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*Original Article*

# Correlation of Doppler Ultrasound Resistive Index in Benign Prostatic Hyperplasia with Prostate Volume, Urinary Tract Obstruction, and Urinary Bladder Wall Thickness in Diabetic and Non-Diabetic Patients

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## ABSTRACT

Background: Benign prostatic hyperplasia is a common cause of lower urinary tract symptoms in older men, and diabetes mellitus may worsen urinary dysfunction through metabolic, microvascular, and autonomic mechanisms. Doppler ultrasound resistive index may provide additional information regarding prostatic vascular resistance beyond conventional sonographic measurements. Objective: To compare Doppler ultrasound resistive index, prostate volume, bladder wall thickness, and urine volume parameters between diabetic and non-diabetic patients with benign prostatic hyperplasia. Methods: This cross-sectional observational study included 100 male patients with benign prostatic hyperplasia, comprising 53 diabetic and 47 non-diabetic patients. Transabdominal ultrasound was used to measure prostate volume, bladder wall thickness, pre-void urine volume, and post-void residual urine volume, while Doppler ultrasound was used to assess prostatic arterial resistive index. Group comparisons were performed using independent samples t-tests, with statistical significance set at $p \leq 0.05$. Results: Diabetic patients had higher prostate volume than non-diabetic patients (46.24 ± 8.24 ml vs. 37.06 ± 7.75 ml; $p < 0.001$), greater bladder wall thickness (6.39 ± 0.95 mm vs. 4.80 ± 0.88 mm; $p < 0.001$), higher pre-void urine volume (337.44 ± 74.04 ml vs. 311.17 ± 50.99 ml; $p = 0.044$), higher post-void residual urine volume (98.21 ± 34.79 ml vs. 59.96 ± 23.18 ml; $p < 0.001$), and higher prostatic arterial resistive index (0.751 ± 0.048 vs. 0.687 ± 0.050; $p < 0.001$). Age did not differ significantly between groups. Conclusion: Diabetic patients with benign prostatic hyperplasia demonstrated greater prostate enlargement, bladder wall thickening, residual urine burden, and prostatic vascular resistance than non-diabetic patients. Doppler resistive index may serve as an adjunctive non-invasive parameter in the sonographic evaluation of benign prostatic hyperplasia, particularly among diabetic patients. Keywords: Benign Prostatic Hyperplasia; Resistive Index; Doppler Ultrasound; Prostate Volume; Bladder Wall Thickness; Diabetes Mellitus; Lower Urinary Tract Symptoms.

## INTRODUCTION

Benign prostatic hyperplasia is a common non-malignant enlargement of the prostate gland and represents one of the leading causes of lower urinary tract symptoms among ageing men. As the prostate enlarges around the prostatic urethra and bladder outlet, patients may develop voiding and storage symptoms, including weak urinary stream, hesitancy, intermittency, incomplete bladder emptying, frequency, urgency, nocturia, and increased post-void residual urine volume. Although prostate size is an important structural marker, the clinical severity of benign prostatic hyperplasia is not determined

by prostate volume alone, because bladder outlet obstruction also depends on dynamic smooth-muscle tone, intraprostatic vascular changes, bladder compensation, detrusor function, and patient-related metabolic factors. Therefore, assessment of benign prostatic hyperplasia requires an integrated approach that includes prostate volume, bladder wall thickness, urine volume parameters, symptom severity, and where feasible, vascular indices derived from Doppler ultrasound (1).

Ultrasonography is widely used as a non-invasive modality for evaluating prostate enlargement and its downstream effects on the urinary bladder. Prostate volume is commonly estimated using the ellipsoid formula by multiplying length, width, and height by 0.52, while bladder wall thickness and post-void residual urine volume provide additional information regarding bladder outlet resistance and detrusor compensation. A prostate volume greater than approximately 30 $cm^3$ is generally considered enlarged in clinical imaging practice, while increased bladder wall thickness and elevated post-void residual urine may indicate chronic outlet obstruction and impaired bladder emptying. However, structural measurements alone may not fully reflect the hemodynamic changes that occur within the enlarged prostate. Doppler ultrasound provides additional vascular information through the resistive index, which reflects downstream vascular resistance in the sampled prostatic arterial flow. A higher resistive index may indicate increased intraprostatic vascular resistance and has been associated with more severe benign prostatic enlargement and obstruction-related changes (2).

