into mild (0–7), moderate (8–19), and severe (20–35) categories (21, 22).

Diabetes Mellitus and Prostatic Health

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by peripheral insulin resistance and progressive pancreatic beta-cell dysfunction (23, 25, 26). T2DM has reached pandemic proportions globally, with IDF estimates predicting over 783 million affected adults by 2045 (27). In Pakistan, national epidemiological surveys demonstrate an alarmingly high prevalence of T2DM, affecting over 26% of the adult population (29, 30).

Diabetes exerts profound physiological effects on the lower urinary tract, giving rise to diabetic bladder dysfunction (DBD) and diabetic cystopathy in 25–90% of patients (27, 33). Mechanistically, chronic hyperglycemia induces vascular atherosclerosis, chronic systemic inflammation, autonomic neuropathy, and hyperinsulinemia (36, 37). Elevated circulating insulin stimulates hepatic production of IGF-1, which acts directly on prostatic stromal and luminal cells to drive cellular proliferation and tissue hypertrophy (16, 38). Concurrently, diabetic autonomic neuropathy impairs detrusor contractility and alters bladder sensation, compounding the mechanical urinary obstruction caused by BPH (33, 35).

The interaction between prostate enlargement, diabetes mellitus, and LUTS severity has been extensively investigated, though published findings demonstrate considerable heterogeneity across diverse geographical and clinical populations.

Ko et al. (2017) demonstrated that anatomical urethral distortion and the ratio of prostatic urethral length to total volume strongly correlated with IPSS voiding scores, emphasizing that mechanical urethral compression significantly influences symptom manifestation independent of total volume alone (43). Sundaram et al. (2017) examined 120 men with LUTS, establishing an age-dependent increase in BPH prevalence (reaching 27% in men aged 61–70 years) and confirming that larger prostate volumes (mean 45 cm^3) were associated with reduced maximum urinary flow rates (Qmax) and increased post-void residual (PVR) urine (44).

Evaluating the specific impact of diabetes, Ozcan et al. (2017) compared 100 diabetic and 200 non-diabetic BPH surgical candidates. Diabetic patients exhibited significantly higher prostate volumes (85.3 mL vs. 59.0 mL, $p < 0.05$) and higher IPSS scores (19.5 vs. 18.4), accompanied by higher BMI and lower serum testosterone, supporting a metabolic-endocrine model of BPH acceleration (45). Conversely, Lim et al. (2018) highlighted that while PV alone showed moderate predictive ability for symptoms, combining volume with age, PSA, and IPSS substantially improved diagnostic precision (AUC 0.82–0.91) (46). In a Congolese

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cohort undergoing TURP, Mubenga et al. (2019) observed significantly higher IPSS in diabetic men, though PV enlargement was restricted primarily to younger diabetic subgroups (<65 years), suggesting complex inter-ethnic and demographic variations (47).

Evaluating multi-parameter clinical models, Hamid et al. (2020) demonstrated significant positive correlations between IPSS and PSA, PV, PVR, and QoL in Sudanese urology patients (48). Meta-analyses by Omran et al. (2021) involving 90,206 participants further confirmed that metabolic syndrome and hyperglycemia are strongly associated with enlarged prostate volume and LUTS severity (49). Similarly, Awaisu et al. (2021) in Nigeria found a statistically significant positive correlation between PV and IPSS ($r = 0.179$, $p = 0.002$) and an inverse correlation with Qmax ($r = -0.176$) (50). Systematic review data compiled by Xin et al. (2022) across 18 studies (1,686 BPH cases, 4,653 controls) conclusively demonstrated that diabetic BPH patients suffer from worse LUTS, larger PV, and higher PSA levels than non-diabetics (51).

