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Original Article

Correlation between Benign Prostatic Hyperplasia and Coronary Artery Disease

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Abstract

Introduction: Coronary artery disease (CAD) and benign prostatic hyperplasia (BPH) both are considered as multifactorial process. Smooth muscle proliferation is an important and possibly an androgen-dependent step in the development of atherosclerosis and BPH. Insulin such as growth factor, inflammation, and metabolic syndrome plays a central role in pathogenesis of BPH and CAD.

Purpose: The purpose of this study is to study occurrence of CAD among subjects with BPH, occurrence of BPH among subjects with CAD, and correlation between them.

Materials and Methods: A total of 150,75 subjects with BPH (lower urinary tract symptoms with ultrasonography [USG] evidence/raised age-specific serum prostate-specific antigen [PSA]) and 75 subjects without BPH were included. PSA was measured by chemiluminescence method, prostate volume with transabdominal USG. CAD was diagnosed with the help of electrocardiogram, 2D-ECHO, Tread Mill Test (TMT), and documented history of angioplasty.

Results: The occurrence of CAD among 75 subjects with BPH (30.66%) was significantly higher than 75 subjects without BPH (12%) ($P < 0.05$) and even after excluding subjects with risk factor/factors (22.5% versus 7.4%: $P < 0.05$, respectively). Among subjects with CAD (32/150), occurrence of BPH was 27.81% more as compared to subjects without CAD (118/150). Mean serum PSA level and mean prostatic volume were also significantly higher in subjects with CAD as compared to subjects without CAD.

Conclusion: The occurrence of CAD was found to be significantly higher among subjects with BPH (even after excluding subjects with risk factors). Occurrence of BPH was also significantly higher among subjects with CAD along with mean serum PSA and mean prostatic volume. Thus, a significant correlation can exist between CAD and BPH.

Key words: Benign prostatic hyperplasia, Coronary artery disease, Prostate-specific antigen, Tread mill test

INTRODUCTION

Ischemic heart disease (IHD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of myocardium and is a consequence of myocardial oxygen supply and demand mismatch.[1]

Coronary artery disease (CAD) is a term used for atherosclerotic changes in coronary artery itself. There can be numerous causes of ischemic heart disease such as cardiomyopathy causing relative ischemia, embolus in coronary artery from anywhere in body, and arteritis. However, the most common cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery (or arteries) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery.[1] Hence, for practical purposes, the terms CAD and IHD can be used interchangeably.

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The various modifiable and non-modifiable risk factors for atherosclerosis include age, diabetes, male gender, smoking, hypertension, dyslipidemia, obesity, family history, chronic inflammation, hyperhomocysteinemia, renal diseases, and increased calcium score.

It is well documented that the frequency of CAD is influenced by gender as well as hormonal status. The risk

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of CAD is significantly lower in premenopausal women as compare to men and postmenopausal women.[2]

Atherosclerosis, the underlying pathology responsible for CAD, is an inflammatory disease. Recent observations suggest that the atherosclerotic process is characterized by a low-grade inflammation altering the endothelium of the coronary arteries and is associated with an increase in levels of markers of inflammation such as acute-phase proteins and cytokines. Cumulative evidence indicates that inflammation, at both focal and systemic levels, plays a key role in destabilization and rupture of atherosclerotic plaques, leading to acute cardiovascular events.[3]

Benign prostatic hyperplasia (BPH), also called benign enlargement of the prostate (BEP or BPE), is a non-cancerous increase in size of the prostate. BPH involves hyperplasia of prostatic stromal and epithelial cells, resulting in the formation of large, fairly discrete nodules in the transition zone of the prostate.[4]

It appears to be androgen-dependent process, as castrated boys do not develop BPH when they age.[5]

The modifiable and non-modifiable risk factors for BPH include age, genetics, hormones, metabolic syndrome, obesity, diabetes, and chronic inflammation.[6]

BPH may itself may be considered as a form of asymptomatic inflammatory prostatitis, whose pathogenesis may be triggered by a multitude of factors and pathways. The release of prostatic self-antigens following tissue damage may sensitize the immune system and start autoimmune responses.[7]

Smooth muscle proliferation is an important and possibly an androgen-dependent step in the development of atherosclerosis and BPH. Insulin-like growth factor (IGF-1) also plays a common role in pathology of BPH and CAD.

In the absence of other prostatic pathology, serum prostate-specific antigen (PSA) levels correlate positively with prostatic volume of BPH, as determined by transrectal ultrasound, in a log-linear fashion.

