Original Article

Effect of obesity on International Prostate Symptom Score and prostate volume

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Abstract

Aim: The aim of this study was to investigate the relationship between obesity and lower urinary tract symptoms and prostate volume in patients who underwent prostate biopsies.

Materials and Methods: Between December 2008 and November 2009, transrectal ultrasound-guided prostate biopsy was performed on patients who had elevated prostate-specific antigen levels or abnormal digital rectal examination findings. A total of 211 patients were included in this study. Prostate volumes, International Prostate Symptom Score (IPSS) values, and the patient's height and weight were all recorded during the biopsy. Body mass index (BMI) <18.5 was determined as underweight, 18.5–23.0 normal, 23.0–27.5 overweight, and >27.5 obese.

Results: The mean age of the patients was 68.0 ± 6.3 years, and the mean BMI was 28.0 ± 4.9 kg/m^2. The mean prostate volume of the normal, overweight, and obese groups was 30, 50, and 70 ml, respectively. The positive and statistically significant correlation between BMI and prostate volume was determined ($P < 0.001$). According to BMI, the mean IPSS was 8.0, 16.5, and 20.0 in the groups, respectively. Similarly, a statistically positive correlation between BMI and IPSS was demonstrated ($P < 0.001$).

Conclusions: As the result of a rise in BMI, prostate volumes and IPSS increase in patients. Prostate volume and IPSS decrease due to weight loss, and hence that fewer urinary symptoms occur, and the quality-of-life of patients may increase.

Key Words: Body mass index, International Prostate Symptom Score, lower urinary tract symptoms, prostate volume

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INTRODUCTION

The majority of males over the age of 50 have lower urinary tract symptoms (LUTS) and benign prostatic hyperplasia (BPH). LUTS is generally caused by BPH. It decreases quality-of-life[1] and is prevalent among old men. Research has shown that the prostates of obese men are larger than the prostates of normal-weight men.[2-6] Recent studies determined a relationship between LUTS and obesity.[2,4,7,8] Hyperinsulinemia was the proposed pathophysiology,[9-11] which is caused by tissue insulin resistance that stimulates the autonomic nervous system. Hyperinsulinemia stimulates the sympathetic nervous system in particular.[12] Bladder outlet obstruction and LUTS are caused by the over functioning of the sympathetic nervous system.

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MATERIALS AND METHODS

Two hundred and eleven patients who underwent a transrectal ultrasound (TRUS)-guided prostate biopsy due to elevated prostate-specific antigen (PSA) levels or abnormal digital rectal examination findings were evaluated retrospectively between December 2008 and November 2009. The study was approved by the local research ethics committee.

A clinical evaluation, including the International Prostate Symptom Score (IPSS) questionnaire, PSA level, height and weight measurement, and TRUS findings was performed of each patient. Body mass index (BMI) was calculated by dividing the weight of the patient by his height in meters squared (kg/m^2). The IPSS was used for the evaluation of LUTS. Patients were divided into four groups according to BMI: <18.5 (underweight), 18.5–23.0 (normal), 23.0–27.5 (overweight), and >27.5 (obese). According to the IPSS, LUTS is characterized as mild between 0 and 7, moderate between 8 and 19, and severe between 20 and 35.

Exclusion criteria included the use of 5 alfa reductase inhibitor or antiandrogen, which may affect the prostate volume. Furthermore, patients with bladder stones, the presence of neurogenic bladder dysfunction, previous prostate surgeries, recurrent urinary tract infections or a history of acute or chronic prostatitis in the last 3 months were excluded from the study.

Statistical analyses

Data analysis was performed using SPSS for Windows, version 11.5 (SPSS Inc., Chicago, IL, United States). Whether the distributions of continuous variables were normally distributed or not was determined using the Shapiro–Wilk test. Data are shown as mean ± standard deviation or median (min-max), where applicable. The median volume and IPSS differences among groups were compared using the Kruskal–Wallis test. When the P value from the Kruskal–Wallis test statistics was statistically significant, Conover's nonparametric multiple comparison tests was used to identify, which group differed from the others. Categorical data were analyzed using Pearson's Chi-squared test or Fisher's exact test, where appropriate. Degrees of associations among prostate volume, IPSS, and BMI were evaluated using Spearman's rank correlation test. $P < 0.05$ was considered as statistically significant.

RESULTS

The mean age of the patients was 68.0 ± 0.3 years, and the mean BMI was 28.0 ± 4.9 kg/m^2. Our patient population consisted of obese patients. The distribution of the 211 patients according to BMI was: Two underweight patients (0.9%), 46 normal-weight patients (21.8%), 36 overweight patients (17.1%), and 127 obese patients (60.2%). Analysis was performed by including the underweight two patients in the normal group. The mean BMI, prostate volume, and IPSS values of the groups are listed in Table 1.

