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The Impact of Body Mass Index and Benign Prostatic Hyperplasia on Bone Health of Middle-Aged and Older Men

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ABSTRACT

Objective: Osteoporosis significantly affects older adults by reducing bone mass and increasing fracture risk, thereby impacting morbidity and mortality. This study aimed to investigate the relationship between bone mineral density (BMD), body mass index (BMI), and trabecular bone score (TBS) among middle-aged and older men with or without benign prostatic hyperplasia (BPH).

Methods: A retrospective study was conducted using health examination data from male participants aged 50–98 years collected at a regional hospital in southern Taiwan. Simple and multiple linear regression analyses were employed to examine the relationships between TBS and the independent variables. A total of 3714 middle-aged and older men were included in the analysis.

Results: Findings indicated that higher BMI was associated with greater BMD; however, the relationship with TBS suggested potential bone quality degradation in cases of underweight and obesity. Multiple linear regression analysis demonstrated that age, waist circumference, BMD, underweight status, and obesity were significantly associated with TBS.

Conclusion: This study revealed the associative relationship between BMI and bone health: higher BMI was associated with increased bone density but also related to a decline in bone quality as measured by TBS, particularly in cases of obesity. These results emphasized the importance of managing BMI to optimize both bone density and quality, especially in middle-aged and older men with or at risk of BPH.

1 | Introduction

Osteoporosis is a prevalent condition among older individuals and is characterized by decreased bone mass and bone mineral density (BMD). This leads to an increased risk of fractures, which can cause significant disability, decreased quality of life, and mortality [1–3]. Health professionals traditionally diagnose osteoporosis by using dual-energy x-ray absorptiometry (DXA) to measure BMD. However, recent advancements highlight the importance of the trabecular bone score (TBS) in assessing bone

quality and fracture risk [4–6]. TBS offers insights into the bone microarchitecture, providing a better understanding of bone health beyond BMD alone [5].

The relationship between body mass index (BMI) and osteoporosis introduces another layer of complexity. A high BMI is often considered a protective factor against osteoporosis due to the mechanical load on bones, promoting bone formation [7, 8]. However, the intricate balance between metabolic health and the development of age-related conditions, such as osteoporosis and benign prostatic hyperplasia (BPH), is noteworthy. Characterized by nonmalignant enlargement of the prostate gland, BPH significantly impacts the quality of life in aging males, leading to a variety of urinary symptoms and potential complications such as urinary retention and kidney damage [9, 10].

Emerging research suggests that lifestyle interventions aimed at optimizing BMI could mitigate risks associated with both osteoporosis and BPH [11, 12]. A balanced BMI supports not only a healthier prostate but also contributes to better bone microarchitecture, as reflected by TBS, alongside traditional BMD measurements. In addition, the interplay between metabolic syndrome, cardiovascular health, and these conditions cannot be overlooked. Studies have shown that metabolic syndrome is a common thread linking the risk factors for both osteoporosis and BPH [13, 14], with cardiovascular health playing a pivotal role in mediating these relationships [15]. Furthermore, the role of diet, physical activity, and overall lifestyle choices in managing these conditions has been increasingly recognized, offering potential for preventative strategies [16–18].

Pharmacological interventions also play a critical role, with medications for BPH potentially affecting bone health and vice versa [19]. Selective alpha-blockers, 5-alpha reductase inhibitors, and phosphodiesterase type 5 inhibitors used in BPH management have been scrutinized for their impact on bone metabolism and overall health [15, 20, 21], necessitating a careful approach to treatment selection. Selective alpha-blockers effectively relieve lower urinary tract symptoms but may indirectly impact bone health by increasing fall and fracture risk, especially in older adults with osteoporosis [22, 23]. Similarly, 5-alpha reductase inhibitors, by altering hormonal balance through the reduction of dihydrotestosterone, may affect bone density [24], while phosphodiesterase type 5 inhibitors exhibit anti-inflammatory properties with uncertain effects on bone remodeling [25].

This study aims to investigate the association between osteoporosis and BPH, both of which significantly impact the health and quality of life of middle-aged and older men. Furthermore, it examines the correlation between BMI and its effects on bone density to assess the likelihood of fracture risk. The findings are intended to inform public health and health policy considerations.

