

Evaluation of carotid intima-media thickness and cardiovascular risk factors in benign prostatic hyperplasia patients

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Abstract

Objective: To assess the carotid intima-media thickness and cardiovascular risk factors in patients with benign prostatic hyperplasia.

Material and methods: This was a prospective observational study conducted over two years in 100 patients who presented with benign prostatic hyperplasia (BPH). IPSS questionnaire was used to evaluate the symptomatology of BPH. Patients were examined for cardiovascular risk factors. All patients underwent echocardiographic examination for left ventricular function. A single experienced sonographer, blinded to all clinical information, assessed the common carotid artery.

Results: The patients mean age was 69.32 years. The mean BMI was 28.91 kg/m². Of the total mild symptom score cases, 75 % (N=6) cases had Grade I prostatomegaly (26 – 40 cc). 98.38 % (N=61) cases had Grade II prostatomegaly (41 – 60 cc), 93.33 % (N=28) cases had Grade III prostatomegaly (>60cc). A mean carotid intima-media thickness (IMT) of 0.56 mm was found in cases having mild IPSS scores (Score 1-7), 0.73 mm was found in cases having moderate IPSS score (Score 8-19), 0.92 mm was found in cases having severe IPSS scores (Score 20-35). Significant association was present between mean BMI, diabetes mellitus, hypertension, hyperlipidemia, smoking and mean Carotid IMT with IPSS as p-value was <0.05. Significant association was present between total cholesterol, HDL, LDL and prostate volume with p-value <0.0001.

Conclusion: This study found that prostatic tissue had a strong relationship with carotid IMT and cardiovascular risk factors in BPH patients. These data suggest a possible link between carotid IMT and cardiovascular risk factors and BPH.

Key words: benign prostate hyperplasia, carotid, diabetes mellitus, hypertension, intima-media thickness, sonography

Introduction

BPH (benign prostatic hyperplasia) is a significant health problem that affects more than half of all men in their sixties [1]. Despite the fact that BPH is common, the cause of the ailment is unknown. Other than age and sex steroid hormones, evidence suggests that BPH has modifiable risk factors. Diabetes, obesity, metabolic

syndrome, smoking, and high blood pressure are all risk factors for BPH [2-4].

The unregulated growth of connective tissue, smooth muscle, and glandular epithelium inside the prostatic transition zone is a histological feature of BPH [1]. Prostate tissue and the prostatic utricle have a similar embryologic genesis to the uterus and upper

vaginal canal. These are derived from the Müllerian duct's joined ends [5]. Female uterine leiomyomas are the most often seen benign tumours [6]. Uterine leiomyoma has been associated with cardiovascular risk factors such as diabetes, hypertension, obesity, and smoking [7-9]. Furthermore, a recent study found a link between carotid intima-media thickness (IMT) and the occurrence of uterine leiomyoma [10].

As a result, we were interested in studying carotid IMT and cardiovascular risk factors in BPH patients.

Objective: To assess the carotid intima-media thickness and cardiovascular risk factors in patients with benign prostatic hyperplasia.

Material and methods

This was a prospective observational study conducted over two years in 100 patients who had presented to the department of urology.

Inclusion criteria were all patients above 45 years who presented with benign prostatic hyperplasia associated symptoms to the department of urology and who were willing to give consent for this study. Exclusion criteria were patients below 45 years of age, Patients who were not willing to give consent for this study. The institutional ethics committee gave its approval to the project. After receiving written informed permission, patients were enrolled (correctly outlining the objectives, procedures, anticipated advantages, and any risks important for choosing to take part in the study).