Since serum PSA is an objective measurement and readily obtained, it was used as a BPH surrogate. This technique allows us to assess a large number of men in non-invasive manner.[8,9]

PSA is a serine protease that cleaves insulin-like growth factor-binding protein-3 (IGFBP3), thereby decreasing its affinity for IGF-I. Dissociation of the IGF-I - IGFBP3 complex renders IGF-I available to bind to its receptor and stimulates cellular proliferation.[10]

The significantly lower color pixel density and higher resistive index in color Doppler ultrasonography (USG) of transition zone of prostate in patients with vascular disease than in healthy subjects support the hypothesis that an age-related impairment of blood supply to the prostate may have a key role in the development of BPH.[11]

Nocturia is one of the common lower urinary tract symptoms (LUTS), which causes sleep disturbances, daytime fatigue, lower level of general well-being, repeated awakening, and voiding episodes. This increases the sympathetic activity and may disturb blood pressure rhythmicity which in turn may lead to high cardiovascular disease morbidity such as angina pectoris and myocardial infarction.[12]

MATERIALS AND METHODS

A observational study was carried out in the Department of Medicine, NSCB Medical College, Jabalpur, during March 2015 to August 2016. Informed consent was obtained from all the subjects. Approval from the Institutional Ethical Committee was taken before conducting the study.

Study population: A total of 150 subjects of different age groups and male sex were included in the study. The subjects comprised of two groups: 75 subjects with BPH and 75 subjects without evidence of BPH.

A detailed history of each subject was taken which includes symptoms of CAD-anginal chest pain, shortness of breath, chest pain on exertion, symptoms of BPH/LUTS (difficulty in micturition, urine urgency, dribbling of urine, nocturia), urinary tract infection (UTI) (fever with chills and rigor, burning micturition), history of smoking, past history of diabetes, past history of CAD, hypertension, and drug history. Thorough physical examination including body mass index (BMI), blood pressure (BP) measurement and all other relevant systemic examination was carried out.

Subjects with evidence of either raised age-specific serum PSA value or USG volume of prostate >30 ml with LUTS were considered as BPH (n = 75) and subjects with no evidence of BPH were included (with the help of history, transabdominal ultrasound/serum PSA) for comparison (n = 75). Further groups for the study were created from these two groups.

Following points were considered for BPH in subjects: LUTS with either volume of prostate >30 cc or raised

age-specific serum PSA. PSA is a useful tool in clinical setting. It can be accurately measured with a simple clinical test, and this technique allows us to assess large number of subjects in an objectively yet non-invasive fashion. In this study, we have excluded the following subjects with serum PSA > 10 ng/ml as there are more chances to have prostatic cancer; subjects who have undergone any transurethral procedures, prostatic biopsy or had been exposed to any drug known to change the value of serum PSA and subjects having UTI were also excluded with the help of history and urine routine microscopy.

Volume of prostate was measured by transabdominal USG not by transrectal USG (TRUS) as TRUS is an invasive procedure and many subjects may not give consent for it, and digital rectal examination (DRE) not included in clinical examination as it might raise PSA and could have incurred false-positive result.

Following points were considered for CAD in subjects:

1. CAD - Acute coronary syndrome (STEMI/NSTEMI/unstable angina):
    A. Symptoms of ischemia along with detection of a rise and/or fall of cardiac biomarker values with at least one value above the upper reference limit were considered as acute MI.
    B. Symptoms of ischemia along with one of the following in the absence of raised cardiac biomarkers:
        a. New or presumed new significant ST-segment-T wave (ST-T) changes or new left bundle branch block.
        b. Development of pathological Q-waves in the electrocardiogram (ECG).
        c. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
2. Evidence of prior myocardial infarction (any one of the following)
    A. Pathological Q-waves with or without symptoms in the absence of non-ischemic causes.
    B. Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischemic cause.
3. Positive TMT for inducible ischemia irrespective of history of anginal chest pain.
4. Documented history of coronary angioplasty or coronary artery bypass grafting or significant coronary artery stenosis (>50%) on coronary angiography.

Inclusion criteria - subjects with BPH and/or subjects with CAD were included in the study. Age group 35-80 years; subjects having history of MI, H/O coronary angioplasty, IHD; subjects willing to give informed consent for participation in this study; subjects having BPH having history of established CAD/proved on provocative test (TMT)/finding in ECG/2D-ECHO; subjects willing to give informed consent for participation in this study were included in the study.