However, some literature reports weak or non-significant correlations. Obiesie et al. (2022) found a weak, non-significant correlation between PV and IPSS ($r = 0.109$, $p = 0.28$) in 100 Nigerian men, postulating that detrusor dysfunction plays a major role independent of size (52). In contrast, Udoh et al. (2022, 2024) consistently observed that diabetic men displayed significantly greater PV (79.6 mL vs 52.2 mL) and elevated symptom scores compared to non-diabetics (54, 60). Sadiq et al. (2024) in Pakistan ($r = 0.437$, $p = 0.003$), Ngwa-Ebogo et al. (2023) ($r = 0.410$, $p = 0.006$), and Saeed et al. (2025) ($r = 0.620$, $p < 0.001$) all reported moderate-to-strong positive correlations between PV and IPSS (56, 58, 64). Recent large-scale epidemiological investigations by Zhang et al. (2025) and Yu, Duan, and Xie (2026) in over 8,500 subjects reaffirmed T2DM as an independent major risk factor (22% increased risk) for BPH progression and LUTS severity (65, 67).

Study Rationale and Objectives

Although prostate volume is widely presumed to dictate LUTS severity, conflicting findings in general populations and a relative scarcity of data in diabetic cohorts—particularly within South Asia—highlight a critical knowledge gap. In regions like Peshawar, Pakistan, where dietary patterns, lifestyle factors, T2DM prevalence, and healthcare access differ markedly from Western cohorts, establishing the precise relationship between PV, diabetes duration, and LUTS severity is clinically vital. This study aims to quantify the correlation between prostate volume (measured via TAUS) and LUTS severity (measured via IPSS) in diabetic men with BPH.

MATERIAL AND METHODS

Study Design and Setting

This prospective cross-sectional study was conducted in the Department of Radiology at Rehman Medical Institute (RMI), Peshawar, Pakistan, from Nov 2025 to Feb 2026. Prior to study initiation, formal ethical approval was granted by the Institutional Review Board (IRB) and Ethics Committee of RMI.

Sample Size and Sampling Technique

A total sample size of 138 patients was calculated using the OpenEPI software calculator based on the single proportion formula:

$n = [Z^2 \times P \times (1 - P)] / d^2$

$n = [(1.96)^2 \times 0.90 \times 0.10] / (0.05)^2 = 0.3456 / 0.0025 \approx 138$

Parameters specified included a 95% confidence interval ($Z = 1.96$), a 5% margin of error ($d = 0.05$), and an estimated BPH prevalence rate of 90% ($P = 0.90$) derived from prior literature (Awaisu et al.,

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2021) (50). Non-probability convenience sampling was utilized to recruit eligible participants.

Eligibility Criteria

Inclusion Criteria:

1. Male patients aged 40 to 90 years.
2. Documented diagnosis of Type 2 Diabetes Mellitus.
3. Clinical presentation with LUTS undergoing abdominopelvic ultrasound evaluation for BPH.

Exclusion Criteria:

1. Prior history of prostate or urethral surgery (e.g., TURP, open prostatectomy).
2. Current medical treatment for BPH (alpha-blockers, 5-alpha reductase inhibitors) or ongoing urological pharmacotherapy.
3. Confirmed prostate cancer, neurogenic bladder non-related to diabetes, or active acute urinary tract infection.

Data Collection Procedure

Informed written consent was obtained from all participants. Clinical data were recorded using a structured standardized questionnaire covering demographic details (age categorized as 40–49, 50–59, 60–69, 70–79, and 80–90 years) and diabetes duration (<5 years, 5–10 years, and >10 years).

LUTS severity was quantified using the validated IPSS questionnaire, evaluating seven urinary symptoms (incomplete emptying, frequency, intermittency, urgency, weak stream, straining, and nocturia) on a 0–5 Likert scale (total score 0–35). Symptoms were categorized into mild (0–7), moderate (8–19), and severe (20–35). Quality of life (QoL) was evaluated on a 1–7 single-item scale ranging from 1 ('Delighted') to 7 ('Terrible').