The IPSS questionnaire was utilised to assess the BPH symptomatology. The IPSS is a quick and simple self-administered questionnaire used to evaluate BPH in patients. An eight-question written screening test called the IPSS questionnaire is used to quickly diagnose, monitor, and prescribe treatment for BPH. It is based on responses to one quality of life question and seven questions on urinary symptoms. The patient can select one of six options for each question about urinary symptoms, with the answers representing the increasing severity of that specific symptom. The solutions are given scores ranging from 0 to 5. Consequently, the final score might be between 0 and 35 (asymptomatic to very symptomatic). Patients are rated on their quality of life, from wonderful to terrible (0-6) [11]. Questions about urine symptoms include: incomplete emptying, frequency, intermittency, urgency, weak stream, straining, and nocturia. The IPSS classifies symptoms as follows: Mild (symptom score 1-7), Moderate (symptom score 8-19), and Severe (symptom score range 20-35).

Routine demographic information, personal history including smoking and alcohol addiction history, physical examination including digital rectal examination (DRE), haematological data such as complete blood profile, biochemical data such as renal function test and lipid profile, and transabdominal ultrasonography for the urinary system and carotid intima-media thickness of patients were recorded in an Excel sheet. The patients' cardiovascular risk factors were investigated. Age, BMI, diabetes mellitus (known diabetes treated with diet, drugs, or both, or a fasting serum glucose of more than 126 mg/dL), hypertension (known hypertension treated with antihypertensive drugs, two or more blood pressure recordings higher than 140/90 mm Hg), and hyperlipidemia with one or more factors (known case and treated with drugs, high fasting or non-fasting serum cholesterol concentrations) were all recorded. Current cigarette smoking was defined as actively smoking cigarettes within the past 12 months. The prostatic volume was measured by transabdominal ultrasonography (in

cc). For a cardiac status and left ventricular function evaluation, echocardiography was performed on all patients. All patients had B-mode ultrasonographic exams using a 7.0-MHz linear array transducer (HD 11XE; PHILIPS Ultrasound). One skilled sonographer who was unaware of any clinical details checked the common carotid artery. The previously described technique was used to quantify the intima-media thickness (IMT) of the common carotid artery [12]. A lengthy axis of the common carotid artery, 10 mm from its bifurcation, was examined. At the conclusion of diastole, the IMT was measured in the posterior wall from the B-mode screen to a location inside the 10 mm section close to the bifurcation of the common carotid artery. During carotid scanning, both the right and left common carotid arteries were examined. Additionally determined was the average carotid intima-media thickness (IMT). To compare the clinical characteristics, cardiovascular risk factors, and mean carotid IMT, patients were divided into three IPSS categories: mild symptom group (Score range 1-7), moderate symptom group (Score range 8-19), and severe symptom group (Score range 20-35). They were also divided into three prostatic volume grades: Grade I (26 cc - 40 cc), Grade II (41 cc - 60 cc), and Grade III (>60 cc) [13].

Statistical analysis

To compare clinical features, cardiovascular risk factors, and mean carotid IMT, patients were classified into three IPSS groups and three prostatic volume grades. The following variables were analysed statistically: age, hypertension, diabetes mellitus, smoking, total cholesterol levels, LDL levels, HDL levels, BMI and mean carotid IMT. Continuous values were reported as mean standard deviation, and categorical variables as a percentage. To test the association between the groups, the Pearson chi-square test was used. Using the ANOVA (Analysis of Variance) test, the mean difference between the three groups was examined. The statistical analysis was carried out utilising IBM SPSS version 24.0. (SPSS Inc, Chicago, IL). In all tests, statistical significance was defined as a P value 0.05.

Results

A total of 100 cases of BPH were included in this study from January 2019 to December 2020. They were evaluated based on International Prostate Symptom Score (IPSS) and prostate volume done by transabdominal ultrasonography. Cardiovascular risk factors and carotid IMT were also noted in all the cases.

As the IPSS symptom score increased from mild to severe, grades of BPH also increased. Of the total mild symptom score cases, 75 % (N=6) cases had Grade I BPH (26 – 40 cc). Of the total moderate symptom score cases, 98.38 % (N=61) cases had Grade II BPH (41 – 60 cc). Of the total severe symptom score cases, 93.33 % (N=28) cases had Grade III BPH (>60cc) (Table 1).