Exclusion criteria - subjects having serum PSA level >10 ng/ml, established prostatic cancer, urinary infection, history of transurethral resection of the prostate/any transurethral procedure, and history of taking any alpha blocker drug for hypertension were excluded from the study.

After having been selected for the study, each subject underwent the following procedure: Detailed history, careful clinical examination, ECG, blood sugar (fasting blood sugar/post-prandial blood sugar/random blood sugar) or HbA1C ≥ 6.5%, 12 h fasting lipid analysis, 2D-ECHO, treadmill test (ECG stress test)/coronary angiography, transabdominal USG for prostate volume and post-void residual volume of urine, age-specific serum PSA, urine routine, and microscopy.

According to NCEP-ATP III guidelines, dyslipidemia was considered when serum total cholesterol ≥200 mg/dl or/and low-density lipoprotein cholesterol ≥ 130 mg/dl or/and high-density lipoprotein cholesterol ≤40 mg/dl or/and serum triglyceride ≥ 150 mg/dl.

Obesity BMI ≥ 25 kg/m^2, hypertension was defined as systolic BP more than 140 mm Hg and/or diastolic BP more than 90 mm Hg.

Following tools were used: USG machine (Log iQ - 3 expert) model no.: AY-15CUK; ECG machine: Magic R; 2D-ECHO machine - Philips HD-7 XE; ECG machine - Schiller CS-200; Serum PSA - chemiluminescence.

Methods

2D-ECHO was carried out for some subjects who were not affordable for TMT. In 2D-ECHO, regional wall motion abnormality without any other cause of ischemia was considered as a sign of CAD. TMT and ECHO were reported by expert cardiologists.

In this study, first, the occurrence of CAD in age-matched subjects with BPH and without BPH was calculated according to above criteria. After exclusion of risk factors in both groups, the same was calculated to find a direct correlation.

The occurrence of BPH, mean serum PSA, and mean prostatic volume in subjects with CAD and subjects without CAD were calculated from the study group in same

way but by different statistical method because of unequal distribution of subjects in this group and consequently correlation calculated accordingly.

RESULTS

A total number of 150 male subjects was included in the study. Mean age of the whole study group was 50.46 years. Out of 150 subjects, two groups were created each comprising 75 subjects, one with BPH, and one without BPH. Mean age of subjects with BPH and without BPH was 51.01 ± 7.63 years and 49.92 ± 7.64 years, respectively, and the difference was not statistically significant ($P > 0.05$). Out of 75 subjects with BPH, 23 had CAD (30.66%), and out of 75 subjects without BPH, 9 had CAD (12%). The difference of occurrence of CAD between both these groups was statistically significant ($P < 0.05$), but after taking risk factor/factors (obesity, diabetes, hypertension, smoking, and dyslipidemia) into consideration, (46.66% vs. 28%) both groups had statistically significant difference ($P < 0.05$) (Table 1).

As the presence of risk factors might have affected the occurrence of CAD because the difference of presence of risk factors was significant, so further study groups were created after exclusion of subjects with risk factor/factors. After exclusion of subjects with risk factor/factors, subjects with BPH and without BPH were 40 and 54, respectively, mean age of the two groups was 51.18 ± 8.30 years and 50.44 ± 7.75 years, respectively, and the difference between mean age was not statistically significant ($P > 0.05$). Out of 40 subjects with BPH, CAD was present in 9 subjects (22.5%) as compared to a group of 54 subjects without BPH, 4 had CAD (7.4%). The difference of occurrence of CAD between these age and risk factor matched group was statistically significant ($P < 0.05$).

Out of 150 subjects, CAD was present in 32 subjects. Mean age of subjects with CAD was 51.03 ± 5.78 years and that of subjects without CAD was 50.31 ± 8.07 years, and the difference between mean age of both these age groups was not statistically significant ($P > 0.05$). Among subjects with CAD, risk factors were present in 14/32 (43.75%), and among subjects without CAD, risk factors were present in 56/118 (47.45%), and the difference was not statistically significant ($P > 0.05$). The occurrence of BPH among subjects with CAD was 23/32 and among subjects without CAD was 52/118, which was 27.81% higher among subjects with CAD ($P < 0.05$). Mean serum PSA among subjects with CAD and among subjects without CAD was 2.96 ± 0.97 ng/ml and 1.80 ± 1.09 ng/ml, respectively, and the difference was statistically significant ($P < 0.05$). Mean prostatic volume among subjects with CAD and subjects without CAD was 34.72 ± 4.67 cc and 26.90 ± 5.72 cc, respectively, and the difference was statistically significant ($P < 0.05$).

