Original Article

The Correlation between Body Mass Index and Routine Parameters in Men Over Fifty

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Purpose: This study aimed to investigate the relationships between body mass index (BMI) and prostate-specific antigen (PSA) levels, international prostate symptom score (IPSS), quality of life (QoL), and prostate volume (PV).

Materials and Methods: Height, weight, PSA levels, PV, and IPSS were analyzed in 15,435 patients who underwent a prostate examination between 2001 and 2014. Patients aged < 50 years or with a PSA level ≥ 10 ng/mL were excluded. The relationships between BMI and PSA, IPSS, QoL, and PV were analyzed by a scatter plot, one-way analysis of variance, and the Pearson correlation coefficient.

Results: The mean age was 71.95 ± 7.63 years, the mean BMI was 23.59 ± 3.08 kg/m^2, the mean PSA level was 1.45 ± 1.45 ng/mL, the mean IPSS was 15.53 ± 8.31, the mean QoL score was 3.48 ± 1.25, and the mean PV was 29.72 ± 14.02 mL. PSA, IPSS, and QoL showed a tendency to decrease with increasing BMI, and there were statistically significant differences for each parameter ($p \leq 0.001$). PV showed a significant tendency to increase with BMI ($p < 0.001$). In the correlation analysis, BMI showed a statistically significant correlation ($p < 0.001$) with PSA, IPSS, and QoL, although the correlations were very weak. In contrast, BMI showed a significant correlation with PV ($p < 0.001$), with a meaningful Pearson correlation coefficient of 0.124.

Conclusions: Higher BMI was associated with lower PSA levels and higher IPSS and QoL scores. Meanwhile, PV increased with BMI. Although obese individuals had a greater PV, obesity did not aggravate lower urinary tract symptoms.

Key Words: Body mass index; Lower urinary tract symptoms; Prostate-specific antigen; Prostatic hyperplasia

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INTRODUCTION

Obesity is an issue in medicine that has recently received considerable attention worldwide. The prevalence of obesity is increasing at a greater rate than ever before [1]. Various studies have demonstrated that obesity causes physical and mental problems, including diabetes, cardiovascular disease, as manifested via risk factors, such as hyperlipidemia and hypertension, several types of cancer, osteoarthritis [2], and depression [3]. Its negative impact on socioeconomic parameters is making it as much a problem of society as of personal health [4].

Prostate-specific antigen (PSA) is the most widely used tumor marker in prostate cancer screening [5,6]. There is some debate about the appropriate PSA cut-off value to trigger performing a prostate biopsy to diagnose prostate cancer [7,8], and an elevated PSA level is not entirely specific to prostate cancer. Nevertheless, it is clear that PSA plays an important role in the screening and diagnosis of prostate cancer. In a study by Freedland et al [9] in 2008, it was reported that body mass index (BMI) could affect prostate cancer detection, since PSA levels were found to decrease with increasing BMI. The relationship between obesity and PSA levels needs to be clearly elucidated in order to enable appropriate prostate cancer detection—that is, discovering cancer at the right time, before it develops into high-grade prostate cancer, but without performing excessive histological examinations. Although there have been studies on the effects of obesity on PSA levels, the relationship between them is still not clear.

Patients complaining of lower urinary tract symptoms (LUTS) experience considerable physical and mental suffering and reduced quality of life (QoL) [10]. The prevalence of symptoms increases with age [11], meaning that the global increase in life expectancy is producing an increasing number of patients with LUTS. It has been reported that prostate volume (PV) increased with the severity of obesity [9]. However, debates about LUTS continue, because some studies have reported that obesity was associated with LUTS [12], while others have reported that obesity had no effect on LUTS [13]. Moreover, some reports have suggested that LUTS severity increases with PV, but this is not clear either. Neither the effect of obesity on LUTS nor its interaction with QoL has been clearly demonstrated.

Even though the relationship between PSA levels and LUTS is an important element in male health, the interaction of these parameters with obesity has not yet been conclusively assessed. Therefore, this study examined the relationships of the BMI, an objective measure of obesity, with PSA levels, international prostate symptom score (IPSS), QoL, and PV.

