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ORIGINAL ARTICLE

The value of measuring the prostatic resistive index vs. pressure-flow studies in the diagnosis of bladder outlet obstruction caused by benign prostatic hyperplasia

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ABBREVIATIONS

BPE, benign prostatic enlargement; PFS, pressure-flow studies; (p)RI, (prostatic) resistive index; PDI, power Doppler imaging;

Abstract ***Objective:*** To compare the prostatic resistive index (RI) and measurements from pressure-flow studies (PFS) for the diagnosis and follow-up of bladder outlet obstruction (BOO) in patients with benign prostatic hyperplasia (BPH).

Patients and methods: In all, 338 men (aged 55–82 years) presenting with lower urinary tract symptoms were evaluated prospectively for BOO secondary to BPH. In all patients, the prostatic RI was measured by transrectal power Doppler ultrasonography. PFS were assessed in all patients and depending on the results the patients were divided into an obstructive and an unobstructive group. The receiver operating characteristic (ROC) curve was used to determine the prostatic RI threshold value for predicting BOO secondary to BPH. Patients who were confirmed to have BOO secondary to BPH received either medical or surgical treatment, and they were re-evaluated after 3 and 6 months with prostatic RI measurements.

Results: According to the PFS the obstructive group included 158 patients and the unobstructive group 180 patients. The mean (SD) prostatic RI was significantly higher in the obstructive group, at 0.73 (0.04), than in the unobstructive group, 0.65 (0.05) ($P < 0.001$). Using the ROC curve a prostatic RI of $\geqslant 0.71$ predicted BOO

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Q_{max}, maximum urinary flow rate; Pdet, detrusor pressure; AG, Abrams–Griffiths; PV, prostate volume; TZ, transition zone; ROC, receiver operating characteristic.

secondary to BPH, with 84.6% sensitivity, 78.4% specificity and 83.8% overall predictability. After management, the prostatic RI of the obstructive group, 0.69 (0.08), was significantly lower than before treatment, 0.73 (0.04) ($P < 0.05$).

Conclusions: The prostatic RI can predict BOO with high specificity and sensitivity. We believe that the prostatic RI could be a useful variable for the diagnosis and follow-up of patients with BPH.

Introduction

LUTS are common complaint in the ageing male, and the progressive growth of the aged population has broadened the social effect of LUTS. The EPIC study [1], using the 2002 ICS standardised terminology for LUTS [2], showed an overall prevalence of LUTS of about two-thirds of men aged $\geqslant$40 years. The term LUTS was introduced by Abrams and has been adopted as the correct terminology to apply to any patient, regardless of age or sex, with urinary symptoms but without implying the underlying problem. The term LUTS has replaced the term 'prostatism', as the latter implies causality. Prostatism unfortunately implied that the cause of the problem was prostate, which later was found clearly not to be the case in some patients [3]. Despite this, men with LUTS are often presumed to have BOO resulting from BPH. However, numerous studies have shown that the association between LUTS and BOO is uncertain [4].

BPH is one of the most common benign diseases in men that can lead to benign prostatic enlargement (BPE), LUTS, and/or BOO. A third to a half of men with histological signs of BPH have a prostate volume of >25 mL (BPE), and up to 28% have moderate to severe LUTS [5]. BOO was detected in ≈60% of symptomatic and 52% of asymptomatic men with BPH [6]. No clear association between LUTS, BPE and BOO has been found so far [7]. Therefore, each variable of this disease must be evaluated separately. Estimating prostate size, by a DRE or ultrasonographic measurement, and LUTS, by a history or the IPSS questionnaire, is quick and simple. The evaluation of BOO is more difficult.

Pressure-flow studies (PFS), measuring voiding detrusor pressure and urinary flow rate, remain the standard for diagnosing BOO [8], but they are invasive, expensive and have associated morbidity. Accordingly, a noninvasive test would be a useful adjunct for diagnosing BOO and planning the management of patients with LUTS.

The hyperplastic prostate looks like a closed system in which the outer capsule surrounds the inner glandular tissue. In patients with BPH the intraprostatic pressure increases. This has been supported by the correlation of the urethral pressure profile with the size of the prostatic adenoma resected at surgery [9]. Along with the prostatic urethra, the increased intraprostatic pressure must also compress the blood vessels running in the prostate. In recent years the prostatic resistive index (pRI), measured by power Doppler imaging (PDI), has been used to evaluate patients with BPH [10]. Kojima et al. [11] were the first to propose the pRI as a diagnostic tool to differentiate patients with BPH and 'normal' patients. Researchers reported that the development of BPH leads to an increase in vascular resistance and pRI values. Also, the RI of the prostatic capsular arteries positively correlated with the IPSS and negatively with the maximum urinary flow rate (Q_{max}) [12]. Several reports have shown that the pRI is increased in patients with BOO and is related to the severity of BOO [10,11,13].