Diabetes mellitus may further complicate the clinical and sonographic profile of benign prostatic hyperplasia. Several biological mechanisms may explain this interaction, including insulin resistance, hyperinsulinemia, chronic low-grade inflammation, altered androgen metabolism, endothelial dysfunction, autonomic neuropathy, and microvascular impairment. These mechanisms may contribute to increased prostatic growth, altered prostatic perfusion, impaired detrusor contractility, and greater post-void residual urine volume. In diabetic men, lower urinary tract symptoms may therefore result from both mechanical bladder outlet obstruction and diabetes-related bladder dysfunction. This dual mechanism is clinically important because diabetic patients with benign prostatic hyperplasia may present with greater prostate volume, increased bladder wall thickness, higher residual urine volume, and altered Doppler vascular indices compared with non-diabetic patients (3).

Previous studies have evaluated the relationship of prostate volume, post-void residual urine, bladder wall thickness, and Doppler-derived vascular indices with the severity of benign prostatic hyperplasia and bladder outlet obstruction. Evidence suggests that prostatic resistive index may increase with prostate enlargement and may decline after surgical decompression, supporting its potential role as a non-invasive adjunctive marker of obstruction-related vascular resistance. Studies have also suggested that bladder wall thickness and post-void residual urine volume can serve as indirect markers of chronic outlet obstruction, particularly when interpreted alongside prostate size and clinical symptoms. However, fewer studies have specifically examined whether these sonographic and Doppler parameters differ between diabetic and non-diabetic men with benign prostatic hyperplasia, especially in local clinical settings where diabetes is common and may influence both vascular resistance and bladder function (4).

The present study was therefore designed to assess Doppler ultrasound resistive index in relation to prostate volume, bladder wall thickness, and urinary volume parameters among men with benign prostatic hyperplasia, with comparison between diabetic and non-diabetic patients. The study addresses the clinical question of whether diabetic status is associated with higher prostatic resistive index and more advanced sonographic features of bladder outlet obstruction in patients with benign prostatic hyperplasia. It was hypothesized that diabetic patients with benign prostatic hyperplasia would demonstrate higher resistive index values, larger prostate volume, greater bladder wall thickness, and increased post-void residual urine volume compared with non-diabetic patients.

## MATERIALS AND METHODS

This study was conducted as a cross-sectional observational study to evaluate Doppler ultrasound resistive index and related sonographic parameters in male patients diagnosed with benign prostatic hyperplasia. The study was carried out at Omar Hospital, Lahore, over a period of 90 days after approval of the synopsis. The design was selected because the objective was to compare sonographic and Doppler parameters between diabetic and non-diabetic patients at a single point of assessment and to examine the relationship of resistive index with prostate volume, bladder wall thickness, and urinary volume parameters.

Male patients with clinically diagnosed benign prostatic hyperplasia and lower urinary tract symptoms were selected for inclusion. Eligible participants were men in the older adult age range who were willing to undergo ultrasound and Doppler evaluation. Both diabetic and non-diabetic patients were included to allow comparative assessment according to diabetic status. Patients were excluded if they had a history of prostate surgery, known or suspected prostate cancer, urinary tract infection, bladder stones, neurogenic bladder, uncontrolled diabetes, or uncontrolled hypertension, because these conditions could independently alter urinary bladder function, prostate morphology, post-void residual urine volume, or Doppler vascular measurements. The final analysis was conducted on 100 male participants, including 53 diabetic and 47 non-diabetic patients.