MATERIALS AND METHODS

A retrospective analysis was conducted on the prostate examination results of male patients in a single community in South Korea from January 1, 2001 to January 1, 2014. Age, medical history, height, weight, PSA level, PV, and IPSS were investigated in a total of 72,679 patients. PSA levels were measured at a single institution, and PV was measured using a biplane transrectal ultrasound probe. An additional QoL item was included on the IPSS, ranging from 0 (delighted) to 6 (terrible). The 7 items of the IPSS and the QoL item were measured by self-response.

Of the patients, 16,265 were excluded because they had been treated for benign prostatic hyperplasia (BPH), were under 50 years of age, or had a PSA level ≥ 10 ng/mL. Of the remaining 56,414 patients, 40,895 patients were excluded because one of the examination items had been omitted, such as height or weight, making it impossible to calculate the BMI, or the IPSS. Of the remaining 15,519 patients, 84 patients were excluded because their medical history included the diagnosis or treatment (e.g., surgery) of prostate cancer, a history of treatment for BPH, a diagnosis of bladder cancer or radical cystectomy, or a diagnosis of diabetic bladder or neurogenic bladder. The remaining 15,435 patients were included in the final analysis. We calculated the BMI as weight in kilograms divided by height in meters squared (kg/m^2). In accordance with the National Institutes of Health standards, we classified subjects into 5 groups by BMI: underweight for BMI values of < 18.5 kg/m^2, normal weight for BMI values of $18.5 \sim 23.0$ kg/m^2, overweight for BMI values of $23.0 \sim 25.0$ kg/m^2, obese I for BMI values of $25.0 \sim 30.0$ kg/m^2, and obese II for BMI values of ≥ 30.0 kg/m^2 [14].

Together with the subjects' basic information, one-way analysis of variance (ANOVA) was performed to examine

the statistical significance of differences among the 5 BMI groups in mean PSA levels, IPSS, QoL, and PV. The Scheffé method was used for post hoc testing. In order to investigate the correlations of BMI with PSA levels, IPSS, and QoL, a univariate analysis was performed using the Pearson correlation coefficient. Additionally, we used a scatter plot to investigate the relationship between BMI and PSA as continuous variables. Statistical analyses were performed using SPSS ver. 21 (IBM Co., Armonk, NY, USA) and considered p-values < 0.05 to be statistically significant.

Ethics statement

The present study was exempted from institutional review board approval.

RESULTS

The study subjects consisted of a total of 15,435 patients, and their mean age was 71.95 ± 7.63 years, ranging from 50 to 96 years. The subjects' BMI ranged from 13.2 to 40.0 kg/m^2, with a mean of 23.59 ± 3.08 kg/m^2. The mean PSA level was 1.45 ± 1.45 ng/mL, the mean total IPSS was 15.53 ± 8.31, and the mean QoL was $3.48 \pm$

1.25. PV ranged from 7.28 to 233.30 mL, with a mean of 29.72 ± 14.02 mL (Table 1). After classifying subjects into 5 groups according to BMI, there were 702 subjects in the underweight group (BMI < 18.5 kg/m^2), 5,949 subjects in the normal weight group (BMI $18.5 \sim 23.0$ kg/m^2), 3,831 subjects in the overweight group (BMI $23.0 \sim 25.0$ kg/m^2),

Table 1. Baseline characteristics of the subjects (n=15,435)

Characteristic	Mean ± standard deviation	Range
Mean age (y)	71.95±7.63	50~96
BMI (kg/m^2)	23.59±3.08	13.2~40.0
Mean PSA (ng/mL)	1.45±1.45	0.10~9.83
Mean IPSS	15.53±8.31	0~35
Subscore 1	2.16±1.63	0~5
Subscore 2	2.08±1.56	0~5
Subscore 3	2.09±1.61	0~5
Subscore 4	2.11±1.58	0~5
Subscore 5	2.84±1.59	0~5
Subscore 6	1.93±1.67	0~5
Subscore 7	2.33±1.34	0~5
Mean QoL	3.48±1.25	0~6
Mean PV (mL)	29.72±14.02	7.28~233.30

BMI: body mass index, PSA: prostate-specific antigen, IPSS: international prostate symptom score, QoL: quality of life, PV: prostate volume.