The aim of the present study was to review the reliability and practical implications of the pRI for investigating men with BOO due to BPH.

Patients and methods

The study was conducted prospectively between January 2010 and November 2010. In all, 338 men (aged 55–82 years, mean 61) with LUTS were included. Patients with a known history of previous lower urinary tract surgery, prostate or bladder carcinoma, urinary retention, prostatitis, bladder calculi, patients with a PSA level of >4 ng/mL, VUR, urethral stricture, neurological deficit or using an α-blocker, anticholinergics, antiandrogens or any medications that affect micturition, were excluded from the study.

The initial evaluation consisted of a past medical history, IPSS and the quality-of-life score, physical examination, neurological examination, DRE, urine analysis, renal function tests and serum PSA estimate.

PFS were assessed using the Delphis (Laborie, Canada) machine with computer software for calculations and graphs. PFS start with uroflowmetry to measure the Q_{max}, and then the patient is catheterised to measure the residual urinary volume. A double-lumen catheter was introduced through the urethra. The catheter allows for bladder filling and monitoring intravesical pressure. The bladder was filled with saline at 30–50 mL/min. Rectal pressure was recorded through a 10-F balloon catheter in the rectum. Filling cystometry was stopped

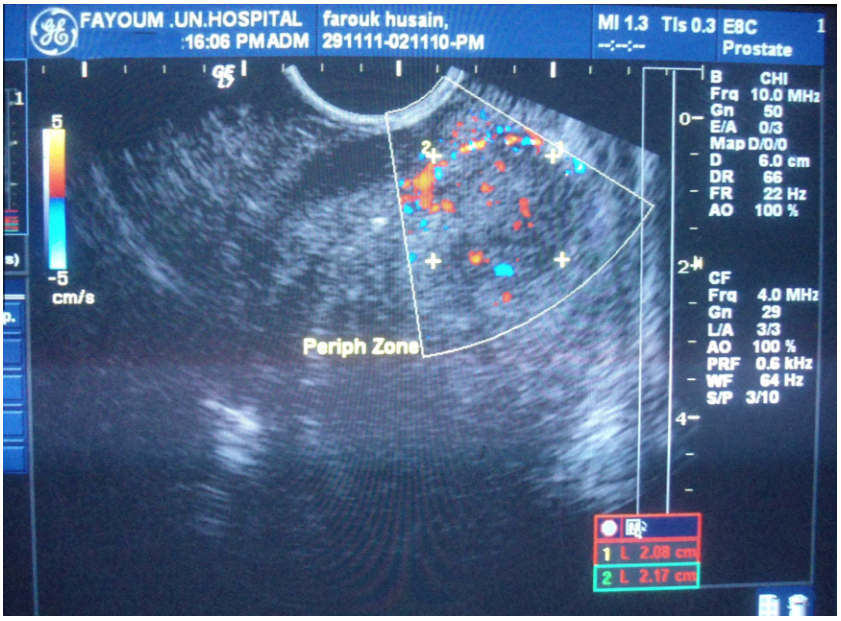

Figure 1 TRUS PDI showing a typical image from which the values were derived.

when patient had a strong desire to void; the patient was instructed to hold while the system was prepared for the voiding study. The patient was then instructed to try to void normally into the collecting cylinder of the uroflowmeter. This yields separate plots of intravesical, intra-abdominal and subtracted detrusor pressure (Pdet, obtained by automated subtraction of intra-abdominal pressure from intravesical pressure), as well as urinary flow rate, each as a function of time.

The Pdet at Q_{max} was recorded and the Abrams-Griffiths (AG) number was calculated (Pdet Q_{max} – $2Q_{max}$). In our study patients were divided into two groups, an obstructive group (AG number $\geqslant 40$) and an unobstructive group (AG number < 40).

The pRI was measured using PDI by one radiologist and to avoid inter-observer variability the radiologist was not aware of the results of the PFS for each patient. A Sonoline Elegra unit (Siemens Corp., Germany) with a convex, 7-MHz transrectal probe was used for TRUS and PDI. Images were obtained with the patient in the left lateral decubitus position. TRUS was used while the urinary bladder was empty to preclude compression of the prostatic vasculature. Prostate volume (PV) was calculated with the help of the in-built software, by measuring three dimensions of the prostate in transverse and longitudinal sections. Blood flow was measured in capsular arteries on the largest transverse section of prostate, followed by spectral waveform analysis. The pulse repetition frequency was adjusted to the point where aliasing did not occur. In most cases, a pulse repetition frequency of 1–3 kHz worked well. The Doppler frequency and sample volume were set at 5 MHz and 2 mm, respectively.

