Original Article

Transrectal Doppler Sonography of Benign Prostatic Enlargement in Nigerian Men

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Abstract

Background: Transrectal ultrasonography (TRUS) is the best route for examining the prostate gland because of transducer proximity, elaboration of zonal anatomical details, and Doppler assessment of prostatic arteries' hemodynamics. **Materials and Methods:** This was a cross-sectional study of 300 men with benign prostatic enlargement (BPE) and 300 healthy age-matched controls. The resistive index (RI) of the left capsular, right capsular and urethral arteries were assessed by TRUS and correlated with these parameters: maximum urine flow rate (Q_{max}), total prostatic volume (TPV), transitional zone volume (TZV), transitional zone index (TZI), presumed circle area ratio, and the International Prostatic Symptoms Score (IPSS). **Results:** The RI of capsular and urethral arteries correlated significantly with Q_{max}, TPV, TZV, TZI, and IPSS. Of the three different RIs evaluated, the RI of UA showed the strongest correlation with Q_{max} (r =- 0.51; $P < 0.0001$). The RIs were significantly higher in obstructive BOO than the non-obstructive group (Q_{max} of <15 ml/sec and ≥15 ml/sec, respectively). The mean RI values were 0.73 ± 0.05 vs. 0.63 ± 0.04 for the RCA; 0.73 ± 0.05 vs. 0.62 ± 0.04 for the LCA; and 0.73 ± 0.06 vs. 0.62 ± 0.05 for the UA in the BPE and controls, respectively ($P < 0.001$). The TPV values were 52.36 ± 28.67 and 18.28 ± 4.26 in BPE and controls, respectively ($P < 0.001$).**Conclusion:** Prostatic artery RIs are elevated in BPE. Increase in RI correlated with increase in TPV, TZV and TZI, urinary symptoms' severity, poor QOL, and the severity of BOO.

Keywords: Benign prostatic enlargement, bladder outlet obstruction, prostatic artery resistive index, transrectal Doppler ultrasound, uroflowmetry

Introduction

Benign prostatic hyperplasia (BPH) is the most common benign pathological condition affecting elderly males, resulting in benign prostatic enlargement (BPE), and can be complicated by lower urinary tract symptoms (LUTS) and/or bladder outlet obstruction (BOO). BPH is the second most common indication for surgical intervention in men above the age of 60 years worldwide.[1]

Parameters of this disease which could be evaluated include intraprostatic arteries (capsular and urethral arteries) resistive index (RI), total prostatic volume (TPV), transitional zone volume (TZV), transitional zone index (TZI), presumed circle area ratio (PCAR), maximum urine flow rate (Q_{max}), international prostatic symptoms score (IPSS), quality of life (QOL) score, and postvoidal residual (PVR) volume.[1]

Many ultrasonographic technologies such as B-mode, Color Doppler Ultrasonography (CDUS), and power Doppler ultrasonography have been used in the study of BPH and its hemodynamics through either transrectal or transabdominal route. However, transrectal ultrasonography (TRUS) is the most useful for assessing the volume of BPH,[2-4] delineating the zonal anatomy of the prostate,[5] and for demonstrating the TZI which is a good parameter for predicting acute urinary retention in patients

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with BPH.[4] Addition of Doppler ultrasound to TRUS makes it useful for visualizing the prostatic vascular architecture.[6,7]

Nelson and Pretorius revealed that the RI obtained by Doppler imaging correlates with vascular resistance because velocity is related to both blood flow and pressure.[8] They postulated that RI might be the most relevant index for analyzing small vessels in the prostate.[8] Other researchers have also reported that BPH leads to increased prostatic vascular resistance and elevated prostatic resistive index (PRI).[3,8-10]

The purpose of this study was to describe the transrectal power Doppler ultrasound findings in patients with BPE in our locality and correlate the Prostatic arteries Resistive Index (PRI) with various clinicofunctional parameters of BPE.

Materials and Methods

This was a prospective, cross-sectional study carried out at the radiology department of Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Osun State, Nigeria between February 2014 and June 2015. Three hundred patients with BPE and 300 age-matched controls were recruited consecutively.

The patients were men aged 40–90 years with serum prostatic-specific antigen (PSA) ≤10 ng/ml, clinicoradiological diagnosis of BPE using history and physical findings of an enlarged prostate gland on digital rectal examination (DRE) along with findings of an enlarged prostate gland on transabdominal ultrasound scan (TAUS). An equal number of age-matched and apparently healthy control group was also recruited. The control group was made up of men in whom BPE had been excluded by the absence of LUTS, the presence of normal prostate gland texture on DRE, and a prostatic size <25 cm^3 on TAUS.

Individuals with clinical and sonographic features suggestive of the following diseases were excluded from the study: BOO with severe hydronephrosis or creatinine levels of more than 132.5 μmol/l, prostatitis, urinary bladder stone, urethral stricture as confirmed by retrograde urethrography, acute urinary retention, neurogenic bladder, bladder or prostate cancer, BPE patients on drug treatment, history of prostatic resection, previous lower urinary tract surgery, anal stenosis, and previous extensive rectal surgery. Approval for the study was granted by the hospital's ethics and research committee, and a written informed consent was obtained from all the study participants.