Prostate volume values for the overweight and obese group according to BMI were significantly higher than the normal group ($P < 0.001$). In addition, prostate volumes in the obese group were statistically higher than in the overweight group ($P = 0.002$) [Table 2 and Figure 1].

The level of IPSS was significantly higher in overweight and obese patients than patients in the normal group ($P < 0.001$). Furthermore, IPSS levels were significantly higher in the obese group than the overweight group ($P = 0.010$) [Table 2 and Figure 1].

In addition, a higher proportion of patients with low levels of IPSS were in the normal group, as opposed to the overweight or obese groups ($P < 0.001$). Likewise, a lower proportion of individuals with high levels of IPSS was found in the normal group compared with the other groups ($P = 0.011$ and $P < 0.001$, respectively). Among the three groups, there was no statistically significant difference in terms of intermediate IPSS level ($P = 0.225$) [Table 2].

Table 1: Demographical and clinical characteristics

Variable	*n*=211 (%)
Age	68.0±6.3
Age range	56-90
BMI (kg/m^2)	28.0±4.9
BMI groups	
<18.5 kg/m^2	2 (0.9)
18.5-23.0 kg/m^2	46 (21.8)
23.0-27.5 kg/m^2	36 (17.1)
≥27.5 kg/m^2	127 (60.2)
Volume	55 (18-137)
IPSS	18 (3-30)
IPSS groups	
Mild	18 (8.5)
Moderate	101 (47.9)
Severe	92 (43.6)

BMI: Body mass index, IPSS: International Prostate Symptom Score

Table 2: Prostate volume and IPSS findings in terms of BMI

Variables	Normal (*n*=48)	Overweight (*n*=36)	Obese (*n*=127)	*P*
Volume	30 (18-113)[a,b]	50 (30-90)[a,c]	70 (18-137)[b,c]	<0.001
IPSS	8 (6-30)[a,b]	16.5 (6-28)[a,c]	20 (3-30)[b,c]	<0.001
IPSS groups (%)				
Mild	16 (33.3)[a,b]	1 (2.8)[a]	1 (0.8)[b]	<0.001
Moderate	25 (52.1)	21 (58.3)	55 (43.3)	0.225
Severe	7 (14.6)[a,b]	14 (38.9)[a]	71 (55.9)[b]	<0.001

[a]The difference between the normal and overweight groups was found to be statistically significant ($P<0.05$), [b]The difference between the normal and obese groups was found to be statistically significant ($P<0.001$), [c]The difference between the overweight and obese groups was found to be statistically significant ($P<0.01$). BMI: Body mass index, IPSS: International Prostate Symptom Score

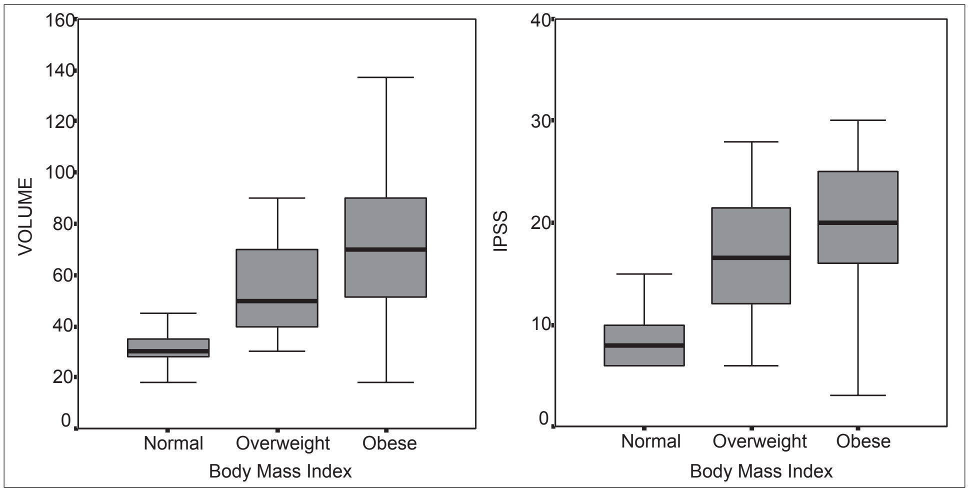

Figure 1: Prostate volume and International Prostate Symptom Score findings in terms of body mass index

We examined the relationships among prostate volume, IPSS, and BMI. In a univariate analysis using Spearman's rank correlation test coefficient, BMI was positively correlated with prostate volume ($r = 0.630$ and $P < 0.001$) and IPSS ($r = 0.604$ and $P < 0.001$) [Figure 2].

