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Original Research Article

Family History, Smoking and Diet as Risk Factors for Benign and Malignant Lesions of Prostate

Mitesh Shah[1], Puja Singh[2], Ruchi Agrawal[3], Rajesh Gaur[4], Amar Gangwani[5]

[1]Assistant Professor, Department of Pathology, Bundelkhand Medical College, Sagar, MP
[2]Associate Professor, Department of Pathology, Bundelkhand Medical College, Sagar, MP
[3]Demonstrator, Department of Microbiology, Bundelkhand Medical College, Sagar, MP
[4]Professor, Department of Pathology, Gajra Raja Medical College, Gwalior, MP
[5]Professor, Department of Pathology, Bundelkhand Medical College, Sagar, MP

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Corresponding author: Dr. Ruchi Agrawal
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Abstract:

Background: Around the world more than 1.1 million cases of prostate cancer were recorded in 2012, accounting for around 8 per cent of all new cancer cases and 15 percent in men. Various risk factors for prostatic diseases under investigation are migration, diet, obesity, height, endogenous androgen level, concomitant medical conditions. Positive family history is a significant risk factor for prostate cancer. Present study is an effort to assess family history, smoking and diet as risk factor for prostatic lesions.

Material & Methods: Study was conducted after institutional ethical committee clearance in Department of Pathology, Gajra Raja Medical College and associated hospital, Gwalior (M.P.). Family history of prostatic disease, smoking and dietary history was taken from patients and their correlation is statistically assessed.

Result: No statistically difference was found between control and study group for vegetarian and mixed diet, while statistically significant difference was found for amount (odds ratio- 20.0, p-value-0.004, risk ratio-12.4) and duration (odds ratio-13.75, p-value-0.006, risk ratio-7.95) of smoking with malignant lesions while not for benign lesions. Family history was found only in 1 case of benign lesion.

Conclusion: Present study shows only correlation of amount and duration of smoking and prostatic carcinoma while no correlation with diet and family history found. But further studies on large scale should be conducted to find correlation so as to assess risk factors.

Keywords: Prostatic lesions, family history, diet, smoking.

Introduction

Around the world more than 1.1 million cases of prostate cancer were recorded in 2012, accounting for around 8 per cent of all new cancer cases and 15 percent in men. The highest incidence of prostate cancer was in Oceania and Northern America; and the lowest incidence in Asia and Africa [1]. The most recent population based cancer registries of different cities for the time period (2008-2011) shows that prostate cancer has ranked among top ten leading sites of cancer in many cities including Bangalore, Bhopal, Nagpur etc. [2].

Various risk factors for prostatic diseases under investigation are migration, diet, obesity, height, endogenous androgen level, concomitant medical conditions like liver cirrhosis, diabetes, BPH , prostatitis, and certain drugs [3]. Association between cigarette smoking and prostatic volume was investigated in men with BPH. Mean prostate volume was greater in non-smokers than smokers. There was no significant difference in serum testosterone, dihydroepiandrosterone (DHEA) and dihydroepiandrosterone sulphate (DHEA-S) levels and weak correlation between the degree of prostatic enlargement, the presence of obstructive symptoms and urinary flow rates found between smokers and non-smokers [4].

Emerging evidence indicates that benign prostate hyperplasia (BPH) and its related lower urinary tract symptoms (LUTS) are frequently observed in subjects with metabolic syndrome which is a well-recognized cluster of cardiovascular risk factors including obesity, hypertension, dyslipidemia, and hyperglycaemia. Several modifiable factors involved in metabolic syndrome determinism, such as inadequate diet, lack of physical exercise; smoking and drinking behaviours are emerging as main contributors to the development of BPH. Metabolic syndrome and its components, hypogonadism, and prostate inflammation probably play an important role in inducing BPH/LUTS in

aging males. The pathogenetic mechanisms underlying the connection between MetS and BPH have not been completely clarified [5]. Racial differences had been noted with black men had higher prevalence and mortality than white men. Differences in socioeconomic status have been suggested as a reason for this difference among various racial groups. Compared with sporadic prostate cancer, hereditary factors are responsible for a low percentage of cases (approximately 9% of all cases) and most commonly affect men with early onset of disease. The evidence linking premalignant entity to clinically detectable and potentially significant prostate cancer is much better established for prostate intraepithelial neoplasia than for atypical adenomatous hyperplasia. Role of occupation, cigarette smoking, sexual activity, sexually transmitted infections, vasectomy and benign prostatic hyperplasia as risk factors for prostate cancer were not conclusive [6].