Clinical and laboratory assessment

Clinically, all study participants were assessed by full history and physical examination which included general and DREs. Other routine laboratory and radiological investigations were ordered for all the BPE patients. These included urinalysis, urine microscopy, culture and sensitivity (urine MCS), serum prostate specific antigen (PSA), and abdominopelvic ultrasound (used to exclude other associated pathologies). The clinical assessment was coordinated by the referring urologist.

The IPPS was assessed based on calculated values from the answers given by the individuals to seven questions concerning urinary symptoms and one question concerning the quality of life.[11] Each question concerning urinary symptoms allows the patient to choose one out of six answers indicating increasing severity of the particular symptom. The answers are assigned points from 0 to 5. The total score can range from 0 to 35 (asymptomatic to very symptomatic). Symptoms are categorized as follows: mild (symptom ≤7), moderate (symptom score of 8–19), and severe (symptom score of 20–35).[11]

Venepuncture of the antecubital vein of all BPE patients was done under sterile conditions without any preference for a specific period of time. A syringe was used to collect 5 ml of venous blood into a plain sample bottle; the samples collected were left to clot for 30 min, and the serum was separated and stored in the freezer for bulk assessment of PSA.

Uroflowmetric assessment

Uroflowmetry was done for each BPE patient by the urologist using Albyn Medical uroflowmetry machine with an inbuilt printer (Albyn Medical Ltd, Dingwall, Ross-Shire, Scotland, UK). When the BPE patients had a strong desire to void, they were instructed to hold their urine while the uroflowmeter was prepared for the voiding study for about 10–15 s. The patients were then instructed to void normally into the collecting cylinder of the uroflowmeter. Several automated parameters including the Q_{max} were generated as a function of time. The Q_{max} was the only parameter selected and recorded for each patient with BPE.

Abdominal and transrectal ultrasonographic evaluation

All sonographic examinations were done by the first author, a fourth-year resident in radiology under the supervision of a consultant radiologist. The radiologists were blinded to the status of the patients so as to eliminate bias. An abdominal sonographic evaluation was carried out with the patients lying supine on the examining couch. This was done to rule out some of the exclusion criteria such as severe hydronephrosis and urinary bladder stones.

A transrectal biplanar endocavitary probe with a frequency range of 5.0–10.0 MHz of a Mindray real-time ultrasound scanner model DC-7 (Shenzhen Mindray Bio-medical Electronics, Nanshan, Shenzhen, China) was used for the TRUS. All the participants were scanned in left lateral decubitus position with both knees flexed toward the chest and participant's head was comfortably positioned on a pillow. Coupling gel was generously applied to the probe surface, and a double latex sheath was used to cover the surface of the transrectal probe and secured to its base. The probe was then gently inserted into the rectum by directing the tip of the probe toward the sacrum to follow the curve of the rectum until the probe was properly positioned into the rectum. The surface of the probe was inclined anteriorly to locate the prostate gland. The transducer output and receiver gain settings were optimized for each patient.

Multiple transverse and sagittal sections were obtained to visualize the transitional zone (TZ) and the whole prostatic outline. The transverse diameter (TD) and anteroposterior diameter (APD) of the whole prostate and the transition zone at the largest cross-sectional area were measured and recorded. Furthermore, the superoinferior diameters (SID) of the whole prostate and the TZ were measured on the midline sagittal images and at their largest sagittal diameter respectively and also recorded [Figures 1 and 2].

TPV and TZV were calculated using the ellipsoid formula: Volume = 0.52 × TD × APD × SID.[12] Transition Zone Index (TZI) = TZV/TPV. The PCAR is defined as the ratio of the area of maximum horizontal section of the prostate to the area of a presumed circle with a circumference equal to that of the maximum horizontal section.[13,14] Prerequisite parameters for PCAR were obtained using TRUS, and PCAR was calculated and recorded for only the BPE patients [Figure 3].

During power Doppler imaging, care was taken to minimize probe pressure on the rectal wall, and an empty or nearly empty urinary bladder was ensured so that compression effect by either the probe or full urinary bladder was minimized, which would have increased the intraprostatic pressure and alter the PRI. The power Doppler gain was set to just below the threshold so that blood flow was identified with minimum background noise, and the low flow setting was used for optimal visualization of low flow intraprostatic vessels in the TZ and both sides of the peripheral zone (PZ). Then, the pulsed wave spectral Doppler images were obtained from the left capsular artery (LCA), right capsular artery (RCA), and

