EFFECT OF ANTHROPOMETRIC MEASUREMENTS AND PERSONAL DATA PARAMETERS ON BENIGN PROSTATIC HYPERPLASIA AND CARCINOMA PROSTATE

Zehra Naz, Saadia Anjum*

Department of Biochemistry, Islamic International Medical College, Rawalpindi, *Frontier Medical College, Abbottabad, Pakistan

Background: With advancing age the chances of benign prostatic hyperplasia (BPH) and carcinoma prostate (CaP) increases. Although previous studies on physical parameters and personal data parameters have shown the positive associations between BMI, family history, and marital status but they have not seen the effect of occupation and socioeconomic status on BPH and CaP. The objective of the present study was to see the effects of anthropometric measurements and personal history parameters on BPH and CaP in our community. **Methods:** Ninety-three willing subjects from the outpatient department of Sindh Institute of Urology and Transplant (SIUT) were selected for the study. Height and weight were measured, BMI calculated and personal data information, i.e., occupation, marital status, smoking history, family history and socioeconomic history were recorded. Data were analysed for association of various parameters with BPH and CaP. **Results:** Significant association between weight, BMI and skilled labourers was found when normal were compared with BPH cases. **Conclusion:** No significant association between other parameters and BPH, CaP cases was found, except weight, BMI and skilled labour. Body weight should be managed well as it can lead to both cardiovascular as well as prostatic diseases.

Keywords: Body mass index, Benign Prostatic Hyperplasia, CaP, Carcinoma prostate

INTRODUCTION

With advancing age the chances of benign prostatic hyperplasia (BPH) and carcinoma prostate (CaP) increases.[1–3] Age and dihydrotestosterone (DHT)[4] have been found to play an important role in the development of BPH whereas, age, race, family history, androgens, estrogens and other environmental factors have a great impact on CaP.[5]

As the population ages and other causes of disability and death are brought under control the burden of prostate cancer will continue to increase. In Pakistan it's the most common cancer[6] and is the second leading cause of death from cancer. More than one million men of more than 50 years of age who are alive today in the US are destined to die of prostate cancer unless better methods of prevention and treatment are found. The American Cancer Society estimated 3,334,500 new cases (revised to 209,900) and 41,800 deaths from prostate cancer in the US in 1997. This disease now accounts for 32% of cancers and 14% of cancer deaths in American men. The figures are similar in most Western countries, with the highest rate adjusted mortality rates in Scandinavian countries.[7] When compared with white men, black men are diagnosed with prostate cancer at a younger age.[8]

Several studies have identified family history as a risk factor for prostate cancer incidence, typically associated with a two to four fold increase in risk. Some investigators have found that younger men are at a higher risk of family history associated prostate cancer than older men, suggesting that cancers developing due to genetic factors occur early in life. Risks associated with a history on the father's side of the family and in a brother were similar.[9]

Since age, family history, smoking have impact on many types of cancers, therefore, this study was designed to see the effects of anthropometric measurements, i.e., height, weight, body mass index (BMI), and personal data parameters, i.e., occupation, marital and socioeconomic status, in addition to the above mentioned factors in our community.

SUBJECTS AND METHODS

Subjects randomly enrolled for the study were 250 but only ninety-three were willing to participate in the study. These subjects were selected from out patients department of Sindh Institute of Urology and Transplantation (SIUT), Karachi. All the subjects were of 40 years and above in age. The subjects were divided onto 3 groups consisting of normal, BPH and CaP cases; each group was comprised of thirty-one subjects.

Verbal consent was obtained from the subjects. A performa regarding general information about age, occupation, marital status, smoking history, family and socioeconomic history was duly filled. Anthropometric measurements like height (Cm) and weight (Kg) were measured and body mass index (BMI) was calculated.[2]

Values like age, height, weight, BMI, duration of smoking and the number of cigarettes per day were expressed as mean and standard error of mean (SEM), and Student's *t*-test was applied. Chi-square test was applied to find an association among occupation, marital status, smoking, family and socioeconomic history with normal, BPH and CaP cases.