The patients mean age was 69.32 years. As the age of patients increased, IPSS grading also increased. The mean BMI was 28.91 kg/m². 33.3% (N=2) hypertension cases and 16.7% (N=1) hyperlipidemia cases were having a mild IPSS score (Score 1-7). A mean carotid IMT of 0.56 mm was found in cases having mild IPSS scores (Score 1-7). 69.2% (N=45) diabetes mellitus cases, 46.2% (N=30) hypertensive cases, 53.8% (N=35) hyperlipidemia cases and 64.6% (N=42) smokers had moderate IPSS (Score 8-19). Mean carotid IMT of 0.73 mm was found in cases having moderate IPSS score (Score 8-19). 89.7% (N=26) diabetes mellitus cases, 75.9% (N=22) hypertensive cases, 93.1% (N=27) hyperlipidemia cases and 82.8% (N=24) smokers

Table 1 Association between International Prostatic Symptom Scores subgroups and prostatic volume subgroups

International Prostate Symptom Score (IPSS)	Prostate Volume (in cc)				P value*
	Grade I (26-40 cc) (N=8)	Grade II (41-60cc) (N=62)	Grade III (>60 cc) (N=30)	Total cases (N= 100)	
Mild Symptom score Cases (%) (Score 1-7)	75% (N=6)	0 % (N=0)	0% (N=0)	6% (N=6)	P<0.0001
Moderate Symptom score Cases (%) (Score 8-19)	25% (N=2)	98.38% (N=61)	6.66% (N=2)	65% (N=65)	
Severe Symptom score Cases (%) (Score 20-35)	0% (N=0)	1.61% (N=1)	93.33% (N=28)	29% (N=29)	
Total cases	(N=8)	(N=62)	(N=30)	(N=100)	

*(<0.05 significant) Pearson ChiSquare Test

Table 2 Association between Demographic, Anthropometric, Cardiovascular parameters and International Prostatic Symptom Scores subgroups

Demographic, Anthropometric & Cardiovascular parameters	International Prostate Symptom Score (IPSS)				P value*
	Mild Symptom score cases (Score 1-7) (N=6)	Moderate Symptom score cases (Score 8-19) (N=65)	Severe Symptom score cases (Score 20-35) (N=29)	Total Cases (N=100)	
Mean Age (years)	67.17±7.223	68.94±8.968	70.62±6.444	69.32±8.201	0.531 (ANOVA)
Mean BMI (kg/m ²)	26.81 ± 0.854	27.79 ± 2.00	31.87 ± 3.18	28.91 ± 3.02	<0.0001 (ANOVA)
Diabetes Mellitus (%)	0.0% (N=0)	69.2% (N=45)	89.7% (N=26)	71.0% (N=71)	<0.0001 (CHI SQUAR E)
Hypertension (%)	33.3% (N=2)	46.2% (N=30)	75.9% (N=22)	54% (N=54)	0.016 (CHI SQUAR E)
Hyperlipidemia (%)	16.7% (N=1)	53.8% (N=35)	93.1% (N=27)	63.0% (N=63)	<0.0001 (CHI SQUAR E)
Smoking (%)	0.0% (N=0)	64.6% (N=42)	82.8% (N=24)	66.0% (N=66)	<0.0001 (CHI SQUAR E)
Mean Carotid IMT (mm)	0.56 ± 0.02	0.73 ± 0.06	0.92 ± 0.06	0.78 ± 0.11	<0.0001 (ANOVA)

*(<0.05significant) Pearson ChiSquare / ANOVA test

Table 3 Association between Lipid Profile parameter and prostatic volume subgroups

Lipid Profile	Prostate Volume (in cc)				P value*
	Grade I cases (26-40 cc) (N=8)	Grade II cases (41-60cc) (N=62)	Grade III cases (>60 cc) (N=30)	Total cases (N= 100)	
Mean Total Cholesterol (TC) (mg/dl)	171.25 ± 42.90	227.33 ± 55.23	336.73±4 6.22	255.67± 75.53	<0.0001
Mean High Density Lipoproteins(HDL) (mg/dl)	43.75 ± 5.84	37.58 ± 6.66	26.97 ± 4.45	34.89 ± 8.09	<0.0001
Mean Low Density Lipoproteins (LDL) (mg/dl)	90.38 ± 26.20	122.00 ± 39.66	192.77 ± 36.63	140.70 ± 51.53	<0.0001

*(<0.05 significant) (ANOVA test)

had severe IPSS (Score 20-35). A mean carotid IMT of 0.92 mm was found in cases having severe IPSS scores (Score 20-35). Overall for 100 cases, the mean carotid IMT was 0.78 mm (Table 2).