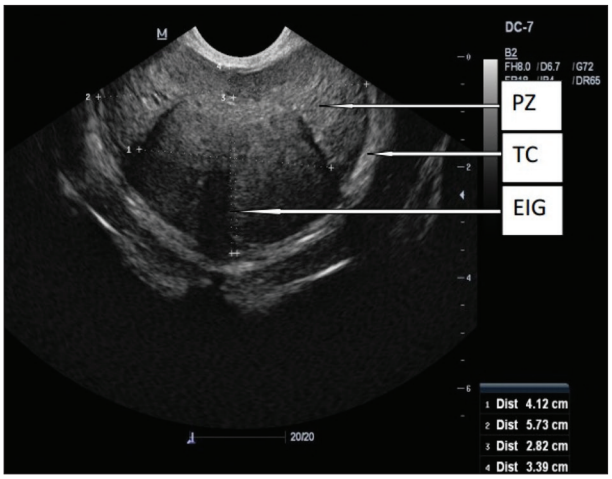

Figure 1: Transrectal sonogram (mid-transverse section) of benign prostatic enlargement showing the slightly compressed peripheral zone by an enlarged inner gland (EIG, Enlarged Inner Gland = central zone + transitional zone). Line 1: Transverse diameter of the enlarged transitional zone; Line 2: Transverse diameter of the whole prostate gland; Line 3: Anteroposterior diameter of the enlarged transitional zone; Line 4: Anteroposterior diameter of the enlarged whole prostate gland. TC: True capsule

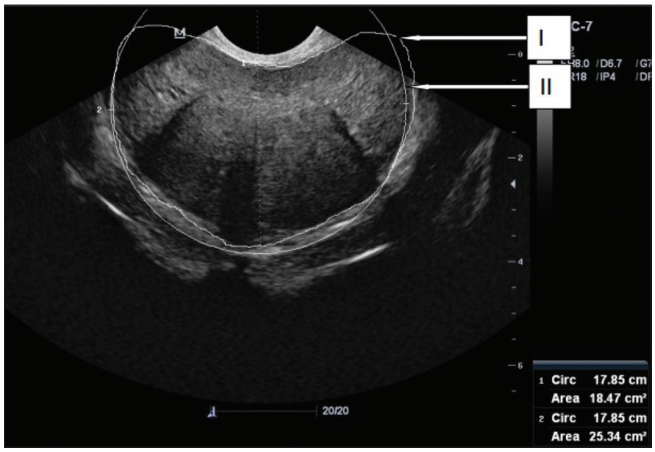

Figure 3: Transrectal sonogram (midtransverse image) of benign prostatic enlargement showing presumed circle area ratio calculation. I: The area of maximum horizontal section of the whole enlarged prostate gland; II: The area of a presumed circle with a circumference equal to that of the maximum horizontal section

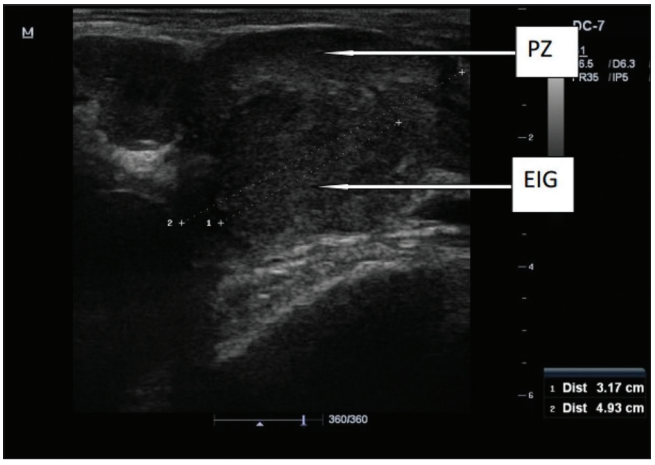

Figure 2: Transrectal sonogram (midsagittal section) of benign prostatic enlargement showing the slightly compressed peripheral zone by an enlarged inner gland (EIG, Enlarged Inner Gland = central zone + transitional zone). Line 1: Superoinferior diameter of the enlarged transitional zone; Line 2: Superoinferior diameter of the enlarged whole prostate

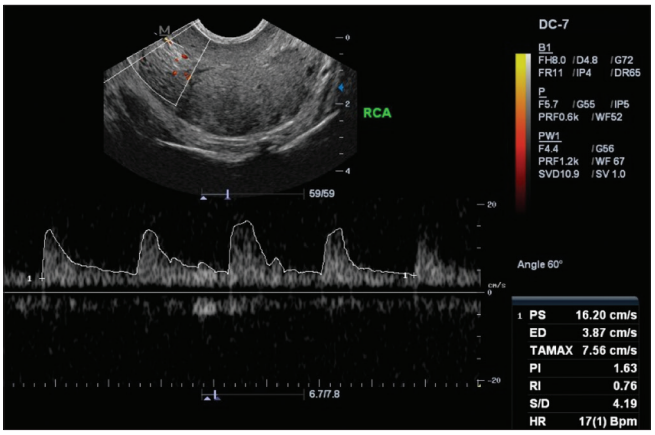

Figure 4: Transrectal triplex power Doppler sonogram of the right capsular artery at the posterolateral aspect of an enlarged prostate gland

