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ORIGINAL ARTICLE

Lowering the percent body fat in the obese population might reduce male lower urinary tract symptoms

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Abstract

Purpose This study aimed to investigate the practicality of percent body fat (PBF), calculated using bioelectrical impedance analysis (BIA), in predicting benign prostatic hyperplasia/lower urinary tract symptoms (BPH/LUTS).

Methods This study included 844 men who underwent medical checkups at our institution between 2014 and 2022. Demographic characteristics, serum PSA levels, and prostate volume were collected using TRUS. BPH was defined as a prostate volume ≥ 30 cc. Subjects were divided into two groups according to their quartiles of PBF: the normal PBF group (first to third quartile; PBF < 27.9%) and the high PBF group (fourth quartile; PBF ≥ 27.9%). Characteristics between the groups were compared using the chi-square test and Student's t-test. Multivariate logistic regression analysis was performed to evaluate risk factors for BPH and severe LUTS.

Results The prostate volume (25.21 ± 8.4 vs 27.30 ± 9.0, p = 0.005) and percentage of BPH (22.9% vs. 32.1%, p = 0.007) were greater in the high PBF group. After multivariate analysis, old age (OR = 1.066, p < 0.001), higher appendicular skeletal muscle mass index (ASMI) (OR = 1.544, p = 0.001), and PBF ≥ 27.9% (OR = 1.455, p = 0.037) were risk factors for BPH. Larger prostate volume (OR = 1.035, p = 0.002) and PBF ≥ 27.9% (OR = 1.715, p = 0.025) were risk factors for severe LUTS. However, a greater ASMI had a protective effect against severe LUTS (OR = 0.654, p = 0.011).

Conclusions This study shows that PBF and ASMI are useful for predicting BPH/LUTS. We suggest that lowering PBF to the normal range in a population with high PBF might prevent BPH, while lowering PBF and maintaining adequate ASMI could lower LUTS.

Keywords Prostatic hyperplasia · Lower urinary tract symptoms · Metabolic syndrome · Bioelectrical impedance analysis

Introduction

Many elderly men experience lower urinary tract symptoms (LUTS). LUTS are important because they impair the quality of life and impose a substantial economic burden [1]. Moreover, moderate-to-severe LUTS are considered a risk factor for major adverse cardiac events [2, 3]. Bladder outlet obstruction caused by benign prostatic hyperplasia (BPH) is a major cause of LUTS in men. The prevalence of BPH/LUTS ranges from 50 to 75% in men 50 years or older, and 80% in men 70 years or older [4]. A prostate volume of 30 cc or more is known to cause bothersome LUTS, which is enough for men to seek treatment [5]. Also, AUA panels suggested the use of 5-alpha reductase inhibitors for patients with prostate volume over 30 cc to improve LUTS and reduce the risk of urinary retention [1]. These suggest that a prostate size of 30 cc is clinically important.

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Many studies have aimed to identify the factors that affect BPH/LUTS. Metabolic syndrome and obesity are important factors that have been known to aggravate BPH/LUTS [6–8]. Research on the relationship between obesity and BPH/LUTS has mainly focused on anthropometric parameters, such as body weight, body mass index (BMI), and waist circumference [6–8]. However, anthropometric parameters are the sum of body composition, which makes it difficult to determine the effect of separate body composition on BPH/LUTS. There are many methods, such as; bioelectrical impedance analysis (BIA), dual-energy X-ray absorptiometry, CT, and MRI, for analyzing body composition. Among them, the use of BIA in medical checkups and daily settings has been increasing, because BIA is non-invasive and more convenient than other methods.

Percent body fat (PBF) is the percentage of fat in the total body weight. Recently, many people have become interested in PBF, working hard to maintain their PBF below the normal range. In addition, in many academic fields, efforts to investigate the effects of fat composition on various pathogeneses are in progress. For example, PBF is associated with the development of chronic kidney disease and cardiovascular disease [9, 10]. However, only a few studies utilized fat compositions for predicting BPH/LUTS until now. This study aimed to investigate the usefulness of PBF in predicting BPH/LUTS.