A significant association was present between mean BMI, diabetes mellitus, hypertension, hyperlipidemia, smoking and mean Carotid IMT with IPSS as p-value was <0.05 (Table 2).

As age increased (from 67.88 years to 70.93 years), grades of BPH increased (from Grade I to Grade III) and hence, the prostate volume also increased.

As mean BMI increased (from 25.89 kg/m² to 32.15 kg/m²), grades of BPH increased (from Grade I to Grade III) and hence, the prostate volume also increased.

Of the total Grade II BPH cases, 71% (N=44) had diabetes mellitus (DM). Of the total Grade III BPH cases, 90% (N=27) had diabetes mellitus. A significant association was present between DM and prostate volume.

Of all the Grade I, Grade II, Grade III BPH cases, 25% (N=2) cases, 46.8% (N=29) cases and 76.7% (N=23) cases had hypertension respectively. A significant association was present between hypertension and prostate volume.

The mean total cholesterol level was 255.67 mg/dl. The mean HDL level was 34.89 mg/dl. The mean LDL level was 140.70 mg/dl. A significant association was present between total cholesterol, HDL, LDL and prostate volume (Table 3).

Out of the total 100 cases, 66% (N=66) were smokers. A significant association was present between smoking and prostate volume.

The mean carotid IMT was 0.78 mm. Grade I, Grade II and Grade III prostate cases had 0.56mm, 0.73mm, and 0.92mm mean carotid IMT respectively. A significant association was present between mean Carotid IMT and prostate volume.

Discussion

BPH and cardiovascular diseases commonly occur in elderly men. The coexistence of these two diseases in the same patient is not by chance alone but indicates a shared common pathophysiologic process [14]. The exact mechanism of atherosclerosis is still unknown despite the great understanding of its pathophysiology. Several studies have suggested a link between atherosclerosis and cardiovascular risk factors and prostatic hyperplasia [15-17].

Studies have proven that baseline prostate size can be considered a strong indicator of BPH progression. Also, prostate volume as a risk factor for acute urinary retention (AUR) has also been proven [18].

The role of IPSS to assess symptom severity of LUTS in BPH patients is already proven as the majority of men with BPH present with LUTS [11,19]. In the present study, 100 BPH patients were assessed for symptoms with IPSS grading. Awaisu M et al. in a prospective correlational study in 290 patients of BPH demonstrated that as the grades of IPSS increase, prostate volume and the severity of BPH increases [20]. In his study, there was a strong positive association between prostate volume and IPSS ($r=0.179$, $p=0.002$), and the majority of the patients reported moderate symptoms on the IPSS (55%) with a mean IPSS value of 16.41 ± 7.43 [20]. In the present study also, 65% (N=65) of BPH cases had moderate symptom scores on IPSS. Also, a similar significant correlation was present between IPSS score and prostate volume. As IPSS score increased, grades of BPH also increased, hence the severity of BPH also increased. In contrast to patients in the normal group, Yelsel K et al. found a strong correlation between grades on the IPSS and overweight and obesity ($P=0.001$). In comparison to the overweight group, the obese group's IPSS levels were considerably greater ($P=0.010$) [21].