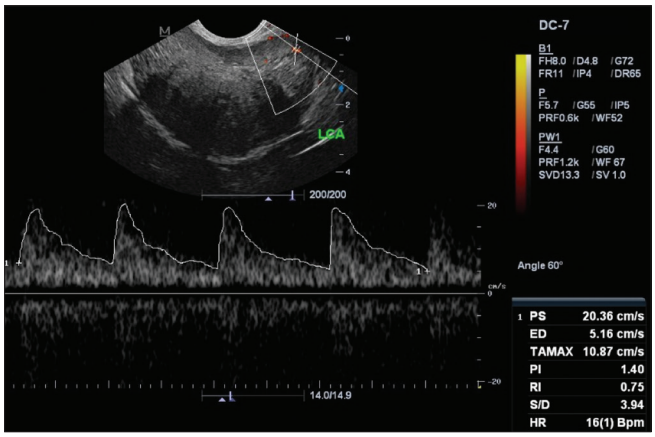

Figure 5: Transrectal triplex power Doppler sonogram of the left capsular artery at the posterolateral aspect of an enlarged prostate gland

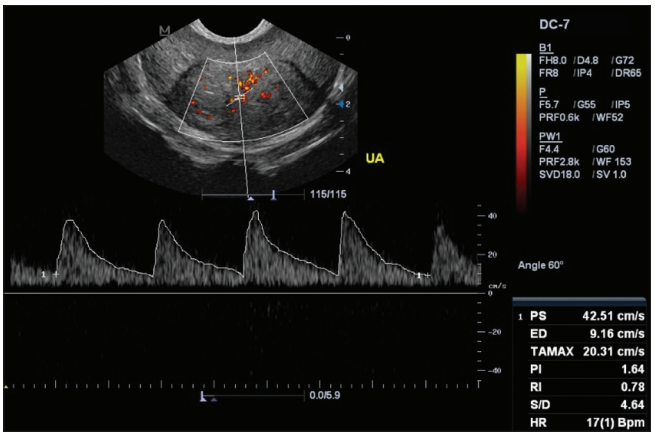

Figure 6: Transrectal triplex power Doppler sonogram of the urethral artery in the periurethral area of an enlarged prostate gland

urethral artery (UA) on the transverse section of the prostate [Figures 4-6].[11]

Data analysis was done using the Statistical Package for Social Science (SPSS) software for windows, version 16 (SPSS Inc., Chicago, IL, USA). Mean ± standard deviations were generated for the TPV, TZV, TZI, PCAR, RI, and other normally distributed continuous variables while the categorical variables were reported as frequencies and percentages. Correlation was determined using Pearson's correlation for normally distributed data or Spearman's correlation for data that were not normally distributed. Student's *t*-test was used to determine differences in mean of the parameters. One-way analysis of variance (ANOVA) was used to compare means of variables where there were three or more groups. $P \leq 0.05$ was considered as statistically significant.

Results

There were 600 participants in this study comprising 300 BPE patients and 300 apparently healthy age-matched controls. There was no statistically significant difference in the mean age of the BPE group (67.92 ± 8.96 years) and that of the control group (67.32 ± 10.60 years); $P = 0.454$.

The mean Q_{max} for the BPE group was 11.86 ± 6.89 m/s. The median value of the IPSS for BPE patients was 18.0 (range = 0.0–35.0). The frequency of the symptom severity based on IPSS is shown in Table 1. The severity of BOO in the BPE patients was further categorized into obstructed group with 214 (71.3%) patients and nonobstructed group with 86 (28.7%) patients using Q_{max} <15 ml/s as obstructed and Q_{max} ≥15 m/s as nonobstructed.

There were significant differences in the mean RI of the LCA, RCA, and UA between the BPE patients and the controls [Table 2]. The mean values of the TPV, TZV, TZI, and PCAR for the BPE patients and the controls are also shown in Table 2. The differences between the intraprostatic arteries mean RI of BPE patients with obstructed BOO and those with nonobstructed BOO were statistically significant [Table 3].

Table 1: Demographic characteristics of study participants

Characteristics	BPE group (n=300)	Controls (n=300)
Age		
Mean±SD (years)*	67.92±8.96	67.32±10.60
Clinical parameters		
IPSS**	18.0 (0.0-35.0)	NA
Q_{max} (ml/s)	11.86±6.89	NA
Symptom severity, *n* (%)		
Mild (IPSS 0-7)	57 (19.0)	NA
Moderate (IPSS 8-19)	106 (35.3)	NA
Severe (IPSS 20-35)	137 (45.7)	NA
BOO, *n* (%)		
Obstructed	214 (71.3)	NA
Nonobstructed	86 (28.7)	NA

*t=0.749, df=598; P=0.454, **Median (range) values. NA: Not applicable; BOO: Bladder outlet obstruction, BPE: Benign prostate enlargement, IPSS: International Prostatic Symptoms Score, SD: Standard deviation, Q_{max}: Maximum urine flow rate

Within the BPE group, there was a negative correlation between the intraprostatic arteries RI (RCA, LCA, and UA) and Q_{max} as well as a positive correlation between the intraprostatic arteries RI and the following variables: TPV, TZV, TZI, and IPSS [Table 4].