Participants underwent transabdominal ultrasound assessment for prostate volume, urinary bladder wall thickness, pre-void urine volume, post-void residual urine volume, and Doppler resistive index. Prostate volume was calculated using the standard ellipsoid formula by multiplying the maximum length, width, and height of the prostate by 0.52. Bladder wall thickness was measured sonographically as an indicator of bladder response to chronic outlet resistance. Pre-void urine volume was recorded before micturition, and post-void residual urine volume was measured after voiding to assess the degree of incomplete bladder emptying. Doppler ultrasound was used to assess prostatic arterial flow, and resistive index was calculated from the Doppler waveform using the formula: peak systolic velocity minus end-diastolic velocity divided by peak systolic velocity. Higher resistive index values were interpreted as reflecting increased vascular resistance within the prostatic arterial bed.

The primary exposure variable was diabetic status, categorized as diabetic or non-diabetic. The principal Doppler outcome was prostatic arterial resistive index. Additional sonographic variables included prostate volume in milliliters, urinary bladder wall thickness in millimeters, pre-void urine volume in milliliters, and post-void residual urine volume in milliliters. Age was recorded in years and used to describe the study population and compare baseline age distribution between diabetic and non-diabetic groups. The main analytical comparison was the difference in prostate volume, bladder wall thickness, urine volume parameters, and resistive index between diabetic and non-diabetic patients. The main association of interest was the relationship between resistive index and prostate volume, with additional clinical interpretation of bladder wall thickness and post-void residual urine as obstruction-related parameters.

The sample size was calculated using the single population proportion formula, with a 95% confidence level, expected prevalence estimate, and 10% margin of error. Although the minimum calculated sample size was 93, the final analyzed sample consisted of 100 participants to strengthen precision and account for complete available observations. Data were checked for completeness before analysis. Continuous variables were summarized using mean, standard deviation, minimum, and maximum values. Group-wise comparisons between diabetic and non-diabetic patients were performed using independent samples t-tests for continuous variables. Levene's test was used to assess equality of variances before interpreting t-test results. Statistical significance was assessed at a p-value threshold of $\leq 0.05$, and p-values less than 0.001 were reported as $p < 0.001$ rather than $p = 0.000$. Correlation analysis was planned to evaluate the relationship between resistive index and prostate volume, as well as other sonographic

obstruction-related parameters where complete data were available. The analysis focused on non-causal interpretation because the cross-sectional design permits assessment of association but not temporal or causal inference.

To reduce measurement and selection bias, the study applied predefined inclusion and exclusion criteria and used standardized ultrasound-based measurements for all participants. Potential confounding was addressed descriptively by comparing age between diabetic and non-diabetic groups, while interpretation of findings considered the possible influence of unmeasured factors such as body mass index, glycemic control, hypertension status, medication use, prostate-specific antigen level, testosterone level, and duration of diabetes. Data were entered and reviewed for consistency before statistical analysis. Participant willingness to undergo ultrasound assessment was required before inclusion, and the study was conducted after institutional synopsis approval.

## RESULTS

A total of 100 male patients with benign prostatic hyperplasia were included in the final analysis. Of these, 53 patients were diabetic and 47 were non-diabetic. The study population had a mean age of 62.98 ± 11.04 years, with an age range of 45 to 82 years. The overall mean prostate volume was 41.93 ± 9.21 ml, mean bladder wall thickness was 5.65 ± 1.21 mm, mean pre-void urine volume was 325.09 ± 65.28 ml, mean post-void residual urine volume was 80.24 ± 35.41 ml, and mean prostatic arterial resistive index was 0.72 ± 0.06.

*Table 1. Descriptive Sonographic and Clinical Characteristics of the Study Participants*

| Variable | N | Minimum | Maximum | Mean ± SD |
|---|---|---|---|---|
| **Age (years)** | 100 | 45.00 | 82.00 | 62.98 ± 11.04 |
| **Prostate volume (ml)** | 100 | 20.60 | 68.60 | 41.93 ± 9.21 |
| **Bladder wall thickness (mm)** | 100 | 3.00 | 8.10 | 5.65 ± 1.21 |
| **Pre-void urine volume (ml)** | 100 | 133.10 | 492.80 | 325.09 ± 65.28 |
| **Post-void residual urine volume (ml)** | 100 | 14.70 | 168.20 | 80.24 ± 35.41 |
| **Prostatic arterial resistive index** | 100 | 0.60 | 0.87 | 0.72 ± 0.06 |

SD, standard deviation.