The four most commonly reported cancers in families were breast (11.8%), lung (10.1%), colorectal (9.4%), and prostate (7.3%) cancer [7].

Positive family history is a significant risk factor for prostate cancer. Men from prostate cancer families with an average age of onset of < 60 years had a significantly higher frequency of PSA positivity as well as cancers than those with a later age of onset. The results suggest that prostate cancer development in genetically predisposed individuals is preceded by a subclinical period when PSA detection can be used for early diagnosis [8].

Relation between smoking and prostate cancer is not clear. In some studies smoking status was poorly defined as unable to separate former smokers from either current or never smokers which led prostate cancer-smoking estimate of effect towards the null value. The four biologic mechanisms most commonly considered to explain how smoking could cause or accelerate the course of prostate cancer involve cadmium, male hormones, genetic mutations, or immune function [9]. Current smokers stratified by amount smoked had significant elevated risk of incident and fatal prostate cancer. The heaviest smokers had a 24% to 30% greater risk of death from prostate cancer as compared to non-smokers [10]. Higher dairy intake had a statistically significant reduced risk of aggressive prostate cancer than lower dairy intake. Dairy foods also protected current, but not former, smokers against aggressive cancer. Associations of dietary fat with prostate cancer risk may vary by type of fat or fat-containing food, and that risk may vary by host factors, including family history and smoking [11]. Animal model studies implicate dietary carcinogens, such as the heterocyclic amines from over-cooked meats and sex steroid hormones, particularly estrogens, as causative agents for prostate cancer acts by causing epithelial cell damage, triggering an inflammatory response that can spawn proliferative inflammatory atrophy (PIA) which represents the earliest of prostate cancer precursor lesions [12].

Aims & Objectives: To assess the risk factor association, particularly family history, smoking and diet with benign and malignant prostatic lesions.

Material & Methods:

Study was conducted after institutional ethical committee clearance in Department of Pathology, Gajra Raja Medical College & associated hospital, Gwalior (M.P.) during December-2015 to October-17. Prostate specimens received with properly filled requisition form and complete history including family history of prostatic disease, smoking history with daily consumption, duration and dietary history whether vegetarian or non-vegetarian diet is taken from patients.

Autolysed samples, specimens without proper documentation and those that cannot be categorized in benign and malignant were excluded from the study. Control groups include random patients admitted in hospital for some other diseases of age ≥ 40 years of age. Histopatholgical examination of protatatic samples was done and categorize in benign and malignant lesions.

For statistical analysis, data were analysed by odds ratio using Epi info software version 7.

Result:

Table 1: Distribution of prostate lesions according to Family history

Group	Family history of Prostatic lesion	
	Present	Absent
Control	0	90
Benign	1 (1.1%)	90
Malignant	0	17

Table 1 shows in the present study family history is absent in control group and in patients with malignant lesions while family history of BPH is present in 1 (1.1%) out of 91 patients with benign lesions.

Table 2: Distribution of prostate lesions according to smoking

Group	Smoking		Total cases
	Present	Absent	
Control	39 (43.3%)	51 (56.7%)	90
Benign	42 (46.2%)	49 (53.8%)	91
Malignant	7 (41.2%)	10 (58.8%)	17

Table 2 shows in control group 43.3% (39) patients are smokers and 56.7% (51) patients are non- smokers. 46.2% (42) patients with benign lesions are smokers and 53.8% (49) patients are non- smokers. 41.2% (7) patients with malignant lesions are smokers and 58.8% (10) patients are non- smokers.