2 | Material and Methods

This was a retrospective analysis which collected data from individuals who underwent health examinations at a preventive medicine center in a regional hospital in southern Taiwan. The collection period spanned from June 2014 to December 2020, focusing on male participants aged between 50 and 98 years. Individuals were excluded if they had cancer, incomplete laboratory data, or if the BMD could not be measured in any one region. Participants who had received repeated examinations were also omitted. Ultimately, 3714 participants were included in this study. This study protocol was reviewed and approved by the Institutional Review Board of the study hospital (IRB# B11102008).

2.1 | Laboratory Data

This study selected factors related to BMD, including physical examination parameters such as age, height, weight, BMI, hip circumference, systolic blood pressure, and diastolic blood pressure. Laboratory data collected included alkaline phosphatase, serum urea nitrogen, creatinine, glomerular filtration rate, fasting blood glucose, total cholesterol, triglycerides, and uric acid. BMD analysis focused on the lumbar spine and bilateral hip BMD, along with TBS analysis.

2.2 | Bone Mineral Density

In this study, BMD of the lumbar spine (L1-L4) and bilateral hips (femoral neck and total hip) regions was analyzed using the DXA (Discovery Wi DXA system, Hologic Inc., Marlboro, MA, USA). BMD values were expressed as absolute values in grams per square centimeter (g/cm^2). Daily scan quality was ensured by passing a standard phantom scan before conducting the study. All radiologic technologists operating the equipment and physicians interpreting the results had received international certification in bone densitometry after completing relevant courses and exams. Absolute BMD values for all patients were calculated. The same DXA equipment was used for all participants to ensure the accuracy of the study comparisons.

2.3 | Trabecular Bone Score

The TBS of the lumbar spine was assessed in the same regions as the BMD, calculating the average value of individual vertebral measurements from the first to the fourth lumbar vertebra. All TBS measurements were performed using the TBS iNsight software version 3.0.2.0 (Headquarters Medimaps Group SA, Geneva, Switzerland) following BMD analysis.

2.4 | Prostate Hyperplasia Ultrasound Confirmation

Prostate size was measured by clinical physicians using color Doppler sonography (Hitachi Aloka, Prosound α7, Hitachi Inc., Tokyo, Japan). All patients were scanned using the same protocol to ensure accurate comparisons. Patients were classified into either a normal group or a BPH group.

2.5 | Statistical Analysis

Statistical analyses were conducted using the IBM SPSS for Windows version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and were analyzed using analysis of variance to compare differences across four BMI categories (underweight, normal, overweight, and obesity) and between two groups of patients, those with or without BPH. Categorical variables were represented as numbers (percentage) and were compared using the chi-square test. To prepare for linear regression analysis, continuous variables underwent log-transformation, including height, weight, waist circumference, systolic/diastolic blood pressure, alkaline phosphatase (ALP), estimated glomerular filtration rate (eGFR), pre-meal glucose (GLU AC), total cholesterol (TCH), triglycerides (TG), and uric acid (UA). In addition, the relationship between TBS and demographic, clinical, and BMD data was evaluated using simple and multiple linear regression models. Trend plots were utilized to investigate the associations between BPH, BMI, and TBS or BMD, enhancing the analysis with a 3D plot to assess the complex interrelations among BPH, BMI, TBS, and BMD.

3 | Results

3.1 | Comparing the Difference of TBS and BMD Across BMI Categories

In this study, 4697 male participants underwent health examinations during the collection period. After excluding those who did not meet the study criteria, 3714 patients (with a mean age of 61.2 ± 7.6 years) were included in the analysis. Based on the results of abdominal ultrasounds, participants were further categorized into two groups: those with BPH (n = 2485), and those without BPH (n = 1229). These groups were then subdivided according to their BMI into four categories: underweight (BMI < 18.5), normal (BMI 18.5–23.9), overweight (BMI 24.0–26.9), and obese (BMI ≥ 27) (Figure 1). Significant variations in demographic characteristics and clinical information were observed among the four BMI categories, except for height, which remained consistent across the groups. No notable differences were identified in patients with or without BPH across the BMI categories. However, significant disparities in BMD, T-scores, and TBS were found among the BMI groups, as detailed in Table 1.