The descriptive findings show that the study population consisted predominantly of older adult men with sonographic evidence of prostate enlargement and bladder outlet-related urinary changes. The mean prostate volume exceeded the commonly used enlargement threshold of approximately 30 ml, while the mean bladder wall thickness was above 5 mm, suggesting structural bladder adaptation in this BPH cohort. The mean post-void residual urine volume of 80.24 ml indicated incomplete bladder emptying in a substantial proportion of patients, and the mean resistive index of 0.72 suggested increased prostatic vascular resistance at the group level.

Among the 100 participants, diabetic patients represented 53.0% of the sample, while non-diabetic patients represented 47.0%. This distribution allowed comparative assessment of sonographic and Doppler parameters between diabetic and non-diabetic men with benign prostatic hyperplasia.

*Table 2. Distribution of Participants by Diabetic Status*

| Diabetic status | n | % |
|---|---|---|
| **Diabetic** | 53 | 53.0 |
| **Non-diabetic** | 47 | 47.0 |
| **Total** | 100 | 100.0 |

The diabetic and non-diabetic groups were relatively balanced, with a slightly higher proportion of diabetic patients. This distribution supports group-wise comparison of prostate volume, bladder wall thickness, urine volume parameters, and prostatic arterial resistive index.

Independent samples t-tests were used to compare continuous variables between diabetic and non-diabetic patients. Diabetic patients had a higher mean prostate volume than non-diabetic patients, with

a mean difference of 9.18 ml. Bladder wall thickness was also higher in diabetic patients, with a mean difference of 1.59 mm. Pre-void urine volume, post-void residual urine volume, and resistive index were all higher in diabetic patients. Age did not differ significantly between the groups.

*Table 3. Comparison of Sonographic and Clinical Variables Between Diabetic and Non-Diabetic Patients*

| Variable | Diabetic n | Diabetic Mean ± SD | Non-Diabetic n | Non-Diabetic Mean ± SD | Mean Difference | 95% CI | t | p-value | Cohen's d |
|---|---|---|---|---|---|---|---|---|---|
| **Prostate volume (ml)** | 53 | 46.24 ± 8.24 | 47 | 37.06 ± 7.75 | 9.18 | 6.01 to 12.36 | 5.720 | <0.001 | 1.15 |
| **Bladder wall thickness (mm)** | 53 | 6.39 ± 0.95 | 47 | 4.80 ± 0.88 | 1.59 | 1.23 to 1.95 | 8.647 | <0.001 | 1.73 |
| **Pre-void urine volume (ml)** | 53 | 337.44 ± 74.04 | 47 | 311.17 ± 50.99 | 26.27 | 1.25 to 51.29 | 2.040 | 0.044 | 0.41 |
| **Post-void residual urine volume (ml)** | 53 | 98.21 ± 34.79 | 47 | 59.96 ± 23.18 | 38.25 | 26.62 to 49.88 | 6.383 | <0.001 | 1.28 |
| **Prostatic arterial resistive index** | 53 | 0.751 ± 0.048 | 47 | 0.687 ± 0.050 | 0.064 | 0.044 to 0.083 | 6.478 | <0.001 | 1.30 |
| **Age (years)** | 53 | 61.36 ± 11.36 | 47 | 64.81 ± 10.48 | -3.45 | -7.79 to 0.89 | -1.571 | 0.119 | -0.32 |

CI, confidence interval; SD, standard deviation. Mean difference was calculated as diabetic minus non-diabetic. Independent samples t-test was used for group comparison.