A point-biserial correlation analysis using the classification of BOO into either obstructed or nonobstructed showed significant correlation between the RI of intraprostatic arteries and grade of BOO. There was a positive correlation between the RI of RCA, LCA, and UA with the grade of BOO with r values 0.34, 0.23, and 0.40, respectively ($P < 0.001$).

The receiver operating characteristic (ROC) coordinates at cutoff RI values of ≥0.70 for the RCA, LCA, and UA are shown in Table 5. One-way ANOVA for differences in mean Doppler parameters by symptom severity (based on IPSS) is shown in Table 6.

Table 2: Transrectal ultrasound parameters among benign prostate enlargement and controls (*n*=600)

Characteristics	BPE patients (*n*=300)	Controls (*n*=300)	*t*	df	*P*
PRI					
RI (RCA)	0.73±0.05	0.63±0.04	31.192	598	<0.0001
RI (LCA)	0.73±0.05	0.62±0.04	31.226	598	<0.0001
RI (UA)	0.73±0.06	0.62±0.05	27.463	598	<0.0001
Prostatic volume					
TPV (ml)	52.36±28.67	18.28±4.26	20.360	598	<0.0001
TZV (ml)	26.63±21.33	NA			
TZI	0.47±0.14	NA			
PCAR	0.76±0.07	NA			

RCA: Right capsular artery, LCA: Left capsular artery, UA: Urethral artery, TPV: Total prostatic volume, TZV: Transition zone volume, TZI: Transition zone index, PCAR: Presumed circle area ratio, RI: Resistive index, PRI: Prostatic resistive index, NA: Not applicable, TRUS: Transrectal ultrasound, BPE: Benign prostate enlargement

Table 3: Variable differences between benign prostate enlargement patients with obstructed and nonobstructed benign prostate enlargement

Variables	Obstructed (*n*=214)	Nonobstructed (*n*=86)	*t*	df	*P*
RI (RCA)	0.74±0.04	0.71±0.04	6.191	298	<0.0001
RI (LCA)	0.74±0.05	0.72±0.04	4.096	298	<0.0001
RI (UA)	0.75±0.05	0.70±0.05	7.629	298	<0.0001
TPV (ml)	58.93±30.99	36.16±10.71	6.667	298	<0.0001
TZV (ml)	32.03±22.73	13.31±7.40	7.49	298	<0.0001
TZI	0.51±0.13	0.36±0.10	9.564	298	<0.0001
PCAR	0.77±0.08	0.74±0.05	2.513	298	0.012
IPSS*	20.27±8.77	10.89±5.64	9.202	298	<0.0001

*Mann-Whitney U-test applied. RCA: Right capsular artery, LCA: Left capsular artery, UA: Urethral artery, TPV: Total prostatic volume, TZV: Transition zone volume, TZI: Transition zone index, PCAR: Presumed circle area ratio, IPSS: International prostate symptom score, RI: Resistive index

Discussion

The term "BPE" rather than "benign prostatic hypertrophy (BPH)" is preferable in the absence of histological confirmation.[15] BPE is common in our population with a community prevalence of about 24%[16] and also accounts for up to 27% of obstructive uropathy cases.[17] Although pressure flow studies (PFS) are considered the most reliable method for evaluating BOO, they are invasive and complicated.[18] TRUS is a promising alternative to PFS because of its minimal invasiveness.[19,20]

The mean TPV in this study (52.36 ± 28.67 ml) is lower than that reported by Abdelwahab *et al*.[21] (75.1 ± 44.7 ml in 82 men with BPE) but higher than that of Chung *et al*.[22] (41.7 ± 8.99 ml in 110 men with BPE). These disparities may be due to differences in sample size and differences in disease severity.[23,24] On the other hand, the mean TPV values obtained in this study for both BPE patients and controls fall within the range reported by some previous studies.[12,21,25] The mean TZV (26.63 ± 21.33 ml) of our BPE patients is similar to that reported by Greene *et al*. (24.81 ± 14.4 ml)[26] while our mean TZI (0.47 ± 0.14) is lower than that reported by Tsuru *et al*. (0.51 ± 0.19).[10]

In the BPE patients, there were significant negative correlations between the RI of the three intraprostatic arteries and BOO (as measured by uroflowmetry), with UA RI showing the strongest correlation with Q_{max} among the three arteries. This may be due to the fact that BPE usually starts from the TZ which is supplied by the UA. Glandular enlargement leads to an increase in vascular resistance thereby resulting in increased RI secondary to elevated intraprostatic pressure. Therefore, as the TZ significantly enlarges, it compresses the PZ and its blood supply (left and right capsular arteries) against the true capsule of the prostate gland. This implies that among the intraprostatic arteries, the UA is the first and the worst affected over time in the hemodynamics of BPE. The opinion that RI is sensitive to intraprostatic pressure in patients with BPE, whereby the enlarged inner gland compresses on the capsule of the prostate, is corroborated by Shinbo *et al*.[18] However, Berger *et al*.[27] reported that vascular damage within the prostate gland induces prostatic enlargement.