3.2 | Comparing BMD and TBS Between Patients With and Without BPH Across Different BMI Categories

Minimal or no significant variation in BMD, T-scores, and TBS was observed between patients with or without BPH across the four BMI categories, as detailed in Table 2. This suggests that BPH's presence did not markedly affect these bone health indicators across different body mass indexes. However, trend analyses between BPH, BMI, and either TBS or BMD revealed more complex relationships. The trend plots indicate a positive correlation between BMI and BMD, suggesting that as BMI increases, so does BMD (Figure 2A). Conversely, the relationship

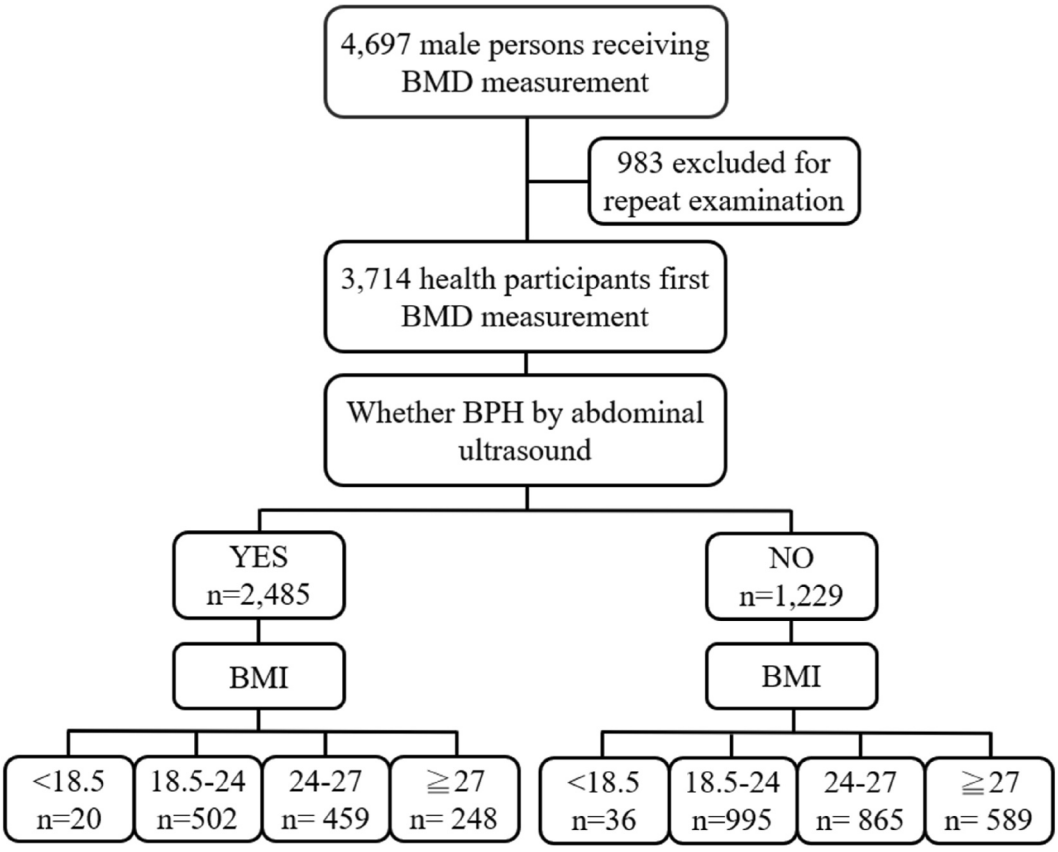

FIGURE 1 | Flowchart illustrating the inclusion and exclusion criteria for selecting study participants, highlighting key variables such as BMD, BMI, and BPH.

TABLE 1 | Basic demographic clinical characteristics stratified by BMI categories.