The comparison between diabetic and non-diabetic patients demonstrated a consistent pattern of more advanced sonographic and Doppler abnormalities among diabetic patients. Prostate volume was substantially higher in diabetic patients, with a large effect size. Bladder wall thickness showed the largest standardized difference between groups, indicating greater bladder wall hypertrophy among diabetic patients. Post-void residual urine volume was also markedly higher in diabetic patients, with a large effect size, suggesting greater incomplete bladder emptying. The prostatic arterial resistive index was higher in diabetic patients by 0.064 units, with a large standardized effect, supporting the presence of increased prostatic vascular resistance in this group. Pre-void urine volume showed a smaller but statistically significant difference, while age showed no statistically significant group difference, indicating that the observed sonographic differences were unlikely to be explained by a major age imbalance between groups.

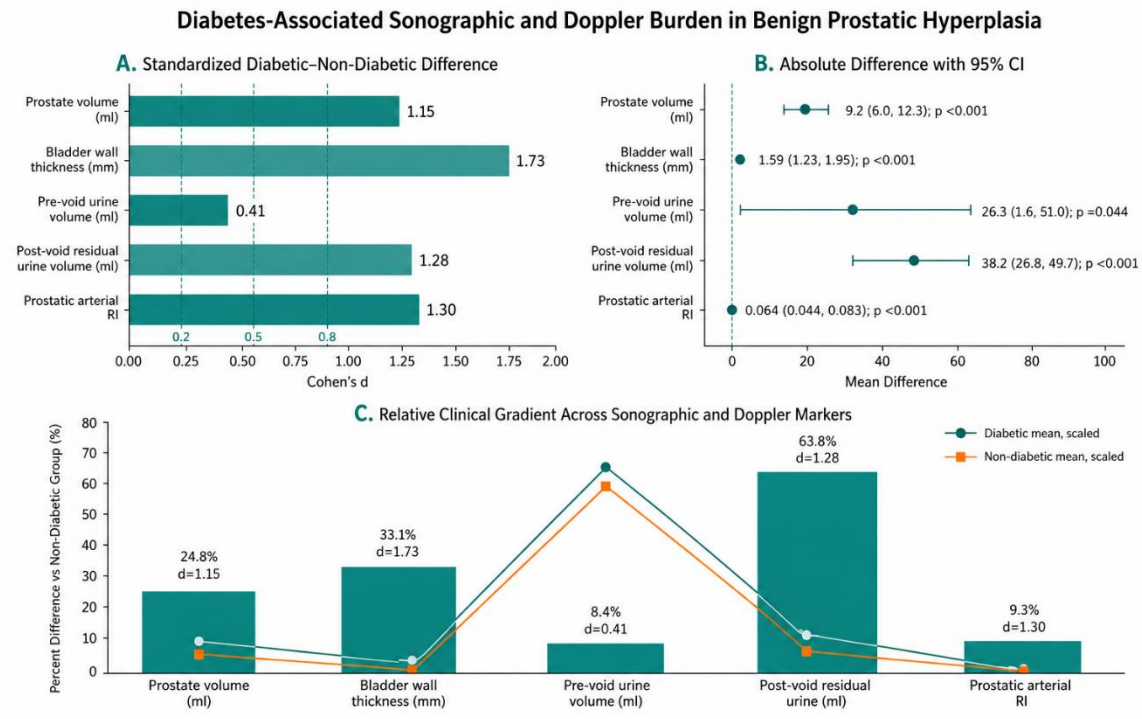


*Figure 1 Diabetes-Associated Sonographic and Doppler Burden in Benign Prostatic Hyperplasia*

The panelled figure demonstrates a consistent diabetes-associated increase across structural, urinary, and Doppler parameters in men with benign prostatic hyperplasia. The largest standardized between-

group difference was observed for bladder wall thickness, with diabetic patients showing a 1.59 mm higher mean value than non-diabetic patients and a Cohen's d of 1.73. Post-void residual urine volume showed the greatest relative difference, being 38.25 ml higher in diabetic patients, corresponding to a 63.8% increase compared with the non-diabetic group and a large effect size of 1.28. Prostatic arterial resistive index was also higher in diabetic patients by 0.064 units, with a large effect size of 1.30, supporting increased prostatic vascular resistance in this subgroup. Prostate volume was 9.18 ml higher in diabetic patients, while pre-void urine volume showed a smaller difference of 26.27 ml. Overall, the figure supports a clinically meaningful pattern in which diabetic patients demonstrate greater prostate enlargement, bladder wall hypertrophy, residual urine burden, and Doppler vascular resistance.