DISCUSSION

We analyzed the relationship among obesity, prostate volume, and IPSS in prostate biopsy patients. Obese patients were found to have higher prostate volumes and IPSS values than normal individuals. There was a statistically significant difference between the prostate volumes and IPSS values of obese patients and patients in other BMI groups. We found a positive correlation between BMI and IPSS and BMI and prostate volume.

A restrictive aspect of our work may be that the population included in our work does not consist of asymptomatic people and that these individuals may not reflect the common characteristics of general society. The patients included in our work consisted of individuals who were selected on the basis of a prostate biopsy.

Kim *et al*. analyzed the relationship between prostate volume and metabolic and anthropometric parameters. The researchers found that there was a correlation between prostate volume and body weight and height; however, multivariable linear regression analysis revealed no statistically significant correlation between BMI and prostate volume.[13] A study carried out with 465 men in a health promotion center reported a positive correlation between prostate volume and central obesity based on waist circumference and BMI, not based on overall obesity.[14] On the other hand, a study carried out in the United States with men who had undergone a radical prostatectomy showed that there was a positive relationship between BMI and prostate volume in the patients under the age of 63.[15] Another study carried out on men who had had benign biopsies reported that there was a direct relationship between BMI and prostate volume.[16]

Obesity can increase sympathetic nervous systems activity, which affects prostatic enlargement and urinary obstructive symptoms.[17] Kristal *et al*. analyzed a number of modifiable life-style factors that affect symptomatic BPH in 5,600 men who participated in the prostate cancer prevention trial. The patients were included in a placebo group of the trial and were followed for 7 years.[18] The authors found that symptomatic BPH (IPSS > 14) significantly increased with obesity. For this reason, Kristal *et al*. suggested that there was a relationship between higher LUTS prevalence and adulthood obesity. Rohrmann *et al*. analyzed the relationship between LUTS and obesity in the National Health and Nutrition Examination Survey III.[7] They found that there was a positive relationship between increased BMI and prevalence of LUTS.

Obesity increases estrogen levels by CYP19 (aromatase) conversion of androgens in adipose tissue. Obese men have a larger prostate volume and lower testosterone concentrations.[19] Obesity lowers free and total testosterone and serum globulin-binding protein levels, but also increases estrogen levels, both free and total estradiol concentrations.[20] Higher estrogen levels and lower testosterone levels can affect prostate cell growth. The ratio of estrogen to androgen and sympathetic nervous activity are known to affect BPH development and LUTS severity.[21] Abdominal obesity increases with both of the conditions discussed above. Obese men are at an increased risk for BPH. The link to the quality of venous drainage to BPH is another pathway that explains this risk.[7]

Gat *et al*. reported that an impairment of the testicular venous drainage system in an erect posture causes BPH.[22] The

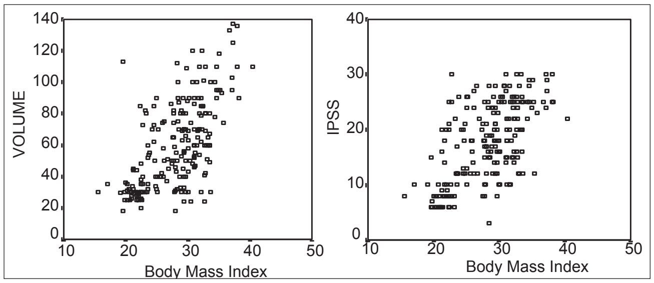

Figure 2: Univariate analysis using Spearman's rank correlation test coefficient for body mass index and prostate volume and BMI and International Prostate Symptom Score

researchers reported that the one-way valves in the vertically oriented internal spermatic veins were destroyed (varicocele) in BPH patients, causing 6 times higher hydrostatic pressure than normal in the venous drainage of the male reproductive system. In addition, a large abdominal mass above the testicular venous system can negatively affect the prostate in obese men.

Recent studies of the pathophysiology of BPH reported that newly identified risk factors, including diet and obesity, can affect the development of BPH. These risk factors are in addition to conventional risk factors such as age, family history, and androgen activity.[23]

CONCLUSIONS

Patients with a high BMI may be at risk for an increased prostate volume and IPSS. Therefore, losing weight may result in a smaller prostate volume in the geriatric period, so that LUTS will be less pronounced and quality of life will be improved.