	BMI category						
	(A) < 18.5 (underweight)	**(B) 18.5–23.9 (normal)**	**(C) 24.0–26.9 (overweight)**	**(*D*) ≥ 27.00 (obesity)**	**Total**	***p***	**Post-hoc test**
N	56	1497	1324	837	3714		
Demographic characteristics							
Age, years (mean ± SD)	63.6 ± 8.65	62.06 ± 7.7	60.99 ± 7.59	60.04 ± 7.34	61.25 ± 7.64	< 0.001	*A = B = C > D***
50–59	21 (1.24)	621 (36.62)	626 (36.91)	428 (25.24)	1696 (100)	< 0.001	
60–69	24 (1.66)	609 (42.06)	501 (34.6)	314 (21.69)	1448 (100)		
70–79	9 (1.75)	240 (46.6)	181 (35.15)	85 (16.5)	515 (100)		
≥ 80	2 (3.64)	27 (49.09)	16 (29.09)	10 (18.18)	55 (100)		
Height (cm)	167.87 ± 4.94	166.45 ± 5.75	166.43 ± 5.86	166.66 ± 5.92	166.51 ± 5.82	0.254	
Weight (kg)	49.64 ± 3.96	61.52 ± 5.79	70.42 ± 5.53	80.93 ± 7.92	68.88 ± 9.99	< 0.001	*D > C > B > A****
BMI (kg/m^2)	17.59 ± 0.75	22.17 ± 1.36	25.39 ± 0.84	29.11 ± 2.15	24.81 ± 3.15	< 0.001	*D > C > B > A****
Waist circumference (cm)	70.72 ± 3.85	80.94 ± 5.26	87.61 ± 4.62	95.69 ± 6.6	86.49 ± 8.03	< 0.001	*D > C > B > A****
Clinical information							
SBP (mmHg)	124.57 ± 24.48	128.78 ± 19.6	133.87 ± 18.86	136.23 ± 19.31	132.21 ± 19.61	< 0.001	*A = B > C = D***
DBP (mmHg)	71.7 ± 12.31	77.12 ± 10.52	80.06 ± 10.71	81.59 ± 11.13	79.1 ± 10.94	< 0.001	*D > C > B > A****
ALP (IU/L)	80.11 ± 20.16	78.19 ± 23.26	74.76 ± 21.8	73.12 ± 22.99	75.85 ± 22.73	< 0.001	*A = B > C** = D****
BUN (mg/dL)	11.96 ± 3.47	12.13 ± 4.03	12.57 ± 4.17	12.78 ± 4.18	12.43 ± 4.12	0.001	*A = B < C = D***
Cr (mg/dL)	0.94 ± 0.14	1.02 ± 0.33	1.07 ± 0.45	1.07 ± 0.23	1.05 ± 0.36	< 0.001	*A = B > C = D***
eGFR ($mL/min/1.73\ m^2$)	88.77 ± 16.34	82.93 ± 16.5	79.32 ± 16.92	78.41 ± 16.29	80.71 ± 16.74	< 0.001	*A = B > C = D****
GluAC (mg/dL)	98.61 ± 12.95	105.84 ± 20.93	110.82 ± 27.62	114.09 ± 26.8	109.37 ± 25	< 0.001	*A = B < C < D****
TCH (mg/dL)	161.32 ± 29.17	177.6 ± 35.36	180.38 ± 38.07	179.29 ± 38.22	178.73 ± 36.98	0.001	*A < B = C = D***
TG (mg/dL)	70.54 ± 44.28	109.52 ± 74.4	130.31 ± 70.4	156.43 ± 125.15	126.92 ± 89	< 0.001	*D > C > B > A***
SONO							
Normal	36 (1.45)	995 (40.04)	865 (34.81)	589 (23.7)	2485 (100)	0.094	
BPH	20 (1.63)	502 (40.85)	459 (37.35)	248 (20.18)	1229 (100)		
Bone mineral density data							
Lspine BMD (g/cm^2)	0.83 ± 0.14	0.95 ± 0.14	1.00 ± 0.15	1.05 ± 0.16	0.99 ± 0.16	< 0.001	*D > C > B > A***
Lspine T-score	−1.74 ± 1.19	−0.73 ± 1.24	−0.25 ± 1.27	0.14 ± 1.31	−0.38 ± 1.32	< 0.001	*D > C > B > A***
TBS (L1-4)	1.35 ± 0.08	1.39 ± 0.08	1.38 ± 0.08	1.35 ± 0.1	1.38 ± 0.09	< 0.001	*A = D < B = C****