Table 3: Distribution of prostate lesions according to amount of smoking

Group	Smoking		Total smoker patients
	<5 bidi or cigarette/day	≥5 bidi or cigarette/day	
Control	30 (76.9%)	9 (23.1%)	39 (100%)
Benign	31 (73.8%)	11 (26.2%)	42 (100%)
Malignant	1 (14.3%)	6 (85.7%)	7 (100%)

Table 3 shows distribution of prostate lesions in smoker patients into those who smoke < 5 bidi or cigarette/day and those who smoke ≥ 5 bidi or cigarette/day. In control group 30 (76.9%) out of 39 smoker patients smoke <5 bidi or cigarette/day and 9 (23.1%) patients smoke ≥5 bidi or cigarette/day. In smoker patients with benign lesions 31 (73.8%) out of 42 patients smoke <5 bidi or cigarette/day and 9 (23.1%) patients smoke ≥5 bidi or cigarette/day. In smoker patients with malignant lesions 1 (14.3%) out of 7 patients smoke <5 bidi or cigarette/day and 6 (85.7%) patients smoke ≥5 bidi or cigarette/day.

Table 4: Distribution of prostate lesions according to duration of smoking

Group	Duration of Smoking		Total smoker patients
	<50 years	≥50 years	
Control	33 (84.6%)	6 (15.4%)	39 (100%)
Benign	33 (78.6%)	9 (21.4%)	42 (100%)
Malignant	2 (28.6%)	5 (71.4%)	7 (100%)

Table 4 shows distribution of prostate lesions in smoker patients into those who smoke for <50 years and those who smoke for ≥50 years. In control group 33 (84.6%) out of 39 smoker patients smoked for <50 years and 6 (15.4%) patients smoked for ≥50 years. In smoker patients with benign lesions 33 (78.6%) out of 42 patients smoked for <50 years and 9 (21.4%) patients smoked for ≥50 years. In smoker patients with malignant lesions 2 (28.6%) out of 7 patients smoked for <50 years and 7 (71.4%) patients smoked for ≥50 years.

Table 5: Distribution of prostate lesions according to diet

Group	Diet		Total patients
	Vegetarian	Mixed (Vegetarian & Non- Vegetarian)	
Control	49 (54.4%)	41 (45.6%)	90 (100%)
Benign	59 (64.8%)	32 (35.2%)	91(100%)
Malignant	8 (47%)	9 (53%)	17 (100%)

Table: 5 shows distribution of prostate lesions according to vegetarian and mixed (vegetarian + non- vegetarian) diet. In control group 49 (54.4%) patients out of 90 patients were vegetarian and 41 (45.6%) patients were taking mixed (vegetarian & non- vegetarian) diet. In patients with benign lesions 59 (64.8%) out of 91 patients were vegetarian and 32 (35.2%) patients were taking mixed (vegetarian & non- vegetarian) diet. In patients with malignant lesions 8 (47%) out of 17 patients were vegetarian and 9 (53%) patients were taking mixed (vegetarian & non- vegetarian) diet.

Discussion

Family History: In the present study family history is not found in control group and in patients with malignant lesions while family history of BPH is present in 1 (1.1%) out of 91 patients with benign lesions. Positive family history is an additional risk factor for BPH though no gene implicated in Roehrborn CG et al. (2005)[13] and Lu SH et al. (2014)[14]. No patient with malignant lesion in the present study has given positive family history of prostate lesion. Family history is an established risk factor in prostate carcinoma Matikainen MP et al. (1999)[8], Pinsky PF et al. (2003)[7]. Awareness regarding disease and good intellectual level of patient and their attender are essential for proper history. In our study most of the patients are uneducated and lack awareness regarding disease, with small sample size of study may be the reasons for negative family history.