Prostate volume was measured via transabdominal ultrasonography (TAUS) using a curvilinear transducer operating at 3.5 MHz. Maximum anteroposterior (Height), transverse (Width), and longitudinal (Length) dimensions were recorded. Prostate volume (PV in grams/mL) was computed using the standard ellipsoid formula:

Prostate Volume (PV) = Length × Width × Height × 0.52

Prostate volume was further stratified into four clinical grades: Grade I (21–30 g), Grade II (31–50 g), Grade III (51–80 g), and Grade IV (>80 g).

Statistical Analysis

Data analysis was conducted using SPSS software version 22.0. Categorical variables (age brackets, diabetes duration, prostate grade, IPSS severity) were summarized using frequencies and percentages. Continuous variables (PV, IPSS, QoL) were reported as mean ± standard deviation (SD). Pearson's Chi-Square test was performed to test the association between diabetes duration and LUTS severity categories. One-Way ANOVA was utilized to assess differences in mean prostate volume across diabetes duration groups. Pearson's correlation coefficient (r) was computed to determine the direction and strength of correlation between PV and IPSS. Statistical significance was defined as $p < 0.05$.

RESULTS

The study evaluated 138 male diabetic BPH patients. The mean age was 2.69 ± 1.15 (coded category scale), with the highest proportion of patients belonging to the 60–69 years age bracket (n = 39, 28.3%), followed by 70–79 years (n = 34, 24.6%) and 50–59 years (n = 33, 23.9%) (Table 1).

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Table 1: *Age Distribution of Patients (N = 138)*

Age Group (Years)	Frequency (n)	Percent (%)	Valid Percent (%)	Cumulative Percent (%)
40–49	27	19.6	19.6	19.6
50–59	33	23.9	23.9	43.5
60–69	39	28.3	28.3	71.7
70–79	34	24.6	24.6	96.4
80–90	5	3.6	3.6	100.0

Table 2: *Descriptive Statistics for Prostate Volume and IPSS*

Variable	N	Range	Minimum	Maximum	Mean	Std. Deviation
Prostate Volume (g)	138	100.0	30.0	130.0	41.78	17.14
Total IPSS Score	138	26.0	3.0	29.0	12.56	6.10

The mean prostate volume was 41.78 ± 17.14 g (range: 30.0–130.0 g). The mean total IPSS score was 12.56 ± 6.10 (range: 3.0–29.0) (Table 2). Most patients were categorized into Grade II BPH (31–50 g; n = 100, 72.5%), followed by Grade I (21–30 g; n = 19, 13.8%), Grade III (51–80 g; n = 14, 10.1%), and Grade IV (>80 g; n = 5, 3.6%) (Table 3).

Table 3: *Classification by Grade of Prostate Gland Volume*

Prostate Grade (Volume)	Frequency (n)	Percent (%)	Valid Percent (%)	Cumulative Percent (%)
Grade I (21–30 g)	19	13.8	13.8	13.8
Grade II (31–50 g)	100	72.5	72.5	86.2
Grade III (51–80 g)	14	10.1	10.1	96.4
Grade IV (>80 g)	5	3.6	3.6	100.0

Regarding LUTS severity, 73 patients (52.9%) presented with moderate symptoms, 42 patients (30.4%) with mild symptoms, and 23 patients (16.7%) with severe symptoms (Table 4). Evaluation of individual IPSS domains revealed nocturia, urgency, and incomplete emptying as the most prevalent and bothersome symptoms.

Table 4: *Severity Categories of LUTS Based on IPSS*

Symptom Severity	Frequency (n)	Percent (%)	Valid Percent (%)	Cumulative Percent (%)
Mild (0–7)	42	30.4	30.4	30.4
Moderate (8–19)	73	52.9	52.9	83.3
Severe (20–35)	23	16.7	16.7	100.0

Duration of diabetes mellitus was <5 years in 59 patients (42.8%), 5–10 years in 32 patients (23.2%), and >10 years in 47 patients (34.1%) (Table 5). The overall mean Quality of Life (QoL) score was 4.86 ± 1.48, with 34.8% (n = 48) reporting feeling 'mostly unhappy' and 21.7% (n = 30) 'unhappy' with their urinary condition (Table 6).