In the present study also, as the mean BMI increased, grades of IPSS increased. There was a positive correlation between IPSS and body mass index (BMI) as the p-value was <0.0001 . In this study, mean BMI is 26.81 kg/m^2 , 27.79 kg/m^2 and 31.87 kg/m^2 in mild, moderate and severe IPSS score cases respectively, thus corresponding with the positive association of overweight and obesity with higher grades of IPSS. Ponholzer A et al. investigated the association between vascular risk factors like diabetes mellitus, hypertension, hyperlipidemia, nicotine use and LUTS in both sexes. In men, the IPSS score was identical in those with no vascular risk factor and one

vascular risk factor but increased to in those with two or more risk factors ($p=0.01$) [22]. A substantial positive link between a number of cardiovascular risk variables, including smoking, hypertension, hyperlipidemia, diabetes mellitus, and IPSS scores, was discovered in the current study as well. In 2014, Lee et al. examined in 799 men the association between carotid artery plaque and the international prostate symptom score (IPSS). They found a significant ($P=0.002$) correlation between the maximal intima-media thickness (max IMT) and the IPSS voiding subscore. They noticed a strong correlation between the severity of the voiding score and plaque severity ($P=0.003$) [23]. In the current study also, the mean carotid IMT is different between the three IPSS groups and is highest ($0.92 \text{ mm} \pm 0.06 \text{ mm}$) for a severely symptomatic group with an IPSS score between 20-35. Previous studies have demonstrated as grades of IPSS increase, prostate volume and the severity of BPH increase [20]. In this study also, maximum mean carotid IMT of 0.92 mm was present in severely symptomatic IPSS group and higher grades of IPSS was associated with higher grades of BPH, thus proving that as carotid IMT increases, grades of BPH and severity of BPH increases. The association between carotid IMT and IPSS was significant ($P\text{-value}<0.0001$).

Liu CC et al. in 2007 demonstrated a significant positive correlation of age with BPH especially with prostate volume ($r=0.309$, $P<0.001$) [24]. In the present study also, as mean age increased, grades of BPH increased corresponding to increased prostate volume but the association was not significant as $p=0.424$. This may be due to the fewer cases in the present study.

In 2008, YD Kim et al. examined the relationships between anthropometric and metabolic variables and prostate volume. After doing a bivariate study, the researchers discovered a positive link between prostate volume and body weight, height, and body mass index (BMI) [25]. There is a role of obesity as an etiological factor for BPH due to its influence on metabolic factors and obesity-related endocrine changes. The development of benign prostatic hyperplasia and the severity of urinary obstructive symptoms are both known to be influenced by abdominal obesity, which raises the oestrogen to androgen ratio and may enhance sympathetic nerve activity. BPH risk was shown to be highly correlated with body mass index (BMI), a measure of general obesity, and waist to hip ratio (WHR), a marker of abdominal obesity [26]. According to research by Hammarsten et al., obesity is a risk factor for developing BPH since it is positively correlated with measures of obesity like BMI [12,27]. In the present study also, higher BMI values were associated with increasing grades of BPH, thus corresponding to an increase in the severity of BPH which was significantly correlated ($p<0.0001$).

Diabetes and BPH become more common as people become older. Several research have revealed a link between BPH and diabetes [28]. Hammarsten J et al. from Sweden showed in a series of early cross-sectional investigations that diabetes was strongly related with an enlarged prostate size consistent with BPH. Men with diabetes had a bigger prostate gland than men without diabetes among LUTS patients (78 mL vs. 45 mL , respectively; $P=0.006$), according to the researchers [12,16,27,29]. Men with diabetes had a two-fold increased risk of having an enlarged prostate (40 mL), as determined by MRI, whereas men with excessive fasting glucose had a three-fold increased risk [30]. Nandeesh H also found a positive association with diabetes mellitus and BPH [28]. Even though process by which diabetes causes BPH is unknown, previous research has revealed that vascular damage caused by type 2 diabetes can exacerbate BPH. It has been suggested that detrusor

