ORIGINAL PAPER

FUNCTIONAL UROLOGY

Correlation of Doppler ultrasound resistive index in the prostatic gland with severity of male lower urinary tract symptoms, prostate volume, and concomitant diabetes mellitus

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Citation: Okedere TA, Asaleye CM, Ayoola OO, et al. Correlation of Doppler ultrasound resistive index in the prostatic gland with severity of male lower urinary tract symptoms, prostate volume, and concomitant diabetes mellitus. Cent European J Urol. 2023; 76: 199-206.

Article history
Submitted: March 18, 2023
Accepted: June 7, 2023
Published online: July 13, 2023

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Introduction Benign prostatic enlargement (BPE) and type 2 diabetes mellitus (T2DM) are common in elderly men. This study aimed to correlate the Doppler resistive indices of prostatic arteries with the severity of lower urinary tract symptoms (LUTS) and prostate volume in men with concomitant BPE and T2DM.

Material and methods Fifty men with T2DM and BPE (BPE-DM) as cases and 50 age-matched men with BPE but no T2DM (BPE-ND) as controls were enrolled. B-mode and power Doppler ultrasonography of the prostate gland were done for both groups.

Results The mean total prostatic volume of the BPE-DM was 79.18 ±8.9 ml, while that of BPE-ND was 60.73 ±10.6 ml ($p < 0.0001$). The mean prostatic resistive index (PRI) was significantly higher among BPE-DM than BPE-ND (0.74 ±0.02 vs 0.68 ±0.09 for right capsular artery; 0.77 ±0.04 vs 0.71 ±0.02 for left capsular artery; and 0.76 ±0.04 vs 0.70 ±0.02 for the urethral artery). BPE-DM with higher glycated haemoglobin, fasting plasma glucose, and longer duration of T2DM experienced more severe lower urinary tract symptoms and had higher PRI.

Conclusions In conclusion, the BPE-DM group presented larger prostate glands and more bothersome LUTS, which correlated with higher PRI. Strict glycaemic control is necessary in men with co-existing BPE and T2DM.

Key Words: arteriosclerosis ‹› benign prostatic enlargement ‹› diabetes mellitus ‹› transrectal Doppler ultrasound

INTRODUCTION

Benign prostatic enlargement (BPE) is common in elderly men and is a leading cause of bladder outflow obstruction and lower urinary symptoms (LUTS) in adult males [1, 2]. BPE results from the proliferation of stromal and glandular elements of the prostate gland [3]. BPE is now the fourth most prevalent disease in men older than 40 years, with approximately one-fourth of men in that age group presenting with BPE-induced LUTS [3]. Worldwide, the prevalence of BPE ranges from 20 to 62% in men over 50 years old [4].

Diabetes mellitus (DM) is a chronic, non-communicable disease that causes long-term damage

to the arteries and other end organs. Type 2 DM (T2DM) accounts for 90% of the estimated 422 million cases of DM globally [5]. T2DM can remain undetected for many years, thus causing extensive microvascular damage in different organs of the body, including the prostate gland [6]. It has been postulated that hypoxia and abnormal blood flow patterns secondary to vascular damage might contribute to hypoxia-stimulated prostate growth [7]. DM, which is notorious for causing vascular damage, may thus predispose to hypoxia of the prostatic tissues, resulting in hypoxia-stimulated proliferation of the stromal and glandular tissues, especially in the transitional zone of the prostate [7].

Some previous studies explored the relationship between T2DM and BPE, and the effect(s) of T2DM on the symptoms of BPE. Van Den Eeden et al., in 2 large multi-ethnic cohorts in California, USA, using the American Urological Association Symptom Index LUTS questionnaire, observed greater severity in LUTS among diabetic men compared to non- diabetic men [8]. In Michigan, United States, Sarma et al. reported significantly more severe LUTS in diabetic males than their non-diabetic counterparts. They also established that diabetic men who were not complying with their medication had an even more severe LUTS score [9]. Another Turkish study, which compared the clinical parameters of BPE in diabetics and non-diabetic patients, found that diabetic patients had significantly higher prostate volume, post-void residual volume (PVRV), and International Prostate Symptoms Score (IPSS). They also recorded a positive correlation between IPSS and PVRV and diagnosis of T2DM, which suggests that co-existing T2DM impacts both static and dynamic components of BPE [10].

The Doppler resistivity index (RI) is related to both blood flow and pressure, and it represents one of the most relevant indicators of vascular damage of small vessels in the prostate [11]. Previous investigators observed that the RI intraprostatic arteries can distinguish normal prostate from those with BPE, and they suggested it to be a useful sonographic parameter for evaluating haemodynamic changes in patients with BPE [12, 13]. Thus, transrectal Doppler ultrasound of the prostatic arteries can be used to assess vascular stiffness in the prostate glands of patients with co-existing T2DM and BPE [14].