Table 2. Comparison of subjects across BMI groups

Variable	BMI group (kg/m^2)					p-value
	<18.5 (underweight)	18.5~23.0 (normal weight)	23.0~25.0 (overweight)	25.0~30.0 (obese I)	≥30.0 (obese II)	
No. of patient	702	5,949	3,831	4,614	339	-
Mean age (y)	75.19±7.42	73.51±7.19	71.31±7.41	70.24±7.76	68.27±8.26	<0.001
Mean BMI (kg/m^2)	17.44±0.99	21.20±1.21	23.98±0.57	26.71±1.23	31.55±1.69	<0.001
Mean PSA (ng/mL)	1.69±1.59	1.51±1.53	1.44±1.39	1.35±1.36	1.28±1.51	0.001
Mean total IPSS	18.11±8.38	16.00±8.45	15.46±8.07	14.75±8.15	13.68±8.88	<0.001
Subscore 1	2.54±1.73	2.16±1.67	2.18±1.58	2.10±1.58	2.03±1.65	0.004
Subscore 2	2.47±1.59	2.12±1.59	2.02±1.51	2.02±1.54	1.91±1.54	<0.001
Subscore 3	2.33±1.72	2.12±1.64	2.11±1.56	2.02±1.59	1.75±1.69	0.008
Subscore 4	2.59±1.66	2.19±1.61	2.08±1.53	1.97±1.56	1.83±1.63	<0.001
Subscore 5	3.16±1.60	2.95±1.58	2.84±1.59	2.69±1.57	2.40±1.68	<0.001
Subscore 6	2.26±1.75	2.01±1.72	1.94±1.64	1.78±1.59	1.60±1.67	<0.001
Subscore 7	2.77±1.38	2.45±1.39	2.28±1.28	2.17±1.30	2.16±1.31	<0.001
Mean QoL	3.79±1.17	3.55±1.24	3.47±1.26	3.37±1.25	3.34±1.37	<0.001
Mean PV (mL)	27.30±12.36	28.43±12.76	29.44±11.63	31.31±15.65	38.88±27.55	<0.001

Values are presented as number only or mean±standard deviation. BMI: body mass index, PSA: prostate-specific antigen, IPSS: international prostate symptom score, QoL: quality of life, PV: prostate volume.

4,614 subjects in the obese I group (BMI 25.0∼30.0 kg/m^2), and 339 subjects in the obese II group (BMI ≥30.0 kg/m^2).

One-way ANOVA was performed to examine the differences among the BMI groups in mean PSA levels, mean total IPSS, mean QoL score, and mean PV. With increasing BMI, the mean PSA level showed a decreasing trend, with values of 1.69, 1.51, 1.44, 1.35, and 1.28 ng/mL, respectively (p=0.001). The mean total IPSS likewise showed a decreasing trend with increasing BMI, with values of 18.11, 16.00, 15.46, 14.75, and 13.68 (p<0.001). QoL also showed a decreasing trend with increasing BMI, with values of 3.79, 3.55, 3.47, 3.37, and 3.34 (p<0.001). In contrast, PV showed an increasing trend with increasing BMI, with values of 27.30, 28.43, 29.44, 31.31, and 38.88 mL (p<0.001) (Table 2).

A correlation analysis was performed using the Pearson correlation coefficient to examine the relationships of BMI with PSA, IPSS, QoL, and PV. Although the correlation between BMI and PSA level was significant, the Pearson correlation coefficient (R value) was −0.056, indicating almost no correlation (p<0.001). There was also a significant correlation between BMI and IPSS, but the Pearson correlation coefficient (R value) of −0.095 indicated almost no correlation (p<0.001). BMI and QoL showed a significant correlation as well, but again, the Pearson correlation coefficient (R value) was only −0.079, meaning that there was almost no correlation (p<0.001). In contrast, BMI and PV showed a significant positive correlation, with a meaningful R value of 0.124 (p<0.001) (Table 3).

A scatter plot was made to show the relationship between BMI and PSA levels as continuous variables. Likewise, BMI and PSA were not found to be related in the scatter plot (Fig. 1).