RESULTS

Table-1 shows the age, height, weight and BMI of the subjects. Table-2 shows the comparison of physical parameters in normal, BPH and CaP cases. There was no statistically significant difference in age and height of subjects of these groups. Weight was statistically significant (p<0.05) in normal versus BPH cases, whereas non-significant in normal versus CaP and BPH versus CaP cases. Body mass index was statistically significant (p<0.05) when we compared normal with BPH cases.

Table-3 shows the association of occupation, marital status, smoking, family and socioeconomic history with normal, BPH and CaP cases. Regarding occupation, subjects of the 3 groups, i.e., normal, BPH, and CaP cases were divided into office workers, skilled labourers, hard labourers and jobless. When compared normal with BPH, for skilled labourers differences were significant (p=0.01), whereas in office workers and hard labourers they were statistically non-significant. When compared normal with CaP cases, and BPH with CaP cases results were statistically non-significant.

Regarding marital status, when comparing normal with BPH cases, Normal with CaP cases and BPH with CaP cases, the results were statistically non-significant. Considering smoking, results showed independence of these three groups with smoking (χ^2=0.09, p=0.95). Duration of smoking in normal, BPH and CaP cases was 26.71±4.57, 24.26±3.63 and 25.89±3.54 years and the number of cigarettes per day in normal, BPH and CaP was 9.82±1.90, 13.88±3.17 and 14.5±3.69 respectively. There was no significant association of duration and number of cigarettes smoked among any of the three groups. Association of family history with normal, BPH and CaP cases, showed no dependency on family history in these groups (χ^2=0.89, p=0.63) In normal, BPH and CaP cases there was no dependency on socio-economic status (χ^2=4.66, p=0.09).

Table-1: Anthropometric measurements in normal, benign prostatic hyperplasia and carcinoma prostate cases (Mean±SEM)

Parameters	Normal (n=31)	BPH (n=31)	CaP (n=31)
Age (Years)	62.90±1.62	62.90±1.62	64.19±1.65
Height (Cm)	166.15±0.84	165.68±1.12	163.88±1.88
Weight (Kg)	68.70±2.52	61.33±2.45	63.81±2.46
BMI (Kg/m^2)	24.96±0.82	22.35±0.77	23.72±0.63

Table-2: Anthropometric measurements of normal, benign prostatic hyperplasia and carcinoma prostate cases

	Normal vs BPH			Normal vs CaP			BPH vs CaP		
Parameters	**t**	**df**	***p***	**t**	**df**	***p***	**t**	**df**	***p***
Age (Years)	0.33	60	0.74	0.56	60	0.58	0.88	60	0.39
Height (cm)	0.33	60	0.74	1.09	60	0.26	0.82	60	0.41
Weight (Kg)	2.08	60	<0.05*	1.35	60	0.17	0.71	60	0.47
BMI (Kg/m^2)	2.29	60	<0.05*	1.18	60	0.24	1.35	60	0.17

*Significant

Table-3: Association of personal data parameters with normal, benign prostatic hyperplasia and carcinoma prostate cases

Parameter		Normal (n=31)	BPH (n=31)	CaP (n=31)	Total	Normal vs. BPH	Normal vs. CaP	BPH vs. CaP
Marital status	Married	31 (33.69%)	30 (32.60%)	31 (33.69%)	92	χ^2=0.02*	χ^2=0.81*	χ^2=0.2*
	Unmarried	--	1 (100%)	--	1	--	--	--
Occupation	Office workers	11(37.93%)	6 (20.68%)	12 (41.37%)	29	χ^2=2.08*	χ^2=0.07*	χ^2=2.90*
	Skilled labourers	7 (21.87%)	16 (50%)	9 (28.12%)	32	χ^2=5.5***	χ^2=0.33*	χ^2=3.22*
	Hard labourers	13(41.93%)	9 (29.03%)	9 (29.03%)	31	χ^2=1.13*	χ^2=1.13*	--
	Jobless	--	--	1 (100%)	1	--	--	--
Smoker	Yes	17(32.69%)	17(32.69%)	18 (34.61%)	52	NA	NA	NA
	No	14(34.14%)	14(34.14%)	13 (31.7%)	41			
	χ^2=0.09*							
Family history	Yes	4 (40%)	2 (20%)	4 (40%)	10	NA	NA	NA
	No	27(32.53%)	29(34.93%)	27(32.53%)	83			
	χ^2=0.89*							
Socio-economic history	Satisfactory	15(46.87%)	7 (21.87%)	10 (31.25%)	32	NA	NA	NA
	Unsatisfactory	16(26.22%)	24(39.34%)	21(34.42%)	61			
	χ^2=4.66*							