The aim of this study was to correlate the Doppler resistive indices of prostatic arteries with the severity of lower urinary tract symptoms (LUTS) and prostate volume in men with concomitant BPE and T2DM.

MATERIAL AND METHODS

Study site, design, and population

This cross-sectional comparative study was carried out at the Department of Radiology of a tertiary hospital from November 2019 to October 2020. The study population comprised 50 adult males with BPE and T2DM (BPE-DM) and 50 age-matched adult males with BPE but no diabetes mellitus (BPE-ND), who were aged 40–90 years, with LUTS, and with a clinical diagnosis of BPE. The study protocol was reviewed and approved by the local Institutional Review Board (Approval number: ERC/2019/08/07). Informed consent was obtained from all subjects at the time of enrolment.

Participants selection

Consenting men with BPE (prostate specific antigen, PSA <4 ng/ml) aged 40 to 90 years, who had been diagnosed with T2DM (fasting plasma glucose level ≥7.0 mmol/l) by the endocrinologist, were recruited consecutively from the endocrinology clinic. Consenting age-matched men with BPE-ND (normal fasting plasma glucose <6 mmol/L) who had been diagnosed with BPE with LUTS based on clinical history, digital rectal examination, PSA <4 ng/ml, and prostatic ultrasonography (prostate gland volume >30 cm^3) [15, 16] were also recruited consecutively from the urology clinic. The exclusion criteria were systemic hypertension, prostate or bladder cancer, prostatitis, urinary bladder calculi, urethral stricture, neurogenic bladder, acute urinary retention, history of transurethral resection of the prostate or previous urinary tract surgery, anal stenosis and previous extensive rectal surgery, and history of smoking.

Clinical and laboratory evaluation

Demographic and clinical data such as age and duration of T2DM were taken. The International Prostatic Symptoms Score and quality of life score were also assessed using standardized IPSS questionnaire [17]. PSA values (≤1 month old) were retrieved from the subjects' records, while their weight and height were measured using a mechanical weighing scale attached to a stadiometer. The BMI was then calculated, using the formula BMI = [weight/height^2 (kg/m^2)]. Fasting venous blood sample, for fasting blood glucose and glycated haemoglobin (HbA1c) assay, was drawn from the antecubital veins of subjects under sterile conditions.

Ultrasonographic technique

All ultrasound examinations were performed using a Mindray Ultrasonography machine (model DC-7; Shenzhen Mindray Bio-medical Electronics, Nanshan, Shenzhen, China, 2014) with Doppler facility equipped with curvilinear transabdominal (3.5–5 MHz) and biplanar endorectal (5.0–10.0 MHz) transducers.

An abdominopelvic ultrasonographic scan (with a full urinary bladder) was done using the curvilinear transducer to exclude urinary tract calculi and masses. Subsequently, the subjects were asked to empty their bowel and urinary bladder in preparation for transrectal ultrasonographic (TRUS) evaluation of their prostate glands. Afterwards, the subjects were positioned in the left lateral decubitus position with the left knee extended and the right knee flexed on the examination table. The head was rested on a pillow to provide additional comfort.

A biplanar endocavitary transducer was used to evaluate the prostate. Coupling gel was applied generously to the transducer surface. A latex sheath condom was used to cover the surface of the transrectal transducer, secured with a rubber band. Additional coupling gel was generously applied to the latex sheath covering. The transducer was inserted gently into the rectum after relaxation of the anal sphincter. The tip of the transducer was initially directed towards the sacrum to follow the curve of the rectum, then inclined anteriorly to locate the prostate gland. The transducer output and receiver gain setting were optimised for each participant.

Multiple transverse and sagittal sections were obtained and recorded after visualizing the transitional zone (TZ) and whole prostate outline. The transverse diameter (TD) and the anteroposterior diameter (APD) of the whole prostate and that of the transition zone (TZTD) and (TZAPD) at the largest cross-sectional area were obtained. Also, the longitudinal diameter (LD) of the whole prostate and that of the transitional zone (TZLD) were measured on the midline sagittal images, respectively. The total prostatic volume (TPV) and transitional zone volume (TZV) were calculated using the formula for volume estimation of an ellipsoid: TPV = (π/6) x LD x APD x TD and TZV = (π/6) x TZLD x TZAPD x TZTD. The transitional zone index (TZI) was also calculated as TZV/TPV [12].