The manuscript objective included assessment of the correlation between prostatic arterial resistive index and prostate volume, bladder wall thickness, and urinary obstruction-related parameters. However, the available manuscript data did not provide Pearson or Spearman correlation coefficients, regression coefficients, obstruction-grade frequencies, or individual-level paired observations. Therefore, correlation coefficients and regression estimates cannot be validly reported from the available aggregated results. For publication completeness, a correlation analysis should be added using the original dataset, preferably reporting the correlation of resistive index with prostate volume, bladder wall thickness, pre-void urine volume, and post-void residual urine volume, along with 95% confidence intervals and p-values.

## DISCUSSION

This cross-sectional observational study evaluated Doppler ultrasound resistive index and related sonographic parameters in diabetic and non-diabetic men with benign prostatic hyperplasia. The main finding was that diabetic patients demonstrated consistently higher values for prostate volume, bladder wall thickness, pre-void urine volume, post-void residual urine volume, and prostatic arterial resistive index compared with non-diabetic patients. These differences were observed despite the absence of a statistically significant age difference between the two groups, suggesting that the sonographic and Doppler differences were not primarily explained by age imbalance in the available data. The findings support the clinical interpretation that diabetic patients with benign prostatic hyperplasia may show a greater combined burden of prostate enlargement, bladder wall adaptation, incomplete bladder emptying, and increased prostatic vascular resistance.

The mean prostate volume was higher in diabetic patients than in non-diabetic patients, with a mean difference of 9.18 ml and a large standardized effect size. This finding is biologically plausible because diabetes mellitus is associated with insulin resistance, hyperinsulinemia, chronic low-grade inflammation, endothelial dysfunction, and altered sex hormone regulation, all of which may contribute to prostatic stromal and epithelial proliferation. Diabetes may also interact with obesity and metabolic syndrome, which have been implicated in benign prostatic enlargement and lower urinary tract symptoms. Although prostate enlargement alone does not fully determine the severity of bladder outlet obstruction, a larger prostate volume may increase the probability of urethral compression, transition-zone enlargement, and clinically relevant voiding dysfunction when combined with dynamic and metabolic factors (3,4).

Bladder wall thickness was also substantially higher in diabetic patients, showing the largest standardized between-group difference among the evaluated parameters. Increased bladder wall thickness is commonly interpreted as a structural response to chronic outlet resistance, reflecting detrusor hypertrophy caused by sustained effort to overcome increased bladder outlet resistance. In diabetic patients, this process may be further influenced by diabetic cystopathy, autonomic neuropathy, altered bladder sensation, and impaired detrusor contractility. Therefore, the greater bladder wall thickness observed in diabetic patients may reflect a combined effect of benign prostatic obstruction and diabetes-related bladder dysfunction rather than mechanical obstruction alone. This interpretation is

consistent with previous work suggesting that bladder wall and detrusor thickness may serve as non-invasive markers of obstruction-related bladder remodeling in men with benign prostatic enlargement (5).

Post-void residual urine volume was markedly higher in diabetic patients, with a mean difference of 38.25 ml compared with non-diabetic patients. This is clinically important because increased post-void residual urine reflects incomplete bladder emptying and may be associated with urinary retention, recurrent urinary tract infection, worsening lower urinary tract symptoms, and reduced quality of life. In men with benign prostatic hyperplasia, elevated residual urine may result from bladder outlet obstruction, detrusor underactivity, or both. Diabetes can contribute to detrusor underactivity through autonomic neuropathy and impaired bladder contractile function, which may explain why diabetic patients in this study demonstrated higher residual urine volume. The smaller but statistically significant difference in pre-void urine volume should be interpreted more cautiously, as pre-void volume is influenced by hydration status, timing of voiding, bladder sensation, and patient preparation before ultrasound.