*Non-significant, **p<0.05, ***p<0.01, ***p<0.001, Key: BPH= Benign prostatic hyperplasia, CaP= Carcinoma Prostate, NA=Not applied

DISCUSSION

Benign prostatic hyperplasia and CaP are diagnosed on the basis of transrectal ultrasound (TRUS) and biopsy, but certain factors like age, family history; environmental causes etc. also play an important role. Consideration of factors that can influence these prostatic diseases should be emphasised whenever we talk of economical burden of a disease.

In the present study, analysis of physical characteristics such as age, height, weight and BMI revealed non-significant association of the first 2 characteristics. This is perhaps understandable and self-explanatory, and needs no discussion. The researchers

failed to find any study (in case of height) conducted in the past also, with a positive and significant association. However the later two factors (weight and BMI) showed significance when normal and BPH cases were compared. Arruda *et al*[10] conducted a study on Amazon community for the sole purpose of verifying the occurrence and investigating any possible association of CaP with overweight (BMI). Only 22 men aged 55 years and above were included in the study. 68.1% were found to be over weight. Irani *et al*[11] found similar values of BMI in BPH and CaP as seen in the present study.

Freeland and Platz[12] found by reviewing the data that there is a positive association of obesity and prostate cancer. Wernyl *et al*[13], Rundle and Neugut[14], and Price *et al*[15] had found the effect of increasing weight but it is on PSA, i.e., with increasing weight and BMI there is a decline in PSA.

In our study marital status was not found to be associated with BPH and CaP cases. Mills *et al*[16] also found no significant association of marital status with CaP case. Weinrich *et al*[17] had smaller proportion of single men; while in present study only one subject was unmarried.

When comparing normal with BPH cases for skilled labourers, results were significant (p=0.01), other results were non-significant. Mills and Yang[18] in their study reported that specific occupational factors have not yet been identified. Their study population only belonged to farmer community (some subjects in present study were farmers but not all). Mills and Yang also found that the risk of CaP was not associated with pattern of employment in any commodity; and that it appeared to be associated with the chemicals used in pesticides.

Regarding smoking history, no association was found between cigarette smoking and BPH, CaP case. Smoking is most likely not associated with prostate cancer incidence; however there is some evidence that smoking may be positively associated with mortality from this cancer. Mills *et al*[16] found that cigarette smoking was not a discernible trend in risk, in case of CaP. Researchers failed to find any other study showing a positive association between smoking and prostate diseases, although extensive work has been done in this respect with other types of cancers. Again this study appears to be one of the first few ever conducted in this subject.

Present study showed no association of family history with BPH and CaP. A number of studies conducted in the past namely, Kalish *et al*[9], Lichtenstein *et al*[19], Catalona *et al*[20] and John *et al*[21], have seen a positive association between carcinoma prostate and family history. Failure of present study to find an association between family history and CaP might be owing to, lacking of awareness among the population, hence they failed to report proper history; sample size in case of present study; lack of awareness among the previous generations towards importance of seeking medical advice given the symptoms and lastly, lack of medical facilities in the time of older generations.

CONCLUSIONS

We failed to find significant association between socio-economic status and BPH and CaP cases. Although, the present study was not able to find significant associations of personal data parameters (except skilled labourers) and anthropometric measurements (except weight and BMI) with BPH and CaP, they may be one of the contributing factors for them if explored further on a larger scale.

ACKNOWLEDGEMENTS

We are thankful to Professor Dr. Adeeb-ul-Hasan, Professor Dr. S. A. Anwar Naqvi, Professor Dr. Mirza Naqi Zafar, and staff of SIUT for their active support. We are also thankful to our study subjects for their cooperation and willingness to participate in the study.