Table 5: *Duration of Diabetes Mellitus Among Participants*

Diabetes Duration	Frequency (n)	Percent (%)	Valid Percent (%)	Cumulative Percent (%)
< 5 Years	59	42.8	42.8	42.8
5–10 Years	32	23.2	23.2	65.9
> 10 Years	47	34.1	34.1	100.0

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Table 6: ***Quality of Life (QoL) Assessment Related to Urinary Symptoms***

QoL Perception Score	Frequency (n)	Percent (%)
Pleased / Satisfied	8	5.8
Mixed	5	3.6
Mostly Dissatisfied	28	20.3
Unhappy	30	21.7
Mostly Unhappy	48	34.8
Terrible	19	13.8

Cross-tabulation and Pearson's Chi-Square test demonstrated a statistically significant association between diabetes duration and LUTS severity ($p < 0.01$) (Table 7). Among patients with diabetes for >10 years, 38.3% (18/47) experienced severe LUTS, whereas only 3.4% (2/59) of those with <5 years of diabetes had severe symptoms.

Table 7: ***Cross-Tabulation of Diabetes Duration and LUTS Severity Categories***

Diabetes Duration	Mild (0–7)	Moderate (8–19)	Severe (20–35)	Total (n)
< 5 Years	30	27	2	59
5–10 Years	10	19	3	32
> 10 Years	2	27	18	47
Total	42	73	23	138

One-Way ANOVA revealed a statistically significant increase in mean prostate volume with increasing duration of diabetes ($p < 0.01$) (Table 8). Mean PV was 37.4 g in patients with diabetes <5 years, 38.1 g in those with 5–10 years, and rose markedly to 49.7 g in patients with diabetes >10 years.

Table 8: ***Mean Prostate Volume According to Diabetes Duration***

Diabetes Duration	Mean Volume (g)	Std. Deviation	95% CI (Lower)	95% CI (Upper)
< 5 Years	37.4	12.8	33.0	41.0
5–10 Years	38.1	14.2	32.0	43.0
> 10 Years	49.7	21.5	45.0	54.0

Pearson's correlation analysis demonstrated a statistically significant moderate positive correlation between prostate volume and total IPSS score ($r = 0.513$, $p < 0.01$) (Table 9). This indicates that as prostate volume increases, LUTS severity increases proportionally in diabetic men with BPH.

Table 9: ***Pearson Correlation Between Prostate Volume and Total IPSS Score***

Variables Evaluated	Pearson Correlation (r)	p-value (2-tailed)
Prostate Volume vs Total IPSS	0.513	< 0.01**

* Statistically significant at $p < 0.01$ level.

DISCUSSION

This prospective study evaluated the correlation between prostate volume (PV) and LUTS severity among 138 diabetic men with BPH in Peshawar, Pakistan. Our primary finding is a statistically significant moderate positive correlation between PV and IPSS ($r = 0.513$, $p < 0.01$). Furthermore, prolonged diabetes duration was strongly associated with both larger prostate volume ($p < 0.01$) and heightened symptom severity ($p < 0.01$). The age distribution in our study, peaking in the 60–69 years age bracket (28.3%), aligns closely with

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findings by Awaisu et al. in Nigeria (47.6% in 60–69 years) (50) and Sundaram et al. in India (51% in 61–70 years) (44). This confirms that symptomatic BPH coupled with T2DM is predominantly a disease of the seventh decade of life, driven by cumulative hormonal shifts and metabolic stress.

The mean prostate volume observed in our cohort (41.78 ± 17.14 g) is consistent with the mean volume of 45 cm^3 reported by Sundaram et al. (44), but lower than the 52.6 mL reported by Awaisu et al. (50) and substantially higher than the 29.4 mL reported by Lim et al. in Malaysia (46). Grade II BPH (31–50 g) was the dominant category (72.5%) in our sample, whereas Sadiq et al. in Rawalpindi observed a higher proportion of Grade III cases (44.4%) (58). These variations in volume across cohorts likely reflect regional differences in presentation timing, genetic factors, and healthcare access.