The prostatic arterial resistive index was higher in diabetic patients than in non-diabetic patients, with a large standardized effect size. Doppler resistive index reflects downstream resistance to arterial flow and may increase in benign prostatic hyperplasia because of stromal enlargement, tissue pressure, vascular compression, and altered intraprostatic hemodynamics. In diabetic patients, microvascular disease, endothelial dysfunction, and arterial stiffness may further increase vascular resistance. The observed increase in resistive index among diabetic patients therefore supports the concept that Doppler ultrasound may provide additional functional vascular information beyond conventional structural measurements such as prostate volume and bladder wall thickness. Previous studies have reported that prostatic resistive index may correlate with prostate enlargement and obstruction-related parameters, supporting its potential role as an adjunctive imaging marker in men with benign prostatic hyperplasia (4,6,7).

Although the study objective included correlation of Doppler resistive index with prostate volume, urinary obstruction, and bladder wall thickness, the available results do not provide correlation coefficients, regression estimates, or individual-level paired data. Therefore, the present interpretation should be limited to group-wise differences between diabetic and non-diabetic patients. The higher mean resistive index observed alongside higher prostate volume, bladder wall thickness, and post-void residual urine in diabetic patients suggests a consistent pattern of increased sonographic and Doppler burden, but it does not prove a direct linear correlation between resistive index and each obstruction-related parameter. For a stronger analytical conclusion, the final manuscript should include Pearson or Spearman correlation analysis using the original dataset, ideally followed by multivariable regression adjusting for age, diabetes status, prostate volume, bladder wall thickness, and other clinically relevant covariates.

The findings have practical implications for ultrasound-based assessment of benign prostatic hyperplasia, particularly in diabetic patients. Conventional sonographic assessment often focuses on prostate volume and post-void residual urine; however, the present findings suggest that bladder wall thickness and Doppler resistive index may add clinically useful information regarding obstruction-related remodeling and vascular resistance. In diabetic patients, this expanded sonographic approach may be especially relevant because urinary symptoms may arise from both bladder outlet obstruction and diabetes-related bladder dysfunction. Doppler resistive index should not be interpreted as a standalone diagnostic test, but it may serve as an adjunctive parameter when integrated with prostate volume, residual urine volume, bladder wall thickness, symptom scoring, biochemical markers, and clinical evaluation.

This study has several limitations. The cross-sectional design prevents conclusions about causality, temporal progression, or whether diabetes directly worsens benign prostatic hyperplasia. The study was

conducted at a single center, which may limit generalizability to broader populations. The manuscript does not report important potential confounders such as body mass index, duration of diabetes, glycemic control, HbA1c, hypertension status, medication use, prostate-specific antigen level, testosterone level, or symptom severity score. Doppler ultrasound measurements may also be operator-dependent, and inter-observer or intra-observer reliability was not reported. In addition, the central correlation objective requires additional statistical reporting because correlation coefficients and regression models are not currently presented. Future studies should use larger multicenter samples, standardized Doppler protocols, biochemical and hormonal profiling, validated lower urinary tract symptom scores, and longitudinal follow-up to determine whether resistive index predicts disease progression or response to treatment.

## CONCLUSION

Doppler ultrasound resistive index was higher in diabetic patients with benign prostatic hyperplasia than in non-diabetic patients and was accompanied by greater prostate volume, increased bladder wall thickness, higher pre-void urine volume, and markedly higher post-void residual urine volume. These findings suggest that diabetic patients may have a greater sonographic and vascular burden of benign prostatic hyperplasia, potentially reflecting the combined effects of prostate enlargement, bladder outlet resistance, impaired bladder emptying, and diabetes-related microvascular or autonomic dysfunction. Prostatic arterial resistive index may therefore be considered an adjunctive non-invasive Doppler parameter in the evaluation of benign prostatic hyperplasia, particularly among diabetic patients, but it should be interpreted alongside conventional ultrasound findings, clinical symptoms, and relevant biochemical markers. Further correlation and multivariable analyses using individual-level data are required before resistive index can be recommended as an independent marker of obstruction severity or disease progression.

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