Materials and methods

Study population

The study protocol was approved by the Institutional Review Board of our institution (IRB No. 10-2022-108). Eligible subjects were men aged 40 years or older who underwent medical health checkups at our institution from 2014 to 2022. Among them, 844 men who underwent BIA, prostate ultrasonography, blood test for the serum level of prostate-specific antigen (PSA), and filled out the international prostate symptom score (IPSS) questionnaire were included. The basic demographics of the subjects (age, BMI, and waist circumference), past medical history (history of hypertension, diabetes mellitus, and dyslipidemia), and metabolic equivalent of task (MET) were collected. The cutoff values for the statuses of overweight and obesity, according to BMI (kg/m^2), were 25 and 30 respectively [11]. Central obesity was defined as a waist circumference $\geq$ 90 cm [12]. MET was obtained after log transformation because of its skewed deviated distribution (Supplementary Fig. 1) [13]. The body composition factors recorded from BIA included PBF (%), skeletal muscle mass (kg), appendicular skeletal muscle mass (kg), fat-free mass (kg), visceral fat area (m^2), soft lean mass (kg), and appendicular skeletal mass index (ASMI) (kg/m^2). Prostate volume was calculated using transrectal ultrasound. BPH was defined as a prostate volume of 30 cc or more. An IPSS score $\geq$ 20 was defined as severe LUTS [5].

Statistical analysis

Subjects were divided into two groups according to their quartiles of PBF: the normal PBF group (first to third quartile (PBF < 27.9%)) and the high PBF group (fourth quartile (PBF $\geq$ 27.9%). The mean value and standard deviation (SD) for continuous variables between groups were compared using the Student's t-test. The frequencies of categorical variables between the groups were compared using the chi-square test. After a comparison of body composition between the two groups, the ASMI was chosen as the other body composition to focus on. ASMI is an index widely used for defining sarcopenia, which is ASMI < 7.0 kg/m2 [14]. There was no statistically significant difference between the two groups regarding ASMI ($p = 0.094$), and ASMI had a low correlation with PBF (Pearson's correlation coefficient = 0.014). In addition, the ASMI was the only variable corrected by the subject's height among the variables of muscle composition. Univariate and multivariate logistic regression analyses were performed to examine risk factors for BPH and severe LUTS. Variables with a p-value < 0.2 after univariate analysis were included in the multivariate analysis. Statistical significance was set at $p < 0.05$, and all statistical analyses were performed using IBM SPSS Statistics version 26.

Results

The mean age of the total subjects was 55.7 ± 8.1 years, and the mean PBF was 24.4 ± 5.6%. Table 1 shows the characteristics of the study population divided into PBF quartiles. As shown, 629 (74.5%) and 215 (25.5%) men belonged to the normal PBF group and high PBF groups, respectively. Subjects in the high PBF group had a higher prevalence of factors of metabolic syndromes: history of hypertension (53.6 vs. 82.8%, $p < 0.001$), diabetes mellitus (36.1 vs. 46%, $p < 0.010$), dyslipidemia (38.3 vs. 62.8%, $p < 0.001$), and central obesity (23.8 vs. 70.7%, $p < 0.001$). Prostate volume (25.2 ± 8.4 cc vs. 27.3 ± 9.0 cc, $p = 0.002$) and percentage of BPH (22.9 vs. 32.1%, $p = 0.007$) were greater in the high PBF group. Men in the high PBF group tend to have more severe LUTS (8.9 vs. 14.9%, $p = 0.013$). However, no statistically significant difference was found in age, MET, and PSA levels.

A comparison of body composition between the two groups is also shown in Table 1. The factors with a statistically significant difference between groups were body