DISCUSSION

In 2004, Thompson et al [15] examined the relationship between BMI and PSA levels in 1,565 subjects in a local community and reported that there was no association between BMI and PSA levels in either the whole-group analysis or the analysis by ethnicity. No association was reported between BMI and PSA levels when comparisons were made across Hispanic, African American, and Caucasian subjects or when subjects were divided based on their family history of prostate cancer. Fowke et al [16] investigated the associations of body size with PV and PSA level, measuring waist and hip circumference and using the waist-to-hip ratio as an index of body size. That study reported that obesity and height were independently related to PV, as well as to an increase in the PSA level. This was thought to suggest an interaction of PSA expression
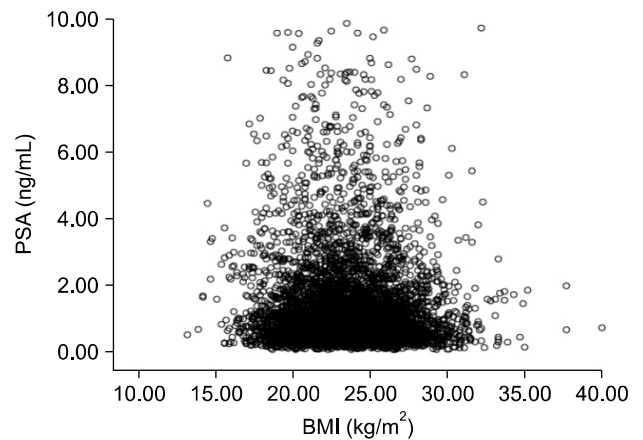
Fig. 1. Scatter plot of the relationship between body mass index (BMI) and prostate-specific antigen (PSA).

Table 3. Relationships of BMI with PSA, IPSS, QoL, and PV

Variable	PSA	IPSS	QoL	PV
R value by the Pearson correlation test	−0.056	−0.095	−0.079	0.124
p-value by the Pearson correlation test	0.000	0.000	0.000	0.000
B value by the linear regression model	−0.026	−0.255	−0.032	0.563
R^2 value by the linear regression model	0.003	0.009	0.006	0.015
p-value by the linear regression model	0.000	0.000	0.000	0.000

BMI: body mass index, PSA: prostate-specific antigen, IPSS: international prostate symptom score, QoL: quality of life, PV: prostate volume.

with insulin regulation or metabolic syndrome. In addition, in a study by Lee et al [17], PSA levels were reported to increase with larger waist circumference, an index of obesity.

In a study in 2010 by Jeong et al [18] on 23,601 subjects, the mean PSA level was confirmed to increase with increasing age and decreasing BMI. However, after correcting for age and BMI, there was no statistically significant association between metabolic syndrome and PSA levels. Of the 5 components of metabolic syndrome, waist circumference and fasting plasma glucose levels were associated with lower PSA levels, while hypertension was associated with a higher PSA level. The authors claimed that these results reflected the heterogeneous relationship between metabolic risk factors and PSA levels.

In 2007, Werny et al [19] also investigated the relationship between obesity and PSA levels in US men aged 40 years or older, by analyzing data from the 2001 to 2004 National Health and Nutrition Examination Survey. After dividing subjects into Caucasians, African-Americans, and Mexican-Americans, weight, BMI, waist circumference, triceps skin fold, and total body water were used as indices of obesity. In this study, Caucasians showed a decrease in PSA levels with increasing obesity, while Mexican-Americans showed a decrease in PSA levels specifically with increasing BMI, and African-Americans showed a decrease in PSA levels with increasing triceps thickness. In a multivariate linear regression analysis correcting for age and ethnicity, PSA levels were found to decrease with increasing weight, BMI, waist circumference, triceps skin fold, and total body water.

Hence, although it has not been completely proven, numerous studies have reported that PSA levels were lower in obese men [20,21]. These studies hypothesized that this is a result of hemodilution, due to an increase in the circulating plasma volume. The results of the present study also confirmed a decrease in PSA level with increasingly severe obesity, and support the claims that this effect is a result of hemodilution of PSA due to increased plasma volume.