REFERENCES

1. Rohrmann S, Smit E, Giovannucci E, Platz EA. Associations of obesity with lower urinary tract symptoms and noncancer prostate surgery in the Third National Health and Nutrition Examination Survey. Am J Epidemiol 2004;159:390-7.
2. Hammarsten J, Högstedt B, Holthuis N, Mellström D. Components of the metabolic syndrome-risk factors for the development of benign prostatic hyperplasia. Prostate Cancer Prostatic Dis 1998;1:157-62.
3. Gupta A, Gupta S, Pavuk M, Roehrborn CG. Anthropometric and metabolic factors and risk of benign prostatic hyperplasia: A prospective cohort study of Air Force veterans. Urology 2006;68:1198-205.
4. Ozden C, Ozdal OL, Urgancioglu G, Koyuncu H, Gokkaya S, Memis A. The correlation between metabolic syndrome and prostatic growth in patients with benign prostatic hyperplasia. Eur Urol 2007;51:199-203.
5. Xie LP, Bai Y, Zhang XZ, Zheng XY, Yao KS, Xu L, *et al.* Obesity and benign prostatic enlargement: A large observational study in China. Urology 2007;69:680-4.
6. Putnam SD, Cerhan JR, Parker AS, Bianchi GD, Wallace RB, Cantor KP, *et al.* Lifestyle and anthropometric risk factors for prostate cancer in a cohort of Iowa men. Ann Epidemiol 2000;10:361-9.
7. Rohrmann S, Smit E, Giovannucci E, Platz EA. Association between markers of the metabolic syndrome and lower urinary tract symptoms in the Third National Health and Nutrition Examination Survey (NHANES III). Int J Obes (Lond) 2005;29:310-6.
8. Joseph MA, Harlow SD, Wei JT, Sarma AV, Dunn RL, Taylor JM, *et al.* Risk factors for lower urinary tract symptoms in a population-based sample of African-American men. Am J Epidemiol 2003;157:906-14.
9. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 1988;37:1595-607.
10. DeFronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care 1991;14:173-94.
11. Rett K, Wicklmayr M, Mehnert H. New aspects of insulin resistance in hypertension. Eur Heart J 1994;15 Suppl C: 78-81.
12. Berne C, Fagius J, Pollare T, Hjemdahl P. The sympathetic response to euglycaemic hyperinsulinaemia. Evidence from microelectrode nerve recordings in healthy subjects. Diabetologia 1992;35:873-9.
13. Kim YD, Yang WJ, Song YS, Park YH. Correlation between prostate volume and metabolic or anthropometric factors in male visitors to a health promotion center. Korean J Urol 2008;49:139-44.
14. Kim GW, Doo SW, Yang WJ, Song YS. Effects of obesity on prostate volume and lower urinary tract symptoms in Korean men. Korean J Urol 2010;51:344-7.
15. Freedland SJ, Platz EA, Presti JC Jr, Aronson WJ, Amling CL, Kane CJ, *et al.* Obesity, serum prostate specific antigen and prostate size: Implications for prostate cancer detection. J Urol 2006;175:500-4.
16. Ochiai A, Fritsche HA, Babaian RJ. Influence of anthropometric measurements, age, and prostate volume on prostate-specific antigen levels in men with a low risk of prostate cancer. Urology 2005;66:819-23.
17. Dahle SE, Chokkalingam AP, Gao YT, Deng J, Stanczyk FZ, Hsing AW. Body size and serum levels of insulin and leptin in relation to the risk of benign prostatic hyperplasia. J Urol 2002;168:599-604.
18. Kristal AR, Arnold KB, Schenk JM, Neuhouser ML, Weiss N, Goodman P, *et al.* Race/ethnicity, obesity, health related behaviors and the risk of symptomatic benign prostatic hyperplasia: Results from the prostate cancer prevention trial. J Urol 2007;177:1395-400.
19. Lee S, Min HG, Choi SH, Kim YJ, Oh SW, Kim YJ, *et al.* Central obesity as a risk factor for prostatic hyperplasia. Obesity (Silver Spring) 2006;14:172-9.
20. Pasquali R, Casimirri F, Cantobelli S, Melchionda N, Morselli Labate AM, Fabbri R, *et al.* Effect of obesity and body fat distribution on sex hormones and insulin in men. Metabolism 1991;40:101-4.
21. Barqawi AB, Golden BK, O'Donnell C, Brawer MK, Crawford ED. Observed effect of age and body mass index on total and complexed PSA: Analysis from a national screening program. Urology 2005;65:708-12.
22. Gat Y, Gornish M, Heiblum M, Joshua S. Reversal of benign prostate hyperplasia by selective occlusion of impaired venous drainage in the male reproductive system: Novel mechanism, new treatment. Andrologia 2008;40:273-81.
23. Nandeesha H. Benign prostatic hyperplasia: Dietary and metabolic risk factors. Int Urol Nephrol 2008;40:649-56.

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