Table 1 Characteristics of the study population

	PBF < 27.9%	PBF ≥ 27.9%	*p*
Number of subjects, n (%)	629 (74.5%)	215 (25.5%)	
Age, years (mean ± SD)	55.48 ± 8.3	56.52 ± 9.2	0.121
Hypertension, n (%)	337 (53.6)	178 (82.8)	< 0.001
Diabetes mellitus, n (%)	227 (36.1)	99 (46.0)	0.010
Dyslipidemia, n (%)	241 (38.3)	135 (62.8)	< 0.001
Central obesity, n (%)	150 (23.8)	152 (70.7)	< 0.001
MET, ln (mean ± SD)	5.73 ± 3.0	5.42 ± 3.1	0.186
Prostate volume, cc (mean ± SD)	25.2 ± 8.4	27.3 ± 9.0	0.002
Transition zone volume, cc (mean ± SD)	8.4 ± 5.6	10.0 ± 6.5	0.001
BPH (Prostate volume ≥ 30 cc), n (%)	144 (22.9)	69 (32.1)	0.007
IPSS, n (%)			0.033
Mild	343 (54.5)	116 (54)	
Moderate	230 (36.6)	67 (31.2)	
Severe	56 (8.9)	32 (14.9)	
PSA, ng/mL (mean ± SD)	1.7 ± 1.8	1.9 ± 3.1	0.320
BMI			< 0.001
< 25, n (%)	468 (74.4)	47 (21.9)	
> 30 and 25 ≤, n (%)	159 (25.3)	140 (65.1)	
≥ 30, n (%)	2 (0.3)	28 (13)	
Percent body fat (%), mean ± SD	21.90 ± 4.0	31.51 ± 2.7	< 0.001
Appendicular skeletal muscle mass index (kg/m^2), mean ± SD	7.76 ± 0.6	7.86 ± 0.8	0.094
Visceral fat area (m^2), mean ± SD	92.7 ± 24.7	127.3 ± 29.0	< 0.001
Body fat mass (kg), mean ± SD	15.19 ± 3.9	24.42 ± 4.7	< 0.001
Fat free mass (kg), mean ± SD	53.4 ± 6.2	52.8 ± 7.3	0.165
Skeletal muscle mass (kg), mean ± SD	29.9 ± 3.7	29.5 ± 4.4	0.117
Appendicular skeletal muscle mass (kg), mean ± SD	22.79 ± 3.0	22.48 ± 3.5	0.201
Soft lean mass (kg), mean ± SD	50.6 ± 5.8	49.9 ± 6.9	0.134

P probability value, *PBF* percent body fat, *MET* metabolic equivalent of task, *BMI* body mass index, *PSA* prostate specific antigen, *IPSS* international prostate symptom score; *n* number of subjects

compositions related to obesity, which are visceral fat area (92.7 ± 24.7m^2 vs. 127.3 ± 29.0m^2, $p < 0.001$), and body fat mass (15.2 ± 3.9 kg vs. 24.4 ± 4.7 kg, $p < 0.001$). There were no differences in the ASMI, fat-free mass, skeletal muscle mass, appendicular skeletal muscle mass, or soft lean mass between the groups.

Table 2 shows the results of the multivariate logistic regression analysis of the risk factors for BPH. After univariate analysis, old age (odds ratio [OR] = 1.054, $p < 0.001$), history of hypertension (OR = 1.638, $p = 0.004$), central obesity (OR = 1.446, $p = 0.023$), and high PBF (OR = 1.147, $p = 0.028$) were associated with BPH. Multivariate analysis showed that old age (OR = 1.066, $p < 0.001$), higher ASMI (OR = 1.544, $p = 0.001$), and high PBF (OR = 1.455, $p = 0.037$) were independent risk factors for BPH. Table 3 shows the risk factors of severe LUTS. Univariate analysis revealed that old age (OR = 1.042, $p = 0.002$), large prostate volume (OR = 1.035, $p = 0.002$), low ASMI (OR = 0.689, $p = 0.024$), and high PBF (OR = 1.789, $p = 0.014$) were associated with severe LUTS. Multivariate analysis revealed that larger prostate volume (OR = 1.035, $p = 0.002$) and high PBF (OR = 1.715, $p = 0.025$) were risk factors for severe LUTS. However, a greater ASMI had a protective effect against severe LUTS (OR = 0.654, $p = 0.011$).

Discussion

Understanding the risk factors of BPH/LUTS is important because it allows us to understand the pathogenesis of the disease and to investigate preventive methods. Our study focused on PBF calculated by BIA, enabling a more direct investigation of the effect of obesity on BPH. We showed that PBF is more useful than waist circumference, BMI, and other components of metabolic syndrome when predicting BPH/LUTS. In addition. we showed that a higher ASMI was associated with BPH but had a protective effect on severe LUTS.