A point-biserial correlational analysis in the BPE patients showed a significant correlation between the intraprostatic arteries RI and BOO. The UA RI showed the strongest correlation with BOO while the LCA RI showed the weakest correlation with BOO. These findings are similar to those reported by Ozdemir *et al*.[9] while Tsuru *et al*.[28] documented a significant correlation between RI of the two capsular arteries and BOO but no correlation between RI of the UA and BOO. The reason for this discrepancy could not be fully explained.

Furthermore, statistically significant differences were noted in the relationship between the degree of severity of BOO (i.e., obstructed BOO vs. nonobstructed BOO) and the three

intraprostatic arteries. As the severity of BOO increases, the PRIs increase which is the same pattern reported by earlier researchers.[3,9,10,18,21] Increasing severity of BOO (as assessed by uroflowmetry) is associated with progressive reduction in peak urine flow rate. Therefore, as severity of BOO worsens, Q_{max} decreases and PRIs increase.

Table 4: Correlation between prostatic resistive index and other Doppler parameters among benign prostate enlargement patients ($n=300$)

Variables	*r*	*P*
RI (RCA) versus		
Q_{max}	−0.415	<0.0001
TPV (ml)	0.447	<0.0001
TZV (ml)	0.450	<0.0001
TZI	0.430	<0.0001
PCAR	0.108	0.061
IPSS*	0.437	<0.0001
RI (LCA) versus		
Q_{max}	−0.352	<0.0001
TPV (ml)	0.403	<0.0001
TZV (ml)	0.437	<0.0001
TZI	0.446	<0.0001
PCAR	0.129	0.025
IPSS*	0.410	<0.0001
RI (UA) versus		
Q_{max}	−0.511	<0.0001
TPV (ml)	0.508	<0.0001
TZV (ml)	0.539	<0.0001
TZI	0.482	<0.0001
PCAR	0.229	<0.0001
IPSS*	0.493	<0.0001

*Spearman's correlation applied. Q_{max}: Maximum (peak) urine flow rate, RCA: Right capsular artery, LCA: Left capsular artery, UA: Urethral artery, TPV: Total prostatic volume, TZV: Transition zone volume, TZI: Transition zone index, PCAR: Presumed circle area ratio, IPSS: International prostate symptom score, RI: Resistive index

We correlated the resistive indices of the prostatic arteries with each of the prostatic parameters such as TPV, TZV, TZI, PCAR, and IPSS. There were statistically significant positive correlations among RI of RCA, LCA, and UA with TPV, TZV, TZI, and IPSS, respectively. These findings are corroborated by previous studies.[9,21,22,28]

There was no correlation between RI of RCA and LCA with PCAR, but a very weak correlation was observed between UA, RI, and PCAR. These imply that as PCAR increases, no increase is seen in RI RCA and RI LCA, respectively. This is in contrast to the report of Tsuru *et al*.[28] who obtained an r value of 0.334 ($P < 0.001$) with the capsular arteries, and a value of 0.018 ($P > 0.05$) for the UA.

The prostatic neurovascular bundles run craniocaudally along the posterolateral aspects of the gland.[29] Furthermore, a rich plexus of veins (periprostatic venous plexus) encompasses the prostate gland between the true fibrous capsule of the gland and the lateral prostatic fascia; these are visible landmarks on sonograms.[29] They are seen outside the prostate gland as a number of tortuous echo-void tubular structures disposed along the lateral borders of the gland on B-mode imaging.[6] The prostate gland is supplied by the prostatic artery, which is usually one of the two terminal branches of prostaticovesical artery (a branch of internal iliac artery).[6] The anechoic tubular prostatic artery has a curvilinear course which may be impossible to demonstrate with longitudinal scan.[6] The prostatic artery divides into two groups of intraprostatic arteries which are the UA (supplies the periurethral region) and capsular arteries (supply the peripheral zone on both sides).[6,29] The capsular and urethral arteries supply two-third and one-third of the glandular prostate volume, respectively.[6] The prostatic and both capsular arteries mainly course along the posterolateral border of the prostate sending branches that perforate the capsule of the prostate gland and enter the peripheral zone.