REFERENCES

1. Arrighi HM, Metter EJ, Guess HA, Fozzard JL. Natural history of benign prostatic hyperplasia and the risk prostectomy: The Baltimore longitudinal study of aging. Urology 1991;38(1 Suppl):4–8.
2. Lee F, Littrup PJ, Loft-Christensen L, Kelly BS, McHugh TA, Siders DB, *et al*. Predicted prostate specific results using transrectal ultrasound gland volume. Differentiation of benign prostatic hyperplasia and prostate cancer. Cancer 1992;70(1 Suppl):211–20.
3. Kuriyama M, Wang MC, Papsidero LD, Killian CS, Shimano T, Valenzeula L, *et al*. Quantitation of prostate specific antigen in serum by a sensitive enzyme immunoassay. Cancer Res 1980;40:4568–62.
4. McConnell JD, Prostatic growth-new insights into hormonal regulation. Br J Urol 1995;76:5–10.
5. Bostwick DG, Shan A, Qian J, Darson M, Maihle NJ, Jenkins RB, *et al*. Independent origin of multiple foci of prostatic intra-epithelial neoplasia. Cancer 1992;83:1995–2002.
6. Zahir N, Mubarik A, Abdullah P, Mehmood A, Zahur-ur Rehman. Pattern of malignant tumors in Karachi, is it different? J Coll Phys Surg Pak 2000;10:338–41.
7. Abbas F, Scardino PT. The natural history of prostate carcinoma. Cancer 1997;80:827–33.
8. Freeland SJ, Sutter ME, Naitoh J, Dorey F, Csathy GS, Aroson WJ. Clinical characteristics in black and white men with prostate cancer in an equal access medical center. Urology 2000;55:387–90.
9. Kalish LA, McDougal WS, McKinlay JB. Family history and the risk of prostate cancer. Urology 2000;56:803–6.
10. Arruda HO, Vieira-Filho JP, Ortiz V, Srougi M. PSA and anthropometric measurements among Amazon Indians: an evaluation of the Parkateje community. Rev Saude Publica 2003;37:624–8.
11. Irani J, Lefebvre O, Murat F, Dahmani L, Dore B. Obesity in relation to prostate cancer risk: comparison with a population having benign prostatic hyperplasia. BJU Int 2003;91:482–4.
12. Freeland SJ, Platz EA. Obesity and prostate cancer: Making sense out of apparently conflicting data. Epidemiol Rev 2007;29:88–97.

13. Wernyl DM, Thompson T, Saraiyal M, Freedman D, Kottiri BJ, German RR, *et al*. obesity is negatively associated with prostate specific antigen in US men, 2001–2004. Cancer Epidemiol Biomarkers Prev 2007;16(1):70–6.
14. Rundle A, Neugut AI. Obesity and screening of PSA levels among men undergoing an annual physical examination. Prostate 2008;68(4):373–80.
15. Price MM, Hamilton RJ, Robertson CN, Butts MC, Freeland SJ. Body mass index, prostate specific antigen and digital rectal examination findings among participants in a prostate cancer screening clinic. Urology 2008;71:787–91.
16. Mills PK, Beeson WL, Phillips RL, Fraser GE. Cohort study of diet, life style and prostate cancer in Adventist men. Cancer 1989;64:598–604.
17. Weinrich MC, Jacobsen SJ, Weinrich SP, Moul JW, Oesterling JE, Jacobson D, *et al*. Reference ranges for serum prostate specific antigen in black and white men without cancer. Urology 1998;52: 967–73.
18. Mills PK, Yang R. Prostate cancer risk in California farm workers. J Occup Environ Med 2003;45:249–58.
19. Lichtenstein P, Holm NV, Verkasalo PK, Iliadou A, Kaprio J, Koskenvuo M, *et al*. Environmental and heritable factors in the causation of cancer. Analyses of cohorts of twins from Sweden, Denmark and Finland. N Engl J Med 2000;343:78–85.
20. Catalona WJ, Antenor JA, Roehl KA, Moul JW. Screening for prostate cancer in high risk population. J Urol 2002;168:1980–3.
21. Johns LE, Houlston RS. A systematic review and meta-analysis of familial prostate cancer risk. BJU Int 2003;91:789–94.

Address for correspondence:

Dr. Zehra Naz, 25-A, Lane 3, Tulsa Road, Lalazar, Rawalpindi. **Tel:** +92-51-9240965, **Cell:** +92-333-3392924
Email: zehranaz@hotmail.com