The traced circumference of the prostate (L) and traced circumference of the presumed circular

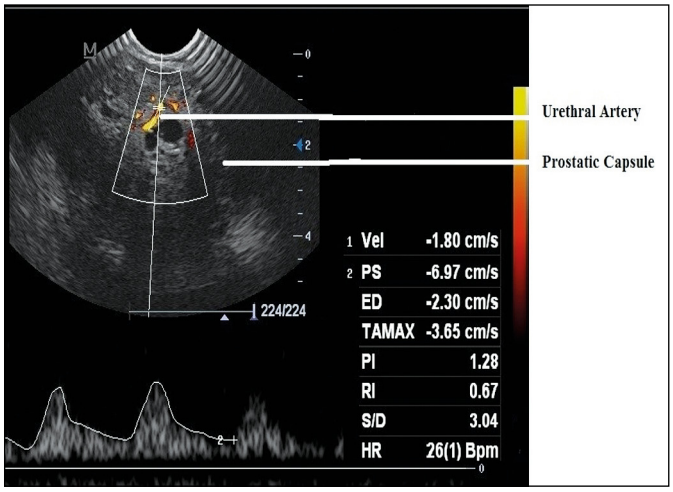

Figure 1. *Triplex Doppler ultrasonogram of the prostate gland in the mid-transverse plane showing the urethral capsular artery.*

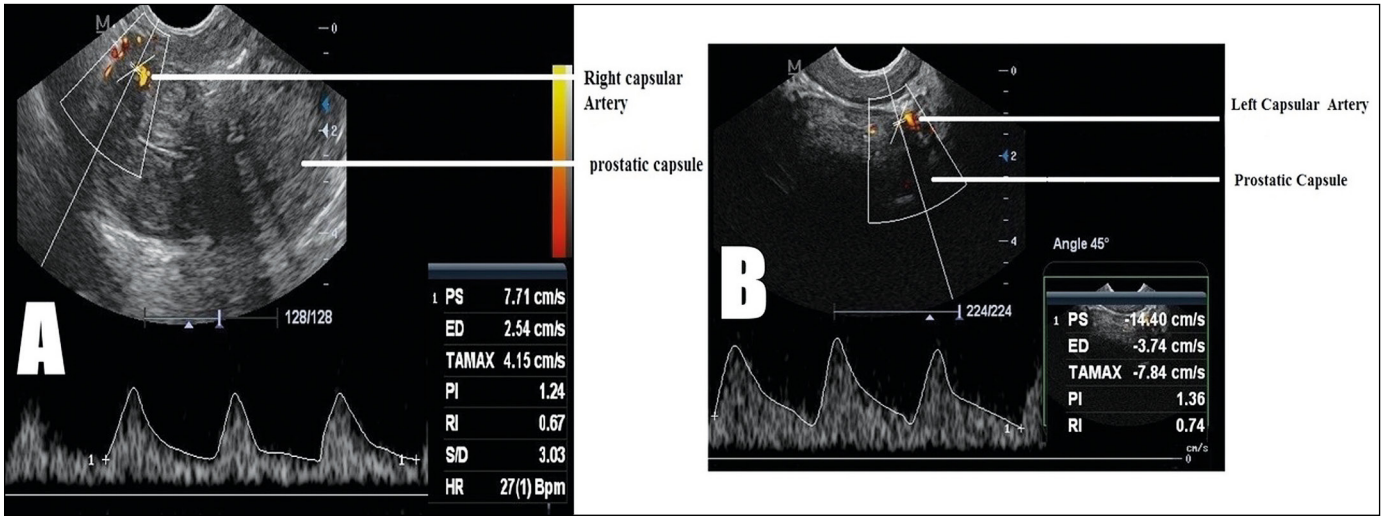

Figure 2. *Triplex Doppler ultrasonogram of the prostate gland in the mid-transverse plane showing the right capsular artery at its right lateral one-third (A) and the left capsular artery at its left lateral one-third (B).*

section of the prostate (L') were measured at the maximum horizontal section of the prostate. The area of the traced circumference of the prostate (S) and the area of the traced circumference of the presumed circular section of the prostate (S') were calculated using the formulas $\pi(L/2\pi)2$ and $\pi(L'/2\pi)2$, respectively. The presumed circle area ratio (PCAR) of the prostate was then calculated as S/S' [18].

Care was taken during power Doppler ultrasonography to minimise transducer pressure on the rectal wall. An empty or near-empty urinary bladder was ensured so that compression effects by either the transducer or full urinary bladder would not falsely increase the intra-prostatic pressure, which could alter the prostatic resistive index. The pulse repetitive frequency (PRF) and power Doppler gain settings were adjusted to allow optimal visualization of the intraprostatic arteries with minimal background noise. Then pulsed wave spectral Doppler images were obtained from the urethral artery, right capsular artery, and left capsular artery on the transverse section of the prostate using an optimised insonation angle of <600 (Figure 1, Figure 2). Once the spectral Doppler waveform was stable, it was traced for 3 pulses. The peak systolic velocity (PSV), end-diastolic flow velocity (EDV), and RI were automatically calculated by the in-built software of the ultrasound scanner. The RI was generated by the software using the equation RI = (PSV) – (EDV)/ PSV [19, 20]. The ultrasound examinations were carried out by the same sonologist (fourth year radiology resident supervised by consultant radiologists) to eliminate interobserver variability. All sonographic measurements were taken thrice, and the mean values were recorded for each subject, to ensure accuracy of the measurements and reduce intraobserver error.