Table 5: Receiver operating characteristic coordinates for resistive index in the right capsular artery, left capsular artery, and urethral artery

	RI						Area under the curve	*P*
	0.70	0.71	0.72	0.73	0.74	0.75		
RCA								
Sensitivity (%)	87.0	81.5	76.0	67.1	58.2	45.2	0.705	<0.0001
Specificity (%)	39.0	44.1	54.2	62.7	71.2	79.7		
Overall predictability (%)	73.2	70.8	69.8	65.8	61.9	55.1		
LCA								
Sensitivity (%)	86.3	79.5	74.0	63.7	54.8	47.3	0.651	0.001
Specificity. (%)	30.5	40.7	50.8	59.3	66.1	69.5		
Overall predictability (%)	70.3	68.4	67.3	62.4	58.0	53.7		
UA								
Sensitivity (%)	82.2	78.1	73.3	65.1	58.9	51.4	0.760	<0.0001
Specificity (%)	50.8	55.9	61.0	72.9	76.3	84.7		
Overall predictability (%)	73.2	71.7	69.8	67.3	63.9	60.9		

RCA: Right capsular artery, LCA: Left capsular artery, UA: Urethral artery, RI: Resistive index, BPE: Benign prostate enlargement, ROC: Receiver operator characteristic curve

Table 6: One-way analysis of variance for differences in mean Doppler parameters by symptom severity

Variables	Symptom severity (IPSS)			*F*	df	*P*
	Mild (*n*=57)	Moderate (*n*=106)	Severe (*n*=137)			
RI (RCA)	0.70±0.04	0.73±0.05	0.75±0.04	25.077	2, 296	<0.0001
RI (LCA)	0.70±0.04	0.73±0.05	0.75±0.05	24.630	2, 296	<0.0001
RI (UA)	0.70±0.04	0.72±0.05	0.76±0.05	32.009	2, 296	<0.0001
TPV (cm^3)	37.16±11.49	45.78±19.37	63.76±34.64	25.071	2, 296	<0.0001
TZV (cm^3)	15.04±8.83	21.34±14.48	35.53±25.45	27.776	2, 296	<0.0001
TZI	0.39±0.12	0.44±0.14	0.52±0.14	22.815	2, 296	<0.0001
PCAR	0.75±0.05	0.75±0.05	0.77±0.09	1.768	2, 296	0.172
BWT (cm)	0.34±0.06	0.42±0.10	0.48±0.13	36.144	2, 296	<0.0001
Scheffe *post hoc* analysis for intergroup differences						
	Mild versus moderate (*P*)	Mild versus severe (*P*)	Moderate versus severe (*P*)			
RI (RCA)	0.002	<0.0001	0.001			
RI (LCA)	<0.0001	<0.0001	0.005			
RI (UA)	0.014	<0.0001	<0.0001			
TPV (cm^3)	0.146	<0.0001	<0.0001			
TZV (cm^3)	0.152	<0.0001	<0.0001			
TZI	0.109	<0.0001	<0.0001			
BWT (cm)	<0.0001	<0.0001	<0.0001			

BWT: Bladder wall thickness; ANOVA: Analysis of variance, RCA: Right capsular artery, LCA: Left capsular artery, UA: Urethral artery, TPV: Total prostatic volume, TZV: Transition zone volume, TZI: Transition zone index, PCAR: Presumed circle area ratio, IPSS: International prostate symptom score, RI: Resistive index

The capsular arteries are seen as transversely oriented color densities in the lateral one-third (periphery) of the gland.[6] Conversely, the urethral arteries course parallel to the prostatic urethra after they enter the prostate at the bladder neck.[6] The urethral arteries are seen as a number of straight longitudinal color densities that are parallel to the prostatic urethra, in the midline of the prostate.[6] Generally, B-mode cannot depict the intraprostatic vasculature because the lumen of the parenchymal vessels are too small to be detectable.[6]

The intraprostatic arteries show low impedance (pulsatile blood flow that was steady throughout systole and diastole) and slow flow velocities on spectral Doppler.[6] This is in contrast to the velocity waveforms of the main prostatic arteries which are high impedance type with sharp, narrow systolic peaks and antegrade low diastolic flow.[6] The mean RI and peak systolic velocity of urethral and capsular arteries are similar.[6]

Conclusion

Combination of TRUS with power Doppler is an effective imaging method for evaluating prostatic enlargement which can identify BPE patients at risk of developing BOO using the PRIs.

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Conflicts of interest

There are no conflicts of interest.