Table 1: Comparison of parameters between subjects with BPH and subjects without BPH

Parameters	Subjects with BPH (*n*=40)	Subjects without BPH (*n*=54)	*P* value
Mean age	51.01±7.63	49.92±7.64	>0.05
Presence of CAD	23/75 (30.66%)	9/75 (12%)	<0.05
Risk factor	35/75 (46.66%)	21/75 (28%)	<0.05

BPH: Benign prostatic hyperplasia, CAD: Coronary artery disease

To know correlation between CAD with serum PSA and prostate volume, group of subjects without BPH was studied. Out of 75 subjects without BPH, there were no risk factors in 54 subjects. Out of these 54 subjects, CAD was present in 4 subjects and was absent in 50 subjects. Mean age of subjects with CAD and subjects without CAD was 48.00 ± 0.81 years and 50.64 ± 8.02 years, respectively, and the difference was not statistically significant ($P > 0.05$). Mean PSA of subjects with CAD and those of subjects without CAD was 1.94 ± 0.52 ng/ml and 1.44 ± 0.96 ng/ml, respectively, and the difference was not statistically significant ($P > 0.05$), but difference between mean prostatic volume was statistically significant (28 ± 1.63 cc versus 22.08 ± 1.61 cc; $P < 0.05$).

DISCUSSION

Among subjects with BPH, CAD was present in 23 out of 75 subjects which was 30.66%. Among subjects without BPH, CAD was present in 9 out of 75 which was 12%, and occurrence was 18.66% more in subjects with BPH than in subjects without BPH. This difference was statistically significant ($\chi^2 = 7.79$; $P < 0.05$) as shown in Table 1.

In a study done by Bruno and Summers on 50 patients with mean age of 73.53 years, there were 6 diabetic patients in this series (6/50). Glands from diabetic patients were significantly larger. Out of the 50 patients, in his study, 27 (54%) had died (including) 12 of myocardial infarction and 3 of arteriosclerotic disease. He found that 30% of the patients (15/50) who died of ischemic heart disease had larger glands (97.3 ± 27.85 g) than the remaining patients in this series (71.24 ± 8.87 g). This was statistically significant ($P < 0.001$).[13]

In study done by Ozden *et al.*, it was found that median annual total prostate (TP) growth rate and median annual transition zone (TZ) growth rate were significantly higher in patients with metabolic syndrome as compared to patients without metabolic syndrome.[14]

Obesity, hypertension, dyslipidemia, smoking, and diabetes are traditional risk factors for CAD, and these factors affect the occurrence of CAD.

Hence, even after exclusion of subjects with even a single risk factor, the difference between occurrence was still significant as shown in Table 2.

Similar findings were also observed in a study done by Weisman *et al.*, on age group of 65-80 years (mean age of cases 72.2 ± 4.2; control 71.6 ± 5.1) in age- and risk factor-matched subjects where they found frequency of CAD as 29% and 9% in cases and control, respectively, which was statistically significant.[15]

In age- and risk factor-matched subjects, it was found that the occurrence of BPH, mean prostatic volume, and mean serum PSA level was more in those with CAD (n = 32) than those without CAD (n = 118) as shown in Table 3.

In study done by Berger *et al.*, on 23 CAD subjects and 31 normal controls, it was found that prostatic volume was higher (39 cc) in subjects with CAD as compared to controls (24 cc) although it was not statistically significant.[11]

Hypoxia/microvascular ischemia-induced smooth muscle proliferation may be a pathogenesis behind this. Hypoxia induces expression not only of hypoxia inducible factor 1 but also of angiogenic growth factors such as vascular endothelial growth factor (VEGF), fibroblast growth factors-2 and -7, and transforming growth factor beta, as well as cytokines such as interleukin-8. Long-term exposure of the prostatic stroma to increased growth factor levels secondary to chronic hypoxia might cause stimulation of stromal growth over the years, and thus, contribute to the pathogenesis of BPH. This concept is supported by the finding that patients with peripheral arterial occlusive disease and diabetics have significantly larger prostates than controls ($P < 0.001$). Furthermore, chronic ischemia has recently been shown to induce marked hyperplasia of the ventral prostate in the rat, through VEGF upregulation.[16]

Table 2: Comparison of parameters between subjects with BPH and subjects without BPH (after excluding subjects with risk factor/factors)

Parameters	Subjects with BPH (n=40)	Subjects without BPH (n=54)	P value
Mean age	51.18±8.30 years	50.44±7.75 years	>0.05
Presence of CAD	9/40 (22.5%)	4/54 (7.4%)	<0.05