Note: Values are presented as mean ± SD (95% CI). Statistical significance tested using ANOVA with Tukey's HSD post-hoc tests.
Abbreviations: ALP, alkaline phosphatase; BUN, blood urea nitrogen; Cr, creatinine; DBP, diastolic blood pressure; eGFR, estimated Glomerular Filtration Rate; GluAC, Glucose (Ante Cibum); SBP, systolic blood pressure; TCH, Total Cholesterol; TG, Triglyceride.
Post-hoc test: * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

TABLE 2 | Comparing the BMD and TBS with or without BPH stratified by BMI categories.

BMI category	< 18.5 (underweight)			18.5–23.9 (normal)			24.0–26.9 (overweight)			≥ 27.00 (obesity)			Total		
BPH	No	Yes	*p*	No	Yes	*p*	No	Yes	*p*	No	Yes	*p*	No	Yes	*p*
N	36	20		995	502		865	459		589	248		2485	1229	
TBS	1.35 ± 0.08	1.34 ± 0.08	0.644	1.39 ± 0.08	1.38 ± 0.08	0.139	1.38 ± 0.08	1.37 ± 0.09	0.045	1.36 ± 0.10	1.35 ± 0.10	0.420	1.38 ± 0.09	1.37 ± 0.09	0.027
BMD	0.83 ± 0.14	0.81 ± 0.14	0.542	0.94 ± 0.13	0.95 ± 0.15	0.044	0.99 ± 0.14	1.00 ± 0.15	0.114	1.04 ± 0.16	1.05 ± 1.55	0.623	0.98 ± 0.15	0.99 ± 0.16	0.068
T-score	−1.68 ± 1.20	−1.86 ± 1.21	0.811	−0.77 ± 1.19	−0.65 ± 1.33	0.094	−0.28 ± 1.25	−0.20 ± 1.32	0.299	0.14 ± 1.31	0.16 ± 1.33	0.865	−0.40 ± 1.30	−0.34 ± 1.37	0.239

Note: *p*-values are derived from independent *t*-tests for comparisons between BPH and non-BPH groups within each BMI category.

between BMI and TBS varies depending on the specific BMI range. Specifically, when BMI was between 17 and 19, there was a slight increase in TBS, which then entered a plateau phase for BMIs between 20 and 25. Notably, once BMI exceeded 25, TBS began to decline rapidly, dropping below 1.35 when BMI surpassed 29 (Figure 2B). These findings highlight the intricate interplay between BMI, BPH, and bone health metrics such as TBS and BMD. While BPH's presence did not significantly alter these metrics across BMI groups, BMI itself influenced bone density and quality, underscoring the importance of considering body composition in the assessment and management of bone health, especially in individuals with or at risk of developing BPH.

3.3 | The Influence Factors of TBS in Simple and Multiple Linear Regression Analyses

In the simple linear regression analysis, several factors significantly affected TBS, including age, height, weight, waist circumference, systolic/diastolic blood pressure, ALP, triglycerides, and BMD, all showing statistical significance. Furthermore, TBS was significantly influenced by being underweight or obese, with normal BMI serving as the reference category. However, upon adjusting for other variables in multiple linear regression, only age, waist circumference, BMD, and BMI remained significant predictors of TBS (Table 3).

The relationship between BPH, BMI, TBS, and BMD was further explored through a 3D plot, revealing a positive relationship between BMI and BMD as well as between BMD and TBS. Conversely, a more complex relationship was observed between BMI and TBS. This complex interplay suggested that while higher BMI was associated with increased BMD, it might have negatively impacted TBS, highlighting the multifaceted nature of these relationships (Figure 3).

4 | Discussion

This study investigated the intricate relationships between BMI, BMD, and TBS. We observed a clear pattern: By categorizing BMI into four categories—underweight, normal, overweight, and obese—we further observed that BMD showed a linear increase across these categories, suggesting that higher BMI correlated with increased bone density. However, the TBS improved from underweight to normal, then declined in overweight individuals and reached its lowest in the obese category. This pattern suggested a critical insight: while a higher BMI may have led to denser bones, it did not necessarily imply improved bone strength, As BMI increased, BMD rose, indicating denser bones at higher BMI, but TBS declined, reflecting compromised bone quality, showing a trade-off between bone density and quality at higher BMI.