hypoxia can play a role in the aetiology of BPH. By affecting angiogenesis, hypoxia can hasten the growth of the prostatic gland. The transcription factor hypoxia-inducible factor 1 (HIF-1) as well as growth factors like vascular endothelial growth factor (VEGF), fibroblast growth factors 2 and 7 (FGF-2 and FGF-7), transforming growth factor- β (TGF- β), and cytokines like IL-8 can all be expressed more when there is hypoxia. By gradually subjecting the prostate stroma to greater growth factor levels, chronic hypoxia may accelerate prostate development and contribute to the pathogenesis of BPH. As a result, BPH treatment may be affected if tissue hypoxia is reduced by boosting oxygen supply with proper diabetes control [28]. In this study also, of 100 patients of BPH, 71 patients (71%) had statistically significant diabetes mellitus, thus correlating with findings of other studies and signifying the association of diabetes with BPH. Men's age-related illnesses including BPH and hypertension are quite common and put a significant strain on the world's healthcare systems. Twenty to thirty percent of individuals with benign prostatic hyperplasia experience arterial hypertension. Recent epidemiological studies have demonstrated that men with hypertension are more likely than age-matched controls to receive surgical intervention for BPH-related irritative voiding symptoms. According to this research, noradrenergic nerves, which control vascular tone, also contribute to the functional aspect of bladder outlet blockage brought on by BPH [28]. Nandeesh H also concluded hypertension as a risk factor for BPH [28]. Guo LJ et al. discovered hypertension in 128 BPH patients (30.3 percent) in a retrospective investigation of 423 BPH cases, and substantial positive associations were established among prostate volume and duration of hypertension [31]. It has been found that the time of incidence of BPH and the requirement for surgical intervention was sooner with in hypertensive group than in the normal blood pressure group [31]. Furthermore, the prevalence of urine retention and haematuria was shown to be greater in BPH patients linked with hypertension, showing that primary hypertension might influence the onset and clinically progression of BPH [31]. The sympathetic nervous system's activity has been the common pathological mechanism that connects BPH with hypertension. A recent study by Achari R et al. backs up this theory [32]. In the current study also, of the total grade III and grade II BPH cases, 76.7% cases and 46.8% cases had hypertension respectively, thus signifying hypertensive patients have more prostate volume corresponding to severe grades of BPH. Of the total BPH patients, 54% of patients had hypertension, which was significantly correlated with a p-value of 0.006, thus corroborating with other studies.

Hyperlipidemia was found in 50.2 percent of BPH patients in a Chinese retrospective investigation by Li PJ et al [31]. In several studies, Hammarsten J et al. hypothesised that dyslipidemia, particularly low levels of HDL cholesterol, is in fact a risk factor for the onset of BPH [12,27]. In comparison to males with slow-growing BPH, they discovered decreased HDL cholesterol levels in men with fast-growing BPH ($p=0.021$). The rate of yearly BPH development was adversely linked with HDL cholesterol ($rs=-0.22$; $p=0.001$) [27]. When compared to controls, BPH patients had considerably higher total cholesterol, lower HDL cholesterol, and higher LDL cholesterol, according to Nandeesh H et al [33]. The inhibitory effect of testosterone on HDL cholesterol levels helps to explain low HDL cholesterol levels in BPH. Low amounts of HDL cholesterol can increase LDL cholesterol synthesis, modify LDL cholesterol by oxidation, and activate Protein C kinase, which leads to aberrant prostate cell growth and BPH [28]. In the present study also, patients with BPH had higher total cholesterol 255.67 mg/dl, low HDL

cholesterol 34.89 mg/dl and high LDL cholesterol 140.70 mg/dl as found in other studies, thus proving lipids role in BPH (Table 3).

According to research by EA Platz et al., current cigarette smoking is positively associated with BPH [34]. In the present study also, of the total grade III and grade II BPH cases, 83.3% cases and 66.1% cases were smokers respectively. The association between smoking and prostate volume was found to be significant. Thus, smoking was associated with a severe grade of BPH corresponding to the severity of BPH.