Data analysis

The age, height, weight, IPSS, quality of life (QOL) score, PSA value, PSV, EDV, RI, TPV, TZV, TZI, and PCAR values of both groups (BPE-DM and BPE-ND) were entered into the Statistical Package for Social Sciences (SPSS) version 20 for Windows (IBM Corp., Armonk, NY, USA). Means and standard deviation were derived for the TPV, TZV, TZI, PCAR, and RI values. Categorical variables like age and IPSS score were matched using the chi-squared test. Spearman's 'ρ' correlation was used to determine the association between prostatic morphological parameters and RI of the prostatic arteries. P ≤0.05 was considered statistically significant at 95% confidence interval.

RESULTS

Demographic, clinical, and laboratory characteristics of the study participants

There were 50 participants with BPE and T2DM (BPE-DM/cases) and an equal number of age-matched participants with BPE but no T2DM (BPE-ND/controls). The mean age of cases was 62.90 ±1.63 years, while that of controls was 62.62 ±1.52 years. The mean PSA values were 2.95 ±0.40 and 3.00 ±0.53 (ng/ml) for the cases and controls, respectively. The other demographic, clinical, and laboratory parameters are as shown in Tables 1 and 2. Only the fasting blood glucose showed a statistically significant difference between cases and controls.

Table 1. *Demographic characteristics of the study participants*

	BPE-DM N = 50 (%)	BPE-ND N = 50 (%)	Statistics (df)	p value
Age in years				
Mean (SD)	62.90 (1.63)	62.62 (1.52)		
n (%)				
40–50	6 (12)	6 (12)	-0.14 (98)	0.88
51–60	13 (26)	14 (28)		
61–70	17 (34)	16 (32)		
>70	14 (28)	14 (28)	0.320 (3)	0.956
Occupation				
Professional	8 (16)	7 (14)		
Artisan/trading	20 (40)	15 (30)		
Farming	8 (16)	11 (22)	4.950 (3)	0.176
Armed forces	1 (2)	2 (4)		
Retired	12 (24)	13 (26)		
Other	1 (2)	2 (4)		

BPE-DM – benign prostatic enlargement with diabetes mellitus; BPE-ND – benign prostatic enlargement only (no diabetes mellitus)

Table 2. *Laboratory and clinical characteristics of the study participants*

Variable	BPE-DM Mean (SD)	BPE-ND Mean (SD)	Stat. test (df)	p value
Height (metres)	1.74 (0.05)	1.72 (0.06)	-0.14 (98)	0.88
Weight (kg)	74.60 (3.83)	73.63 (5.47)	-1.87 (98)	0.07
BMI (kg/m^2)	24.76 (1.13)	24.24 (1.63)	-1.83 (98)	0.06
WHR	0.87 (0.04)	0.86 (0.06)	-1.09 (98)	0.28
PSA (ng/ml)	2.95 (0.40)	3.00 (0.53)	0.44 (98)	0.65
FPG (mmol/L)	7.94 (1.72)	4.77 (0.51)	-12.52 (98)	<0.0001
HbA1c (%)	7.89 (1.25)			
Duration of DM (years)	4.79 (2.57)			

BMI – body mass index; BPE-DM – benign prostatic enlargement with diabetes mellitus; BPE-ND – benign prostatic enlargement only (no diabetes mellitus); DM – diabetes mellitus; FPG – fasting plasma glucose; HbA1c – glycated haemoglobin; PSA – prostate-specific antigen; WHR – waist-hip ratio

B-mode prostate ultrasound parameters

The mean total prostatic volume of the BPE-DM was 79.18 ±8.9 ml, while that of BPE-ND was 60.73 ±10.6 ml (p <0.0001). The same trend was observed with the transitional zone volume (TZV), transitional zone index (TZI), and presumed circle area ratio of the prostate (PCAR) (Table 3).

Doppler indices of prostatic arteries

The mean prostatic arteries RI and mean peak systolic velocity (PSV) were significantly higher in BPE-DM than in BPE-ND (p <0.0001). In contrast, there was no statistically significant difference in end-diastolic velocity (EDV) between the 2 groups (Table 4).

Association between prostatic arteries resistivity indices and prostatic B-mode ultrasound parameters

Intermediate to strong positive correlations (p <0.0001) were found between the prostatic arteries indices (RCA-RI, UA-RI, and LCA-RI) and the prostatic volume parameters (TPV, TZV, TZI, and PCAR) in both study groups, i.e. RI values increased with increasing values of prostatic volume parameters (Table 5).

Association between International prostatic symptoms score, resistivity indices of prostatic arteries, anthropometry, and laboratory parameters

A strong positive correlation was recorded between the prostatic arteries indices (RCA-RI, UA-RI, and LCA-RI) and severity of lower urinary tract symptoms (LUTS) in both study groups (Table 6, Table 7). In the BPE-DM group, there was a strong positive correlation between the mean prostatic arteries indices (RCA-RI, UA-RI, and LCA-RI) and the duration since diagnosis of diabetes, HbA1c, and fasting plasma glucose (i.e. patients with higher HbA1c, FBG, and longer duration since diagnosis had higher RI values, and vice versa) (Table 6, Table 7).