References

1. Poulsen AL, Schou J, Puggaard L, Torp-Pedersen S, Nordling J. Prostatic enlargement, symptomatology and pressure/flow evaluation: Interrelations in patients with symptomatic BPH. Scand J Urol Nephrol Suppl 1994;157:67-73.
2. Kojima M, Naya Y, Inoue W, Ukimura O, Watanabe M, Saitoh M, *et al.* The American Urological Association symptom index for benign prostatic hyperplasia as a function of age, volume and ultrasonic appearance of the prostate. J Urol 1997;157:2160-5.
3. Kojima M, Ochiai A, Naya Y, Ukimura O, Watanabe M, Watanabe H, *et al.* Correlation of presumed circle area ratio with infravesical obstruction in men with lower urinary tract symptoms. Urology 1997;50:548-55.
4. Kurita Y, Masuda H, Terada H, Suzuki K, Fujita K. Transition zone index as a risk factor for acute urinary retention in benign prostatic hyperplasia. Urology 1998;51:595-600.
5. McNeal J. Pathology of benign prostatic hyperplasia. Insight into etiology. Urol Clin North Am 1990;17:477-86.
6. Neumaier CE, Martinoli C, Derchi LE, Silvestri E, Rosenberg I. Normal prostate gland: Examination with color Doppler US. Radiology 1995;196:453-7.
7. Rubin JM, Bude RO, Carson PL, Bree RL, Adler RS. Power doppler US: A potentially useful alternative to mean frequency-based color Doppler US. Radiology 1994;190:853-6.
8. Nelson TR, Pretorius DH. The Doppler signal: Where does it come from and what does it mean? AJR Am J Roentgenol 1988;151:439-47.
9. Ozdemir H, Onur R, Bozgeyik Z, Orhan I, Ogras MS, Ogur E, *et al.* Measuring resistance index in patients with BPH and lower urinary tract symptoms. J Clin Ultrasound 2005;33:176-80.
10. Tsuru N, Kurita Y, Suzuki K, Fujita K. Resistance index in benign prostatic hyperplasia using power Doppler imaging and clinical outcomes after transurethral vaporization of the prostate. Int J Urol 2005;12:264-9.
11. Barry MJ, Fowler FJ Jr., O'Leary MP, Bruskewitz RC, Holtgrewe HL, Mebust WK, *et al.* The American Urological Association symptom index for benign prostatic hyperplasia. The Measurement Committee of the American Urological Association. J Urol 1992;148:1549-57.
12. Brant. WE. The Core Curriculum Ultrasound. 1st ed. Philadelphia:

Lippincott Williams & Wilkins; 2001. p. 499-505.
13. St Sauver JL, Jacobson DJ, McGree ME, Girman CJ, Nehra A, Lieber MM, *et al.* Presumed circle area ratio of the prostate in a community-based group of men. BJU Int 2009;104:58-62.
14. Watanabe H. Prostatic ultrasound. Genitourinary Ultrasonography. New York, Churchill Livingstone. 1979: 125-137.
15. Abrams P. LUTS, BPH, BPE, BPO: A Plea for the logical use of correct terms. Rev Urol 1999;1:65.
16. Ojewola RW, Oridota ES, Balogun OS, Alabi TO, Ajayi AI, Olajide TA, *et al*. Prevalence of clinical benign prostatic hyperplasia amongst community-dwelling men in a South-Western Nigerian rural setting: A cross-sectional study. Afr J Urol 2017;23:109-15.
17. 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;41:556-61.
18. Shinbo H, Kurita Y. Application of ultrasonography and the resistive index for evaluating bladder outlet obstruction in patients with benign prostatic hyperplasia. Curr Urol Rep 2011; 12:255-60.
19. Tyloch JF, Wieczorek AP. The standards of an ultrasound examination of the prostate gland. Part 1. J Ultrason 2016;16:378-90.
20. Tyloch JF, Wieczorek AP. The standards of an ultrasound examination of the prostate gland. Part 2. J Ultrason 2017;17:43-58.
21. 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;38:250-5.
22. Chung JH, Lee DH, Shim BS. Utility of the resistive index measured by color doppler ultrasonography in benign prostatic hyperplasia. Korean J Urol 2002;43:956-9.
23. Fowke JH, Murff HJ, Signorello LB, Lund L, Blot WJ. Race and socioeconomic status are independently associated with benign prostatic hyperplasia. J Urol 2008;180:2091-6.
24. Platz EA, Kawachi I, Rimm EB, Willett WC, Giovannucci E. Race, ethnicity and benign prostatic hyperplasia in the health professionals follow-up study. J Urol 2000;163:490-5.
25. Moore KL DA. Clinically Oriented Anatomy. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 407-9.
26. Greene DR, Egawa S, Hellerstein DK, Scardino PT. Sonographic measurements of transition zone of prostate in men with and without benign prostatic hyperplasia. Urology 1990;36:293-9.
27. Berger AP, Deibl M, Leonhartsberger N, Bektic J, Horninger W, Fritsche G, *et al.* Vascular damage as a risk factor for benign prostatic hyperplasia and erectile dysfunction. BJU Int 2005;96:1073-8.
28. Tsuru N, Kurita Y, Masuda H, Suzuki K, Fujita K. Role of doppler ultrasound and resistive index in benign prostatic hypertrophy. Int J Urol 2002;9:427-30.
29. Shetty S, Faraj K, Shetty Z, Dave CN, Talavera F, Schwartz BF, *et al.* Transrectal Ultrasonography of the Prostate. Available from: https://www.emedicine.medscape.com/article/457757-overview. [Last accessed on 2018 Dec 09].