Increased prostate volume is generally associated with more bothersome LUTS, but only high PBF, not increased

Table 2 Risk factors for BPH in the study population (prostate volume ≥ 30 cc)

	Univariate		Multivariate	
	OR (95% CI)	*p*	OR (95% CI)	*p*
Age (continuous)	1.054 (1.034–1.074)	< 0.001	1.066 (1.045–1.089)	< 0.001
Hypertension (yes vs no)	1.638 (1.175–2.282)	0.004		
Diabetes mellitus (yes vs no)	1.019 (0.741–1.402)	0.906		
Dyslipidemia (yes vs no)	1.298 (0.885–1.903)	0.181		
Central obesity (yes vs no)	1.446 (1.052–1.988)	0.023		
MET, ln (continuous)	0.981 (0.932–1.032)	0.450		
BMI (kg/m^2)				
< 25	Reference			
> 30 and 25 ≤	1.412 (1.021–1.952)	0.037		
≥ 30	1.720 (0.783–3.777)	0.177		
ASMI (kg/m^2) (continuous)	1.205 (0.964–1.507)	0.101	1.544 (1.209–1.971)	0.001
PBF (%)				
< 27.9%	Reference		Reference	
≥ 27.9%	1.147 (1.015–1.295)	0.028	1.455 (1.023–2.071)	0.037

P probability value, *PBF* percent body fat, *OD* odds ratio, *CI* confidence interval, *ASMI* appendicular skeletal muscle mass index, *MET* metabolic equivalent of task, *BMI* body mass index

Table 3 Risk factors for severe LUTS

	Univariate		Multivariate	
	OR (95% CI)	*p*	OR (95% CI)	*p*
Age (continuous)	1.042 (1.015–1.069)	0.002		
Hypertension (yes vs no)	1.196 (0.754–1.897)	0.446		
Diabetes mellitus (yes vs no)	1.235 (0.790–1.930)	0.354		
Dyslipidemia (yes vs no)	1.214 (0.780–1.888)	0.39		
Central obesity (yes vs no)	1.100 (0.877–1.379)	0.41		
MET, ln (continuous)	0.936 (0.875–1.002)	0.059		
BMI (kg/m^2)				
< 25	Reference			
> 30 and 25 ≤	1.008 (0.631–1.610)	0.972		
≥ 30	1.341 (0.451–3.990)	0.598		
Prostate volume (continuous)	1.035 (1.013–1.058)	0.002	1.035 (1.013–1.058)	0.002
ASMI (kg/m^2) (continuous)	0.689 (0.499–0.952)	0.024	0.654 (0.472–0.906)	0.011
PBF (%)				
< 27.9%	Reference		Reference	
≥ 27.9%	1.789 (1.124–2.849)	0.014	1.715 (1.068–2.751)	0.025

P probability value, *PBF* percent body fat, *OD* odds ratio, CI confidence interval, *ASMI* appendicular skeletal muscle mass index, *MET* metabolic equivalent of task, *BMI* body mass index

ASMI, is a risk factor for severe LUTS. We suggest that high PBF and increased ASMI play a role in prostate enlargement in distinct compartments of the prostate gland, and the compartment related to the high PBF is important in LUTS formation. Prostate enlargement with high PBF and ASMI can be explained by the interaction between hormones and hormone receptors. There are androgen receptors (AR) and two types of estrogen receptors (ERs) in the prostate gland: ER-α and ER-β. AR and ER-α mediate prostate cell proliferation, whereas ER-β inhibits prostate cell proliferation [15, 16]. BPH development in the high PBF group can be explained by the increased activity of aromatase, which is an enzyme expressed in adipocytes responsible for converting testosterone into estradiol. Increased estrogen upregulates ER-α, and enlarges the prostate [15]. Prostate enlargement in men with a higher ASMI can be explained by the increased activity of testosterone, which activates muscle protein synthesis and facilitates prostate cell growth [16]. We suggest that androgen and estrogen contribute to the enlargement of different segments of the prostate. This hypothesis is supported by

the results of previous studies [17–19]. The prostate has two histologic components, which are the fibromuscular stroma and the epithelial component [18]. The AR is mainly located in the epithelial component and in a smaller volume in the stroma [19]. The prostate's peripheral zone has a higher percentage of epithelial components, whereas its transition zone is composed of more stroma [18]. Thus, testosterone is presumed to play a role in the enlargement of the peripheral zone. We suggest that the transition zone volume is mainly influenced by estrogen. Ozden et al. [8] showed that the median annual total prostate growth rate (1.0 ml/year) and median annual transition zone growth rate (1.25 ml/yr) were faster in BPH patients with metabolic syndrome, supporting our hypothesis that estrogen and ER play a role in the growth of the transition zone. Also, the transition zone might play an important role in the development of LUTS, but further well-designed studies are needed.