Lower urinary tract symptom severity in benign prostatic enlargement and diabetic patients and benign prostatic enlargement and non-diabetic patients

The participants with BPE-DM showed increased LUTS severity compared to BPE-ND. None of the

Table 3. *Prostatic volume parameters of the study population*

Prostate volume parameters	BPE-DM Mean (SD)	BPE-ND Mean (SD)	Stat. test (df)	p value
TPV (cm^3)	79.18 (8.98)	60.73 (10.61)	9.39 (98)	<0.0001
TZV (cm^3)	63.50 (10.27)	44.26 (11.76)	8.71 (98)	<0.0001
TZI	0.80 (0.04)	0.71 (0.07)	6.74 (98)	<0.0001
PCAR	0.82 (0.04)	0.72 (0.06)	9.66 (98)	<0.0001

TPV – total prostatic volume; TZV – transitional zone volume; TZI – transitional zone index; PCAR – presumed circle area ratio of the prostate

Table 4. *Doppler indices of prostatic arteries of the study population*

Prostatic artery velocimetry parameters	BPE-DM Mean (SD)	BPE-ND Mean (SD)	Stat. test (df)	p value
RCA-PSV (cm/s)	29.86 (2.01)	23.84 (3.91)	9.69 (98)	<0.0001
LCA-PSV (cm/s)	23.69 (2.43)	18.35 (2.24)	11.41 (98)	<0.0001
UA-PSV (cm/s)	26.49 (1.77)	20.72 (3.77)	9.79 (98)	<0.0001
RCA-EDV (cm/s)	7.72 (1.03)	7.47 (1.617)	0.91 (98)	0.37
LCA-EDV (cm/s)	5.42 (1.13)	5.26 (0.86)	0.81 (98)	0.41
UA- EDV (cm/s)	6.47 (1.09)	6.23 (1.33)	0.77 (98)	0.44
RCA-RI	0.74 (0.02)	0.68 (0.09)	4.8 (98)	<0.0001
LCA-RI	0.77 (0.04)	0.71 (0.02)	8.8 (98)	<0.0001
UA-RI	0.76 (0.04)	0.70 (0.02)	9.06	<0.0001

RCA – right capsular artery; LCA – left capsular artery; UA – urethral artery; PSV – peak systolic velocity; EDV – end-diastolic velocity; RI – resistivity index

Table 5. *Association between prostatic arterial resistive indices and prostatic volume parameters*

Variables	BPE-DM		BPE-ND	
	Stat. test (ρ)	p value	Stat. test (ρ)	p value
TPV (cm^3)				
RCA-RI	0.74	<0.0001	0.76	<0.0001
UA-RI	0.80	<0.0001	0.82	<0.0001
LCA-RI	0.84	<0.0001	0.82	<0.0001
TZV (cm^3)				
RCA-RI	0.71	<0.0001	0.75	<0.0001
UA-RI	0.79	<0.0001	0.85	<0.0001
LCA-RI	0.82	<0.0001	0.87	<0.0001
TZI				
RCA-RI	0.61	<0.0001	0.66	<0.0001
UA-RI	0.74	<0.0001	0.79	<0.0001
LCA-RI	0.78	<0.0001	0.83	<0.0001
PCAR				
RCA-RI	0.53	0.0001	0.52	0.0001
UA-RI	0.66	0.0001	0.65	<0.0001
LCA-RI	0.74	<0.0001	0.67	<0.0001

RCA – right capsular artery; LCA – left capsular artery; UA – urethral artery; RI – resistivity index; TPV – total prostatic volume; TZV – transitional zone volume; TZI – transitional zone index; PCAR – presumed circle area ratio of the prostate; BPE-DM – benign prostatic enlargement with diabetes mellitus; BPE-ND – benign prostatic enlargement only (no diabetes mellitus)

Table 6. *Association between International prostatic symptoms score (IPSS), prostatic resistivity indices (PRI), anthropometry, and laboratory parameters in the BPE-DM group*

Variables	Stat. test (ρ)	p value
IPSS		
Duration of DM	0.92	<0.0001
BMI	0.23	0.11
WHR	0.25	0.07
HbA1C	0.94	<0.0001
FBG	0.61	<0.0001
PSA	-0.001	0.99
RCA-RI	0.68	<0.0001
URA-RI	0.79	<0.0001
LCA-RI	0.81	<0.0001
RCA-RI		
Duration of DM	0.77	<0.0001
BMI	0.11	0.48
WHR	0.25	0.07
HBA1C	0.75	<0.0001
FBG	0.63	<0.0001
PSA	0.03	0.86
UA-RI		
Duration of DM	0.77	<0.0001
BMI	0.31	0.029
WHR	0.08	0.57
HBA1C	0.79	<0.0001
FBG	0.72	<0.0001
PSA	0.10	0.47
LCA-RI		
Duration of DM	0.82	<0.0001
BMI	0.15	0.30
WHR	0.27	0.06
HbA1C	0.84	<0.0001
FBG	0.65	<0.0001
PSA	-0.02	0.88