When pulsatile waveforms of a given Doppler spectrum became stable, the RI (maximum velocity – minimum velocity)/(maximum velocity) was measured. The RI was measured at four points in the transition zone (TZ) and the mean value was recorded using the in-built software (Fig. 1).

Descriptive statistics are presented as the mean (SD) and the results assessed using the Pearson correlation coefficient and Student's *t*-test, with $P < 0.05$ considered to indicate statistical significance.

Results

According to the AG number, 158 patients had BOO, with a mean (SD) AG number of 68.5 (12.90), and 180 had no obstruction, with an AG number of 26.3 (4.10). Table 1 gives the mean (SD) of the variables in the two groups.

The mean (SD) age was 65 (7.5) years in the obstructed and 55 (8.3) years in the unobstructed groups. The pRI and post-void residual urine volume

Table 1 Mean (SD) values of the variables in the two groups.

Variable	Obstructed	Unobstructed	*P*
Age (years)	65 (7.5)	55 (8.3)	0.001
IPSS	23.4 (4.3)	21 (2.4)	0.068
Q_{max} (mL/s)	6.9 (3.8)	13.1 (2.5)	0.001
PVR (mL)	145.6 (51.4)	49.5 (18.4)	0.001
TRUS PV (mL)	76.5 (24.6)	26.5 (8.7)	0.001
pRI	0.73 (0.04)	0.65 (0.05)	0.003

Table 2 The correlation between AG and pRI.

Group	Correlation coefficient, *r*	*P*
In all patients	0.639	0.001
In non obstructive patients	0.225	0.008
In obstructive patients	0.262	0.001

were significantly higher in the obstructed group. The Q_{max} was significantly lower in the obstructed group, at 6.9 vs. 13.1 mL/s ($P < 0.001$). The IPSS and PV values in the two groups are also shown in Table 1.

There was a significant correlation between the pRI and AG number in obstructed and unobstructed patients (Table 2).

The receiver operating characteristic (ROC) curve was used to determine the pRI threshold for predicting BOO due to BPH; a pRI of $\geqslant 0.71$ predicted BOO secondary to BPH, with 84.6% sensitivity, 78.4% specificity and 83.8% overall predictability (Fig. 2).

The obstructed patients (158) were managed medically and surgically (107 patients received an α-adrenoreceptor blocker, 48 had a TURP and three an open prostatectomy). After 6 months of BPH management, the pRI of the obstructive patients was re-evaluated, and had significantly decreased from 0.73 (0.04) before treatment to 0.69 (0.08) ($P < 0.05$; Table 3). The IPSS

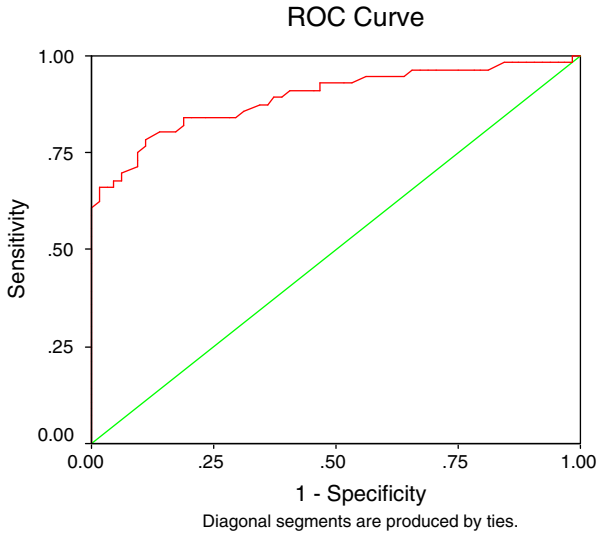

Figure 2 The ROC curve used to determine the threshold pRI values.

Table 3 Variables in the obstructed patients before and after management.

Mean (SD) variable	Before	After	*P*
IPSS	23.4 (4.3)	8.6 (3.6)	0.001
Q_{max} (mL/s)	6.9 (3.8)	16.4 (3.8)	0.001
PVR (mL)	145.6 (51.4)	51.5 (13.4)	0.001
pRI	0.73 (0.04)	0.69 (0.08)	0.001

and PVR also decreased, whereas the mean Q_{max} increased. Furthermore, 70% of the patients had a pRI of <0.71 after their management.