Very few studies have evaluated prostatic hyperplasia with carotid IMT. Erbay et al. objectively assessed carotid IMT in BPH patients. The study comprised 123 participants who were examined for the existence of BPH with concomitant symptoms. They found that in BPH patients, carotid IMT was significantly associated with prostatic volume (beta coefficient: 0.628; confidence interval: 37.02–60.1; $P=.001$) [35]. According to Lee et al., there is a strong correlation between the IPSS voiding subscore and the maximal carotid IMT ($p=0.002$) and the severity of the plaque as it grows ($p=0.003$). They also discovered that, after correcting for age and metabolic syndrome factors, there is a greater chance of having a higher IPSS voiding subscore as plaque severity increases [23]. Previous studies have already established the association of higher IPSS grades with prostate volume and severity of BPH [20]. Hence, there is a higher likelihood of association of carotid IMT and plaque size to prostate volume and severity of BPH. In the present study, mean Carotid IMT is different in three groups of prostate volume, showing an increasing trend in ascending order, as lowest carotid IMT ($0.55\text{mm}\pm0.03\text{mm}$) for Grade I BPH cases to highest carotid IMT ($0.92\text{mm}\pm0.05\text{mm}$) for Grade III BPH cases. The mean carotid IMT was $0.78\text{mm}\pm0.11\text{mm}$ for total cases of BPH. There was a significant correlation between mean carotid IMT and prostate volume ($p\text{-value}<0.05$). Although a link between atherosclerosis and BPH has previously been demonstrated, the specific mechanism behind the association between BPH and carotid IMT remains unclear and requires clarification. Previous studies have discovered a connection between the onset of BPH and non-insulin-dependent diabetes mellitus, hypertension, and dyslipidemia, suggesting that systemic as opposed to local factors may be to blame [35,36]. Hypoxia, neovascularization, oxidative stress, and eventual vascular damage are further theories put out to explain the connection between atherosclerosis and BPH. These factors lead to a decrease in blood flow to the prostatic tissue [15,37]. According to Berger et al., people with severe vascular disease have much worse prostatic tissue perfusion than healthy people. Persistent ischemia brought on by vascular damage may aid in the development of BPH [15]. Carotid IMT was utilised to assess the prostate volume and BPH because there is a positive correlation between atherosclerosis and BPH and because it is a proxy marker for atherosclerosis. These two associated histologic abnormalities (carotid IMT and BPH) share the same risk factors for smooth muscle proliferation, which are increasing age and an unspecific genetic make-up. Therefore, the discovery of a relationship between BPH and carotid IMT in terms of smooth muscle proliferation was not unexpected. Unexpectedly, a connection between uterine leiomyoma, carotid IMT, and cardiovascular risk factors has been established [10]. Similar pathophysiologic pathways are thought to be involved in atherosclerosis, BPH, and uterine leiomyoma since the prostatic and uterine tissues are both descended from the Müllerian duct. These putative pathophysiologic mechanisms lead to the discovery that smooth muscle cells from atherosclerotic plaques and uterine leiomyomas have monoclonal origins and

perform similarly in cell culture [38]. Age-related prevalences of diseases including atherosclerosis, prostatic hyperplasia, and uterine leiomyomas support the idea that pathophysiologic mechanisms connected to smooth muscle cell proliferation may exist. Carotid IMT is a well-recognized sonographic marker for early atherosclerosis, and intima-media complex thickening indicates generalised atherosclerosis. Cardiovascular risk factors and BPH are associated with atherosclerosis [39]. BPH is linked to prostate volume and the IPSS. IPSS and prostate volume are also linked with carotid IMT. As a result, it suggests that there is a direct association between carotid IMT and cardiovascular risk factors and BPH.

The study's limitation was the small number of patients, as more patients would result in a more accurate evaluation and correct conclusion. Another restriction was that, despite the fact that carotid IMT was assessed by a single observer, some differences in measurement might occur by the same observer, altering the value of carotid IMT. Few studies have linked

carotid IMT to BPH, and additional study is needed in the future to strongly indicate and conclude carotid IMT as a marker of BPH.

Conclusion

This research found a substantial link between prostatic tissue and carotid intima-media thickness and cardiovascular risk factors in BPH patients. These findings point to a probable relationship between carotid intima-media thickness, cardiovascular risk factors, and BPH.

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