RCA – right capsular artery; LCA – left capsular artery; UA – urethral artery; RI – resistivity index; BMI – body mass index; WHR – waist-hip ratio; FBG – fasting blood glucose; PSA – prostatic-specific antigen; HbA1c – glycated haemoglobin; DM – diabetes mellitus

Table 7. *Association between international prostatic symptoms score (IPSS), prostatic resistivity indices (PRI), anthropometry, and laboratory parameters in BPE-ND*

Variables	Stat. test (ρ)	p value
IPSS		
BMI	0.12	0.41
WHR	-0.02	0.90
FBG	0.17	0.25
PSA	0.27	0.06
RCA-RI	0.86	<0.0001
URA-RI	0.94	<0.0001
LCA-RI	0.93	<0.0001
RCA-RI		
BMI	0.27	0.06
WHR	0.16	0.28
FBG	0.07	0.61
PSA	0.21	0.15
UA-RI		
BMI	0.20	0.15
WHR	0.08	0.57
FBG	0.15	0.29
PSA	0.22	0.12
LCA-RI		
BMI	0.19	0.18
WHR	0.08	0.57
FBG	0.07	0.65
PSA	0.23	0.10

RCA – right capsular artery; LCA – left capsular artery; UA – urethral artery; RI – resistivity index; BMI – body mass index; WHR – waist-hip ratio; FBG – fasting blood glucose; PSA – prostatic-specific antigen; HbA1c – glycated haemoglobin; DM – diabetes mellitus; ρ – Spearman's rho

BPE-DM subjects had mild symptoms, 16 (32%) had moderate symptoms, while 34 (68%) had severe symptoms. Conversely, 10 (20%), 15 (30%), and 25 (50%) of the BPE-ND group had mild, moderate, and severe symptoms, respectively. Overall, the BPE-DM group had more severe symptoms than the BPE-ND group (Figure 3).

DISCUSSION

The mean age of the BPE-DM and BPE-ND groups was 62.9 and 62.6 years, respectively, in this study. This is comparable to the mean age bracket recorded for BPE-DM subjects (60.5 years) and BPE-ND subjects (63 years) in a similar study conducted by Berger et al. in Austria [14].

The resistive indices (RI) of all the prostatic arteries were significantly higher in the diabetic group relative to controls. The observed higher RI values in the prostatic arteries of the BPE-DM group in this study suggests that subjects with T2DM have a higher prostatic arterial stiffness/damage than BPE-ND. This finding is similar to the observations of Berger et al., who evaluated atherosclerosis as a risk factor for BPE. They observed significantly higher RI values in both the transitional zone (TZ) and the peripheral zone (PZ) among BPE-DM than in controls [21]. In another study, Berger et al. [14] recorded no significant difference in the RI of the peripheral zone (capsular arteries) between BPE-DM and BPH-ND, but the RI of the transition zone (urethral arteries) was significantly higher in the BPE-ND.

There was a positive correlation between the resistivity indices of the prostatic arteries (RCA-RI, UA-RI, and LCA-RI) and the prostatic volume parameters (TPV, TZV, TZI, and PCAR) in both BPE-DM and BPH-ND groups. This observation is similar to the findings of Abdelwahab et al. [13] and Fanimi et al. [12]. The higher prostatic volume parameters (PVP) and PRI in the BPE-DM group, and the positive correlation between PRI and PVP, suggest that arterial stiffness in the diabetic state (measured by PRI) plays a role in the pathogenesis of prostatic enlargement.

The mean peak systolic velocity (PSV) was significantly higher in the BPE-DM than in the BPH-ND group. This trend may be attributed to increased