BPH further complicated BMI's effects on bone health. Trends between BMI and bone health metrics in individuals with BPH closely mirrored those observed in the general population, with BMD increasing and TBS decreasing alongside rising BMI. However, it was noteworthy that TBS values in the BPH group

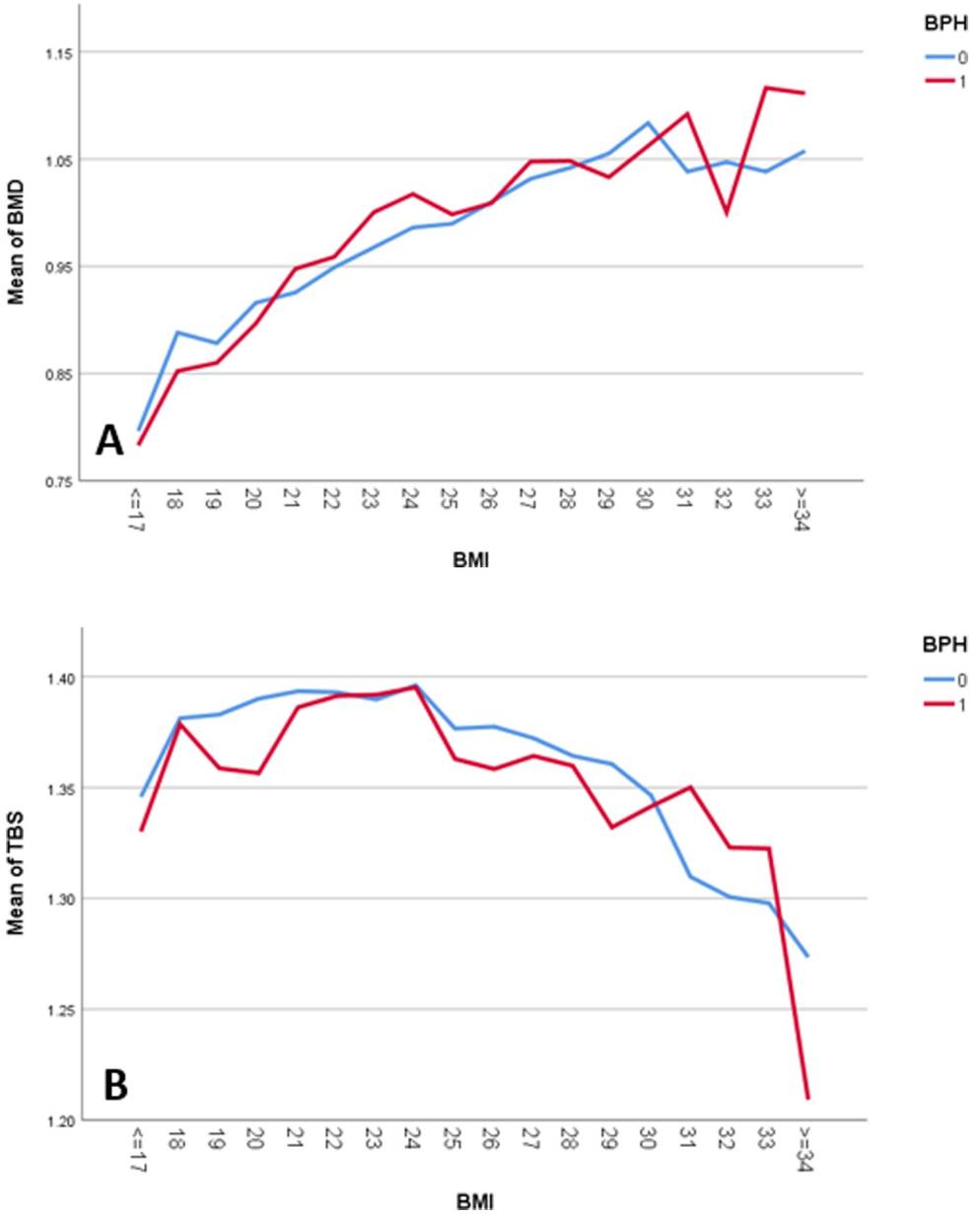

FIGURE 2 | Trend plot showing variations in (A) BMD and (B) TBS across BMI categories, with normal prostate (blue line, coded as 0) and BPH (red line, coded as 1).

were generally lower than those in the normal BMI group. This suggested that BPH might have contributed to an accelerated decline in bone quality associated with higher BMI, signaling that individuals with BPH might have faced a compounded risk of compromised bone microarchitecture.