Although a study by Freedland et al [9] did not show an association between obesity and prostate cancer risk, it did report a 98% increase in prostate cancer risk after correcting for a relatively lower PSA level and larger PV. In 2007, Wright et al [22] performed a study of obesity, prostate cancer incidence, and mortality in 9,986 subjects. In their study, obesity did not increase prostate cancer incidence, but adult weight gain was associated with fatal prostate cancer. In 2010, Hekal and Ibrahiem [23] proposed an equation that corrected for age and BMI by taking the patient's total PSA level, multiplying by their age, and dividing by their BMI. This supports claims that obesity should be reflected in factors that affect the PSA cut-off value used when deciding whether to perform a biopsy during prostate cancer screening.

In the present study, more severe obesity was associated with a decrease in IPSS and QoL and with an increase in PV. In 2000, Haidinger et al [24] examined LUTS risk factors in 1,557 elderly men, and reported that while there was no correlation of IPSS with weight or BMI, men with higher blood pressure and a larger waist size tended to show a higher IPSS. In a prospective study by Kim et al [13] in 2010, a multivariate analysis showed that waist circumference was the only obesity index that was significantly associated with PV, while a univariate analysis showed no statistically significant relationships between LUTS and any obesity-related indices, as well as no associations between PV and LUTS. Yee et al [25] examined the effects of weight loss on LUTS severity in obese men, and reported no relationship between obesity and LUTS severity. However, the authors did suggest that their result could be due to insufficient weight loss, and predicted that weight loss would still alleviate LUTS. In contrast, a study has also reported that PV and IPSS increased with increasing central obesity [17,26]. Regarding these results, although PV has been confirmed to increase with the severity of obesity in several papers, it is thought that the severity of LUTS may have varied according to the statistical analyses used. In the present study, although there was almost no correlation in the correlation analysis, LUTS were found to show improvement with increasing BMI, and PV also showed an increase in the correlation analysis. This shows that an increase in PV does not necessarily mean an increase in the severity of LUTS. It seems that the PV increase is due to an increase in the volume of the peripheral zone, rather than the transitional zone.

The effects of LUTS severity on QoL have been extensively researched [27-29]. In previous studies, several

instruments have been used for the objective evaluation of QoL, showing a decrease in QoL with increasing LUTS severity. According to a study of 4,800 men and 3,674 women by Boyle et al [27], LUTS severity and the QoL question in the IPSS were clearly correlated in both men and women. The authors also reported that this could impact the health of the affected individuals and their partners. The present study found a negative correlation between obesity severity and QoL. While it is likely that PV is not necessarily correlated with LUTS, LUTS and QoL can be considered to be affected in the same way. The present study also showed a negative correlation of obesity severity with the IPSS total score and QoL. In comparison with other studies, we expected increasing prostate size to be unrelated to, or to be associated with, a deterioration in IPSS total score and QoL. However, that was not the case in the present study. Therefore, we suggest that obesity itself does not always make LUTS more severe.

Our study has some limitations, such as being a retrospective study, and the inability to include other physical measurements apart from BMI, such as waist circumference or skin folds. Other studies have made comparisons using BMI and other physical measurements, but the present study used only BMI, without any analysis of other obesity indices. In addition, because the patients self-reported IPSS and QoL, there is the possibility of bias having a negative impact on the objectivity of the IPSS and QoL measurements. We hypothesized that increased PV represented an increase in the peripheral zone, rather than the transitional zone; in future studies, it will be necessary to investigate the transitional zone size to confirm this correlation.

Nevertheless, this was a large-scale study in a local community. It is significant because it could aid in the interpretation of PSA values according to obesity in actual clinical situations, as well as helping to ascertain the effects of obesity on LUTS. Together with existing studies, additional prospective research will be required to adjust the PSA cut-off value according to obesity, which could be expected to increase the accuracy of prostate cancer diagnosis in the obese population. Finally, we expect that additional studies on the association between obesity and LUTS could help identify lifestyle improvement methods that are likely to alleviate symptoms.

CONCLUSIONS

PSA levels were found to decrease with increasing BMI, which was used as a measure of obesity. This shows that the interpretation of PSA levels may need to change according to the obesity status of the patient. BMI showed a positive correlation with PV, but BMI was not associated with exacerbated LUTS or QoL. Additional prospective studies will be required in the future to examine the PSA cut-off values used in prostate cancer screening according to obesity, and to further investigate the effects of obesity on LUTS and QoL.

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