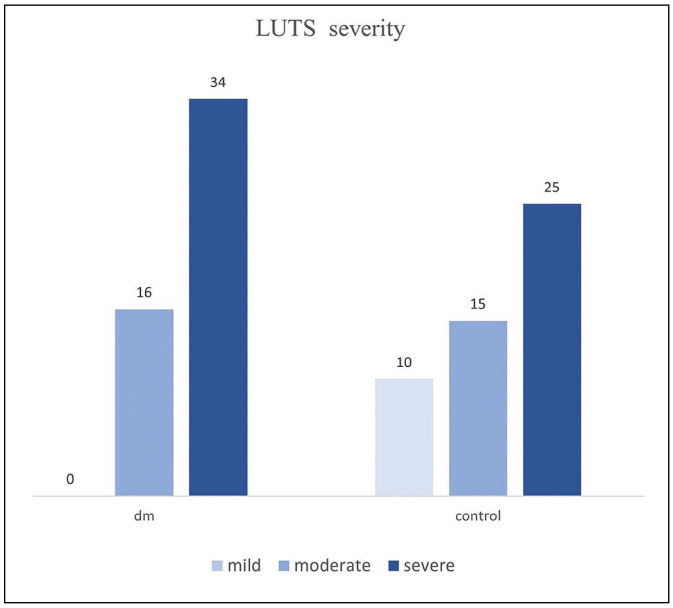

Figure 3. *Bar chart comparing the severity of lower urinary tract symptoms (LUTS) of the diabetic BPE subjects (BPE-DM) and non-diabetic BPE subjects (BPE-ND).*

resistance to blood flow secondary to vascular damage in T2DM. Furthermore, mean PSV values were highest in the RCA and lowest in the LCA, while that of the UA was intermediate between RCA-RI and LCA-RI. We speculate that this pattern might be due to the effect of gravity on blood flow to the LCA in the left lateral decubitus position used in this study. Unfortunately, none of the previous studies commented on the variability of PSV within the prostatic arteries.

There was a positive correlation between the PRI and LUTS severity in both BPE-DM and BPE-ND groups, i.e. subjects with higher RI had more severe LUTS in both study groups. This finding suggests that elevated PRI might be predictive of LUTS severity. Furthermore, there was a positive correlation between the mean PRI and the duration since diagnosis of T2DM, HbA1c, and fasting plasma glucose (FPG) in the BPE-DM group (i.e. BPE-DM patients with higher HbA1c, FPG, and duration since T2DM diagnosis had higher PRI values and vice versa). This finding suggests that poor glycaemic control and increased duration since diagnosis of T2DM worsen the severity of intraprostatic vascular damage, and by extension, LUTS and BPE severity.

The mean total prostate volume was significantly higher in the BPE-DM group (79.2 cm^3) than in the BPE-ND group (60.7 cm^3). This finding corroborates the findings by other researchers who also found significant increases in the observed mean total prostatic volume values among diabetic BPE subjects relative to non-diabetic BPE subjects [10, 22]. This observation suggests that the diabetic state may play a role in the pathogenesis of prostatic enlargement. TZV, TZI, and PCAR were also significantly higher in the BPE-DM group than in the BPE-ND group, further suggesting that T2DM plays a role in the pathogenesis of prostatic enlargement. In contrast, Berger et al. recorded a statistically insignificant difference in the total prostatic volume between BPE-DM (28 cases) and BPE-ND (24 controls) subjects, which might be explained by the smaller sample size in their study [14].

LUTS severity was significantly worse in the BPE-DM group than in the BPE-ND group. This is comparable to the observations of Berger et al. [14], Ferreira et al. [23], and Yin et al. [24]. There was a positive correlation between LUTS severity and duration since diagnosis of T2DM, HbA1c, and FPG in the BPE-DM group (i.e. patients with higher HbA1c, FPG, and duration since diagnosis experienced more severe symptoms, and vice versa). Similarly to the findings of this study, Ferreira et al. demonstrated a statistically significant association between glycaemic control and LUTS severity in their study population [23]. Conversely, Papaefstathiou et al. recorded no significant association between LUTS severity and level of glycaemic control [25].

There were some limitations to this study. Firstly, this study was not population-based, because only patients presenting to the teaching hospital were recruited; hence, elements of selection bias could not be completely eliminated. Secondly, the diagnosis of BPE was clinical and not histological, which may introduce a margin of error into the diagnosis. Finally, the time since diagnosis of T2DM is not entirely accurate in establishing the duration of illness because the illness might have been present before diagnosis.

The findings of this study suggest a possible future role for prostate artery Doppler in patient selection (determining the severity of BPH) for prostatic artery embolization and assessment of treatment response (effectiveness of the procedure) [26].

CONCLUSIONS

In conclusion, the BPE-DM group presented larger prostate glands and more bothersome LUTS, which correlated with higher PRI. Strict glycaemic control is necessary in men with co-existing BPE and T2DM.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