The intricate relationship between BPH and bone health extends beyond simple anatomical considerations, delving into the complex interplay of metabolic and hormonal interactions. Both BPH and bone health are significantly influenced by hormonal factors, particularly androgens and estrogens, which play critical roles in bone metabolism [26–28]. These hormones regulate not only prostate growth but also bone density and structure, maintaining a balance essential for overall health [29]. In individuals with BPH, this balance might have been disrupted, leading to an underlying hormonal imbalance that could have adversely affected bone microarchitecture. The manifestation of lower TBS in the BPH group compared with those without BPH, suggested a more profound impact of these hormonal imbalances on bone quality, indicative of systemic alterations influenced by hormonal dynamics, potentially leading to compromised bone integrity and strength.

Comparisons with recent studies in Asian populations suggest that individuals in these regions often exhibit lower average TBS values compared with their Western counterparts [30]. These disparities may stem from a combination of factors, including differences in body composition, dietary habits, genetic predispositions, and potentially unique environmental influences [30, 31]. Such variations emphasize the need for tailored, region-specific approaches when investigating the

TABLE 3 | Simple and multiple linear regression analyses of TBS.

	Simple linear regression				Multiple linear regression			
		95% CI				95% CI		
	Coefficients (B)	Lower	Upper	*p*	Coefficients (B)	Lower	Upper	*p*
Age (year)	−0.19	0.00	0.00	< 0.001	−0.21	0.00	0.00	< 0.001
Height (cm)	0.16	0.31	0.47	< 0.001				
Weight (kg)	−0.04	−0.05	−0.01	0.006				
Waist circumference (cm)	−0.22	−0.24	−0.18	< 0.001	−0.34	−0.36	−0.30	< 0.001
SBP (mmHg)	−0.10	−0.08	−0.04	< 0.001				
DBP (mmHg)	−0.04	−0.05	−0.01	0.008				
Without BPH	Reference							
With BPH	−0.04	−0.01	0.00	0.027	−0.00	−0.04	0.04	0.990
ALP (IU/L)	−0.05	−0.03	−0.01	0.002				
eGFR (mL/min/1.73 m^2)	0.01	−0.01	0.01	0.697				
GLUAC (mg/dL)	−0.03	−0.03	0.00	0.061				
TCH (mg/dL)	0.01	−0.01	0.02	0.611				
TG (mg/dL)	−0.04	−0.01	0.00	0.014				
UA (mg/dL)	0.00	−0.01	0.01	0.844				
BMD	0.51	0.275	0.303	0.000	0.62	0.34	0.36	< 0.001
BMI ≤ 18.5	−0.04	−0.05	−0.01	0.011	−0.05	−0.05	−0.02	< 0.001
BMI = 24.0–26.9	0.03	0.00	0.01	0.081	0.01	0.01	0.00	0.608
BMI ≥ 27	−0.14	−0.04	−0.02	< 0.001	−0.09	−0.02	0.01	< 0.001

Note: Simple and multiple linear regression analyses were performed. Coefficients (B) indicate the effect size.