Smoking: In the present study 46.2% (42) patients with benign lesions are smokers and 53.8% (49) patients are non- smokers. 41.2% (7) patients with malignant lesions are smokers and 58.8% (10) patients are non- smokers while in control group 43.3% (39) patients are smokers and 56.7% (51) patients are non- smokers. With respect to risk of disease due to smoking there is no significant difference in control group and patients of benign lesions (odds ratio -1.12, p-value - 0.82, risk ratio-1.05), also difference is not significant in control group and malignant lesions (odds ratio-0.92, p-value-1.0, risk ratio-0.93) 31 (73.8%) out of 42 smoker patients with benign lesions smoke <5 bidi or cigarette/day and 9 (23.1%) patients smoke ≥5 bidi or cigarette/day. 1 (14.3%) out of 7 smoker patients with malignant lesions smoke <5 bidi or cigarette/day and 6 (85.7%) patients smoke ≥5 bidi or cigarette/day. 30 (76.9%) out of 39 smoker patients in control group smoke <5 bidi or cigarette/day and 9 (23.1%) patients smoke ≥5 bidi or cigarette/day. With respect to risk of prostatic lesions in patients who smoke ≥5 bidi or cigarette/day there is no significant difference in control and benign lesions (odds ratio- 1.18, p-value-0.94, risk ratio- 1.08), while there is significant difference in control group and malignant lesions (odds ratio- 20.0, p-value-0.004, risk ratio-12.4)

In smoker patients with benign lesions 33 (78.6%) out of 42 patients smoked for <50 years and 9 (21.4%) patients smoked for ≥50 years. In smoker patients with malignant lesions 2 (28.6%) out of 7 patients smoked for <50 years and 5 (71.4%) patients smoked for ≥50 years. In control group 33 (84.6%) out of 39 smoker patients smoked for <50 years and 6 (15.4%) patients smoked for ≥50 years. No statistically significant correlation between benign lesions and control group with respect to duration of smoking for ≥50 years has been found (odds ratio-1.5, p-value-1.2, risk ratio-0.68) while statistically significant correlation between malignant lesions and control group with respect to duration of smoking for ≥50 years has been found (odds ratio-13.75, p-value-0.006, risk ratio-7.95).

Thus a significant difference in percentage with respect to amount of bidi or cigarette smoked /day and duration of smoking found in our study which is comparable to study of Huncharek M et al.[10] (2010). Though the study was conducted in hospital where people of low socioeconomic status mainly come for treatment, so this difference can be coincidental and further study will be required for definite correlation.

Diet: In patients with benign lesions 59 (64.8%) patients were vegetarian and 32 (35.2%) patients were taking mixed (vegetarian & non- vegetarian) diet i.e. more patients are vegetarian while in patients with malignant lesions 8 (47%) were vegetarian and 9 (53%) patients were taking mixed (vegetarian & non- vegetarian) diet i.e. more patients are taking non vegetarian food. In our study no significant correlation between vegetarian diet and benign (p value-0.2) or malignant (p value-0.76) lesions has been found. Also no significant correlation between mixed (vegetarian and non-vegetarian) diet and benign (p value-0.2) or malignant (p value-0.76) lesions has been found. It is difficult to estimate proportion of vegetarian and non-vegetarian food in cases of patients with mixed diet. Study of Neuhouser ML et al. (2007)[11] and Tewari R et al. (2012)[15] found correlation of prostate carcinoma and high fat intake in particularly animal fat. However Neuhouser ML et al. (2007)[11] stated association of dietary fat with prostate cancer risk may vary by type of fat or fat-containing food, and that risk may vary by host factors, including family history and smoking. Study of Lanou AJ et al. (2010)[16] showed reduce risk of cancer in vegetarians while meat particularly red meat increases risk of cancers including prostate cancer. Further studies should be done and on a higher scale to find significant correlation between diet and prostate lesions.

Conclusion

Present study shows only correlation of amount and duration of smoking and prostatic carcinoma while no correlation with diet and family history found. But further studies on large scale should be conducted to find correlation so as to assess risk factors.

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