Our mean IPSS score (12.56 ± 6.10) reflects a predominantly moderate symptom severity (52.9%), closely mirroring values reported by Udo et al. in Nigeria (13.2 ± 4.6) (59). Higher mean IPSS scores reported by Ozcan et al. (18.8) (45) and Sadiq et al. (20.5) (58) may stem from tertiary surgical recruitment where patients present at more advanced stages. Quality of life evaluation showed that over 56% of participants felt 'unhappy' or 'mostly unhappy' with their urological state (mean QoL = 4.86), confirming that LUTS imposes a profound psychosocial burden, matching observations by Obiesie et al. (QoL = 4.8) (52).

A key finding of our study is the highly significant association between diabetes duration and LUTS severity ($p < 0.01$). Patients with diabetes for >10 years exhibited a 38.3% rate of severe LUTS compared to only 3.4% in those with <5 years of diabetes. This supports the meta-analytic findings of Xin et al. (51) and case evidence by Opara (53), which demonstrate that prolonged hyperglycemia accelerates diabetic cystopathy and autonomic nerve damage, impairing bladder detrusor contractility and exaggerating voiding/storage symptoms.

Similarly, diabetes duration was significantly correlated with increased prostate volume (mean PV 49.7 g for >10 years vs. 37.4 g for <5 years, $p < 0.01$). This direct relationship is mechanistically supported by Udoh et al. (54, 60) and Wei et al. (36), who established that persistent hyperinsulinemia and upregulation of IGF-1 signal transduction stimulate prostatic stromal and epithelial cell mitosis, while suppressing physiological cell apoptosis overtime.

Regarding the core correlation between PV and IPSS, our coefficient ($r = 0.513$) aligns with findings from Saeed et al. ($r = 0.620$) (64), Sadiq et al. ($r = 0.437$) (58), and Ngwa-Ebogo et al. ($r = 0.410$) (56). While some studies (Apata et al., Parekh et al.) reported weak or non-significant correlations (57, 66), our robust correlation in a strictly diabetic cohort suggests that hyperinsulinemia-driven prostatic growth adds a substantial mechanical obstructive component onto an already compromised diabetic bladder. Therefore, both mechanical prostatic enlargement and neuropathic bladder dysfunction synergize to exacerbate LUTS in diabetic men.

CONCLUSION

Our study demonstrates a statistically significant moderate positive correlation between prostate volume and LUTS severity ($r = 0.513$, $p < 0.01$) in diabetic men with BPH. Furthermore, longer duration of diabetes (>10 years) is significantly associated with greater prostate enlargement and heightened symptom severity. These findings indicate that while diabetes independently impairs bladder nerve and muscle function, mechanical prostatic enlargement remains a primary contributor to LUTS severity. Clinical

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management of diabetic men presenting with lower urinary tract symptoms must integrate both rigorous glycemic control and precise sonographic prostate volume assessment.

RECOMMENDATIONS

1. Routine Screening: Transabdominal ultrasound (TAUS) estimation of prostate volume and standard IPSS questionnaire scoring should be routinely incorporated into the management protocols of diabetic men presenting with urinary symptoms, particularly those with a diabetes duration exceeding 5 years.
2. Dual-Targeted Clinical Management: Clinicians should avoid attributing LUTS in diabetic patients solely to BPH or diabetic cystopathy in isolation. Combined therapeutic strategies addressing both metabolic/glycemic control and prostatic outlet obstruction are recommended.
3. Multi-Center Longitudinal Research: Multi-center longitudinal studies incorporating objective glycemic parameters (HbA1c) and urodynamic measurements are recommended to further clarify the temporal progression of BPH and diabetic bladder dysfunction.

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