Discussion

LUTS are one of the most common problems in elderly men and BPH is one of the most frequent causes. The appropriate management of LUTS depends on determining the underlying mechanisms and whether patients with LUTS have BOO or not.

There is neither consensus nor clear practical guidelines to define the presence and degree of infravesical obstruction, other than values from PFS [14]. PFS are considered to be the reference standard tool for the diagnosis and categorisation of BOO, differentiating between men with voiding symptoms because of BOO and those with poor bladder contractility. They can also help to identify patients with high-pressure obstruction and normal flow rates. However, PFS are invasive, uncomfortable for the patient, time-consuming and expensive, especially in most developing countries.

Symptom scores are generally used to assess LUTS suggestive of BPH. Although the IPSS and AUA symptom score have a high correlation with the magnitude of urinary symptoms, and are useful in monitoring the effects of therapy for BOO, they lack specificity. Estimates of PVR and uroflowmetry, although being noninvasive tools for diagnosing voiding dysfunction, cannot be used to distinguish between BOO and impaired detrusor contractility [15–18]. These variables correlate mostly with the functional status of the lower urinary tract rather than mechanical obstruction itself [19]. Therefore, noninvasive measurements of the prostate which delineate a morpho-functional correlation would be useful in diagnosing LUTS secondary to benign prostate obstruction.

Previous studies reported that the pRI was higher in the capsular arteries of patients with BPH than in healthy prostatic vessels. It is possible that the enlargement of the TZ might compress, and thus cause mechanical obstruction, of the prostatic vessels. As the TZ is contained within a dense surgical capsule it seems likely that a high pressure accumulates within the TZ, which in patients with BPH might result in compression of the vessels supplying the TZ. Several reports suggested that the RI is higher in BPH due to a higher vascular resistance, which also seems to be related to vascular damage. Studies suggested an association between prostatic disease and the presence of vascular disorders such as coronary heart disease or diabetes mellitus [20].

Kojima et al. [11], using PDI in their preliminary report, found that the pRI was significantly higher in patients with BPH than in normal individuals (0.72 vs. 0.64, $P < 0.001$). In that study the elevated RI decreased significantly to a normal control level after

surgical treatment. In another study the same group of authors measured the pRI of 140 patients with LUTS. The pRI was significantly higher in patients with BPH, at 0.72 (0.06) ($P < 0.001$) than in those with a normal prostate, at 0.64 (0.04). In addition, they compared the pRI with values from PFS. They found a significant correlation between pRI and PFS values. The diagnostic accuracy in their study was 68% with 0.7 as the threshold for pRI [13]. Tsuru et al. [10] evaluated 214 patients with LUTS and showed that an increase of the RI of capsular arteries correlated with increases in the TZ index and the presumed circle area ratio in BPH. The increase in pRI was correlated with a lower IPSS and Q_{max}, but they did not correlate their findings with PFS. Several studies reported a correlation between RI and ultrasonographic prostatic variables such as PV, TZ volume, TZ index and presumed circle area ratio. Further, the RI was higher in patients with BPH than in those with a normal prostate. For subjective symptoms of BPH, an increased IPSS was correlated with an increased pRI value [12].

According to these studies, we evaluated the usefulness of the pRI determined by transrectal pulsed-wave spectral Doppler imaging in diagnosing BOO in patients with LUTS due to BPH. Using the ROC curve, a pRI of $\geqslant 0.71$ predicted BOO secondary to BPH, with 84.6% sensitivity, 78.4% specificity and 83.8% overall predictability. Also, the pRI of the obstructive group significantly ($P < 0.05$) decreased after management from the value before treatment, from 0.73 (0.04) to 0.69 (0.08). This decrease in RI can be explained by a decrease in intraprostatic pressure.

As TRUS is less invasive, cheaper and less time-consuming than PFS, and measures prostatic size, which is useful in planning management, the pRI should be considered when evaluating patients with LUTS.

BPH develops in a variety of gross configurations and periurethral sites, resulting in various anatomical designations, such as median lobe, median bar and lateral lobe hyperplasia. Many patients with LUTS have an enlarged median lobe with no enlargement of either lateral lobe, resulting in mechanical BOO. The pRI in such patients might not increase, as the two lateral lobes do not compress the prostatic capsule, which might give a 'false-negative' diagnosis of BOO in these patients. Further studies are required to determine the pRI in patients with large median lobes alone, and to assess whether pRI values in these patients can be used for the diagnosis of BOO.

In conclusion, the pRI can predict BOO, with high specificity and sensitivity. We believe that the pRI could be a useful variable for the diagnosis and follow-up of patients with BPH.

Conflict of interest

None declared.

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