High PBF and low ASMI were risk factors for severe LUTS, suggesting that hypogonadism plays a key role in LUTS. Increased levels of estrogen in men with high PBF put negative feedback on the hypothalamus-pituitary-testis axis, resulting in decreased secretion of gonadotropin-releasing hormone and luteinizing hormone. This negative feedback causes hypogonadism in men with obesity [20]. In addition, hypogonadism and low ASMI were also related [16]. It is known that testosterone stimulates nitric oxide formation, which plays a role in the dilation of the urethra and bladder neck, and this supports our idea of an association between hypogonadism and bothersome LUTS [21].

Lifestyle modifications may relieve BPH/LUTS, and maintaining PBF in the normal range might prevent the development of BPH/LUTS in the general population. Lowering PBF in individuals with high PBF might improve LUTS and reduce prostate volume by relieving hypogonadism and reducing the action of estrogen. According to Jedamzik et al. [22], testosterone levels increase in obese hypogonadal men after weight loss, supporting our idea that lowering PBF to the normal range in the high PBF group might improve LUTS. Moreover, we suggest that active physical activity and increased ASMI might improve LUTS. Parsons et al. [23] conducted a study and showed that physical activity lowered the risk of BPH/LUTS. Increased physical activity seemed to lower the risk of severe LUTS in our study, but ASMI had a greater protective effect against severe LUTS than MET did. This can be explained by the high correlation between ASMI and MET and the limitations of MET, which were acquired by the questionnaire. Both increased MET and ASMI seem to lower LUTS through a similar mechanism, but ASMI by BIA is more useful in predicting LUTS than MET. Our study provides a clinical basis for educating obese patients to lower their PBF to reduce LUTS. Patients will have a more intuitive target for PBF to relieve LUTS.

This study has some limitations. First, this was a cross-sectional study, and further prospective studies investigating changes in prostate volume/LUTS after modification of body composition are necessary. Second, the reliability and validity of BIA remain controversial. Although some minor systemic bias exists, BIA is a clinically feasible method for assessing body composition, and its feasibility has been supported by many studies [24–26]. However, our study had some strengths. First, we focused on the PBF calculated by BIA, which has not been researched regarding BPH/LUTS despite its many attributes: ready availability, convenience, and non-invasiveness. Second, little research has been conducted on the association between muscle mass and BPH/LUTS. Z Qin. et al. [27] explained that low lean mass aggravates male LUTS; however, to our knowledge, no other studies have investigated the impact of body composition derived from BIA and muscle mass on BPH/LUTS, and our study may be the first to unveil the association between muscle mass and prostate volume. Third, we showed that PBF has a higher predictive value for metabolic syndrome and that PBF can be used for consultation with the general population with metabolic syndrome. PBF can present a more direct cutoff value for reducing BPH/LUTS in patients with BPH and preventing the development of BPH/LUTS in the general population. Our study is novel in that we introduced body composition as a risk factor for BPH/LUTS, and used BIA as a tool for measuring body composition.

Conclusion

In conclusion, this study asserts that maintaining a fit body will help reduce male LUTS and suggests that PBF is a more useful predictor of BPH/LUTS than previous anthropometric parameters and metabolic syndrome. We believe that lowering PBF may help reduce LUTS and prevent the development of BPH/LUTS in the general population. However, further well-designed studies are required to confirm our findings. As there is an interest in modifying body composition among the general population and the use of BIA due to its convenience increases, studies on the relationship between body composition and BPH/LUTS, especially those using BIA, will likely be researched more actively. As a pioneering study on body composition and BPH/LUTS, we believe that our study provides a theoretical background for future studies.

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Data collection or management, Protocol/project development, JBJ: Data collection or management, Protocol/project development.

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Data availability Data analyzed during this study are not available due to the confidentiality of the subjects' information.

Declarations

Conflict of interest All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

Ethical approval This research study was conducted from data obtained for clinical purposes. We consulted extensively with the IRB who determined that our study did not need ethical approval. An IRB official waiver of ethical approval was granted from the Institutional Review Board of our institution (IRB No. 10-2022-108).

Research involving human participants and/or animals None.

Informed consent This is an observational study and therefore consent was waived by the IRB confirming that no ethical approval is required.

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