References

1. Vuichoud C, Loughlin KR. Benign prostatic hyperplasia: epidemiology, economics and evaluation. Can J Urol. 2015; 22 Suppl 1: 1-6
2. Apoku IN, Ayoola OO, Salako AA, Idowu BM. Ultrasound evaluation of obstructive uropathy and its hemodynamic responses in southwest Nigeria. Int Braz J Urol. 2015; 41: 556-561.
3. Lee CL, Kuo HC. Pathophysiology of benign prostate enlargement and lower urinary tract symptoms: Current concepts. Ci Ji Yi Xue Za Zhi. 2017; 29: 79-83.
4. Yeboah ED. Prevalence of Benign Prostatic Hyperplasia and Prostate Cancer in Africans and Africans in the Diaspora. J West Afr Coll Surg. 2016; 6: 1-30.
5. Rasaki SO, Kasali FO, Biliaminu SA, et al. Prevalence of diabetes and pre-diabetes in Oke-Ogun region of Oyo State, Nigeria. Cogent Med. 2017; 4: 1326211.
6. Stehouwer CDA. Microvascular Dysfunction and Hyperglycemia: A Vicious Cycle With Widespread Consequences. Diabetes. 2018; 67: 1729-1741.
7. Ghafar MA, Puchner PJ, Anastasiadis AG, Cabelin MA, Buttyan R. Does the prostatic vascular system contribute to the development of benign prostatic hyperplasia? Curr Urol Rep. 2002; 3: 292-296.
8. Van Den Eeden SK, Ferrara A, Shan J, et al. Impact of type 2 diabetes on lower urinary tract symptoms in men: a cohort study. BMC Urol. 2013; 13: 12.
9. Sarma AV, St Sauver JL, Hollingsworth JM, et al. Diabetes treatment and progression of benign prostatic hyperplasia in community-dwelling black and white men. Urology. 2012; 79: 102-108.
10. Ozcan L, Besiroglu H, Dursun M, Polat EC, Otunctemur A, Ozbek E. Comparison of the clinical parameters of benign prostate hyperplasia in diabetic and non diabetic patients. Arch Ital Urol Androl. 2017; 89: 26-30.
11. Kwon SY, Ryu JW, Choi DH, Lee KS. Clinical Significance of the Resistive Index of Prostatic Blood Flow According to Prostate Size in Benign Prostatic Hyperplasia. Int Neurourol J. 2016; 20: 75-80.
12. Fanimi OO, Asaleye CM, Salako AA, Ayoola OO, Adedeji TA, Idowu BM. Transrectal Doppler Sonography of Benign Prostatic Enlargement in Nigerian Men. J Med Ultrasound. 2019; 27: 169-176.
13. Abdelwahab O, El-Barky E, Khalil MM, Kamar A. Evaluation of the resistive index of prostatic blood flow in benign prostatic hyperplasia. Int Braz J Urol. 2012; 38: 250-257.
14. Berger AP, Deibl M, Halpern EJ, et al. Vascular damage induced by type 2 diabetes mellitus as a risk factor for benign prostatic hyperplasia. Diabetologia. 2005; 48: 784-789.
15. Onigbinde SO, Asaleye CM, Salako AA, Idowu BM, Onigbinde AO, Laoye A. The effect of systemic hypertension on prostatic artery resistive indices in patients with benign prostate enlargement. Prostate Int. 2023; 11: 46-50.
16. Bhat SA, Rather SA, Islam N. An overview of benign prostatic hyperplasia and its appreciation in Greco-Arab (Unani) system of medicine. Asian J Urol. 2021; 6: S221438822100045X.
17. Oruqi M, Podvorica E, Islamaj J. The Self-Administered International Prostate Symptoms Score (IPSS) Questionnaire of Kosovo Men with Benign Prostatic Hyperplasia. Open J Urol. 2021; 11: 367-379.
18. St Sauver JL, Jacobson DJ, McGree ME, et al. Presumed circle area ratio of the prostate in a community-based group of men. BJU Int. 2009; 104: 58-62.
19. Idowu BM, Ibitoye BO. Doppler sonography of perifibroid and intrafibroid arteries of uterine leiomyomas. Obstet Gynecol Sci. 2018; 61: 395-403.
20. Idowu BM, Ibitoye BO, Adetiloye VA. Uterine Artery Doppler Velocimetry of Uterine Leiomyomas in Nigerian Women. Rev Bras Ginecol Obstet. 2017; 39: 464-470.
21. Berger AP, Bartsch G, Deibl M, et al. Atherosclerosis as a risk factor for benign prostatic hyperplasia. BJU Int. 2006; 98: 1038-1042.
22. Parsons JK, Carter HB, Partin AW, et al. Metabolic factors associated with benign prostatic hyperplasia. J Clin Endocrinol Metab. 2006; 91: 2562-2568.
23. Ferreira FT, Daltoé L, Succi G, et al. Relation between glycemic levels and low tract urinary symptoms in elderly. Aging Male. 2015; 18: 34-37.
24. Yin Z, Yang JR, Rao JM, Song W, Zhou KQ. Association between benign prostatic hyperplasia, body mass index, and metabolic syndrome in Chinese men. Asian J Androl. 2015; 17: 826-830.
25. Papaefstathiou E, Moysidis K, Sarafis P, Ioannidis E, Hatzimouratidis K. The impact of Diabetes Mellitus on Lower urinary tract symptoms (LUTS) in both male and female patients. Diabetes Metab Syndr. 2019; 13: 454-457.
26. Wadhwa V, McClure TD. Role of Imaging in Prostate Artery Embolization. Semin Roentgenol. 2021; 56: 410-415.