BPH: Benign prostatic hyperplasia, CAD: Coronary artery disease

Table 3: Comparison of parameters between subjects with CAD and subjects without CAD

Parameters	Subjects with CAD (n=32)	Subjects without CAD (n=118)	P value
Mean age	51.03±5.78 years	50.31±8.07 years	>0.05
Risk factors	14/32 (43.75%)	56/118 (47.45%)	>0.05
Presence of BPH	23/32 (71.87%)	52/118 (44.06%)	<0.05
Mean PSA	2.96±0.97 ng/ml	1.80±1.09 ng/ml	<0.05
Mean prostatic volume	34.72±4.67 cc	26.90±5.72 cc	<0.05

BPH: Benign prostatic hyperplasia, CAD: Coronary artery disease, PSA: Prostate-specific antigen

In the discussion, till now, from Tables 1-3, occurrence of BPH was more in patients with CAD than without CAD, and mean PSA and mean prostatic volume was more in subjects with CAD than without CAD.

The occurrence of BPH was more in subjects with CAD that in turn perhaps has led to an increased mean PSA and mean prostatic volume.

To study the direct effect of CAD on PSA and prostatic volume, it would be better to study the group without BPH.

For further study, 54 out of 75 subjects without BPH were taken as rest 21 subjects had risk factors, and as discussed earlier, the presence of risk factor can affect the occurrence of both BPH and CAD which in turn may affect the value of mean prostatic volume and mean PSA.

This group comprises of a total of 54 subjects. Out of 54, subjects with CAD were 4 and without CAD were 50. The mean age of subjects with CAD (n = 4) was 50.64 ± 8.02 years and that of subjects without CAD was 48.00 ± 0.81 years. No statistically significant difference in the mean age of both these groups was noted as shown in Table 4.

Mean PSA of 4 subjects with CAD in this group was 1.94 ± 0.52 ng/ml and mean PSA of remaining 50 subjects without CAD was 1.44 ± 0.96 ng/ml. Although there was a difference of 0.50 ng/ml, it was not statistically significant (Table 4). To study proper significance, larger sample size is needed.

Satiroglu *et al.*, in their study, on 100 suspected CAD patients with mean age of 57 ± 10 years, coronary angiography results were normal in 13, 87% were diseased (non-obstructive CAD [non-critical plaque formation] in 16%, one-vessel disease in 21%, two-vessel disease in 30%, and multivessel disease in 20%). Mean values of total and PSA were 1.4 ± 1.3 ng/mL. Although there was an increasing trend of PSA with more advanced stages of CAD, no significant relationship was established ($P > 0.05$).[17]

Mean prostatic volume of 4 subjects with CAD in this group was 28.00 ± 1.63 cc, and mean prostatic volume of

Table 4: Comparison of parameters according to presence of CAD (among subjects without BPH without risk factors)

Parameters	Subjects with CAD (*n*=4)	Subjects without CAD (*n*=50)	*P* value
Mean age	48.00±0.81 years	50.64±8.02 years	>0.05
Mean PSA	1.94±0.52 ng/ml	1.44±0.96 ng/ml	>0.05
Mean prostatic volume	28±1.63 cc	22.08±1.61 cc	<0.05

BPH: Benign prostatic hyperplasia, CAD: Coronary artery disease, PSA: Prostate-specific antigen

remaining 50 subjects without CAD was 22.08 ± 1.61 cc. The difference of volume of prostate between these two groups was statistically significant (Table 4).

About similar findings were noted by Inci *et al.*, in their study, on 45 subjects with CAD and 47 controls with mean age 55.7± 11.7 years and 53.5 ± 13.0 years, found the mean prostate volume of CAD subjects 39.1 ± 10.3 cc and controls 33.5 ± 9.4 cc. The difference of mean prostatic volume was statistically significant ($P < 0.05$).[18]

CONCLUSION

In subjects with BPH, a significant association was found with CAD than subjects without BPH which remained so even after exclusion of subjects with risk factor/factors. In age- and risk factor-matched subjects, it was found that those with CAD had significantly higher occurrence of BPH than those without CAD. On further comparison between the two groups (subjects with CAD and without CAD), those with CAD had increased PSA than those without CAD, but after removal of subjects with BPH and/or risk factors, the association became non-significant. Comparison between the groups (subjects with CAD and without CAD), subjects with CAD had higher prostatic volume than without CAD and remained so even after removal of subjects with BPH and/or risk factors.

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