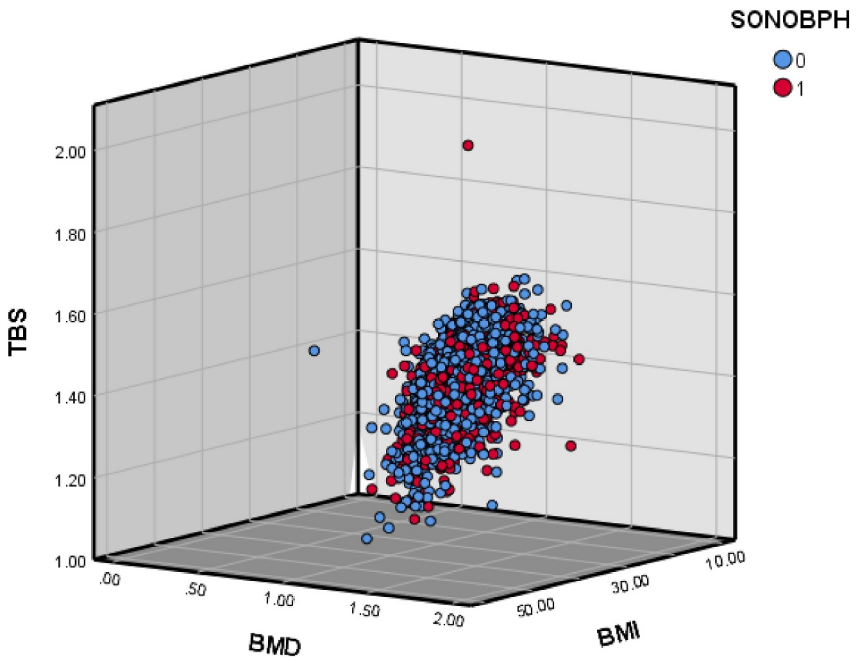

FIGURE 3 | 3D scatter plot visualizing the relationship between TBS, BMD, and BMI, with normal prostate (blue dots, coded as 0) and BPH (red dots, coded as 1).

complex interactions between BMI, BPH, and bone health metrics. By considering these population-specific characteristics, future research can provide more accurate insights and develop interventions better suited to the needs of diverse demographic groups.

In addition, BPH's association with systemic health conditions introduced additional complexity. Conditions such as obesity and insulin resistance, commonly associated with BPH [15], are known to independently affect bone health, influencing bone quality and density [32, 33]. The presence of these metabolic

conditions in individuals with BPH could have compounded the effects on bone health, as indicated by the lower TBS observed in this group. The more pronounced decline in TBS among individuals with both obesity and BPH might have been attributed to the synergistic effects of excessive adiposity and chronic inflammation, which are known to adversely impact bone remodeling. Obesity-related inflammatory cytokines, combined with the metabolic derangements commonly observed in BPH, could have exacerbated bone quality deterioration. This suggested that the impact on bone quality in BPH patients was not solely due to the direct effects of increased BMI but also the cumulative influence of associated metabolic disorders. These intertwined relationships underscore the need for a holistic approach in managing BPH, considering the broader spectrum of metabolic health and its implications for bone quality [34, 35]. The observed decrease in TBS among BPH patients could therefore be representative of the compounded effects of metabolic syndrome components on bone health, necessitating comprehensive management strategies that addressed both BPH and its metabolic comorbidities.

Lastly, the role of inflammation in BPH and its repercussions on bone health could not be overstated [15, 36]. BPH was characterized by an inflammatory component that not only contributed to prostate enlargement but also had systemic implications that might detrimentally impact bone health. Chronic inflammation, a hallmark of BPH, is known to negatively affect bone quality, potentially leading to decreased bone strength and increased fracture risk [37, 38]. The lower TBS seen in individuals with BPH might have reflected this inflammatory impact, highlighting the critical importance of managing inflammation not only for prostate health but also for maintaining bone integrity. This connection pointed to inflammation as a significant bridge between BPH and compromised bone health [37], reinforcing the necessity for anti-inflammatory interventions as part of a comprehensive treatment strategy for BPH [39, 40], aimed at mitigating its systemic effects on bone microarchitecture and overall skeletal health.

The main limitation of this study was its retrospective nature, which inherently limited the documentation of participants' medication use, exercise routines, dietary habits, and other lifestyle variables. In particular, unmeasured variables such as physical activity levels and the use of bone-related supplements, such as calcium or vitamin D, could have significantly influenced bone health outcomes. Differences in these factors among participants might have contributed to variability in BMD and TBS, potentially confounding the observed associations. Moreover, the absence of long-term follow-up with the participants limited the ability to observe the progression of BPH and its potential impacts on bone health metrics such as BMD and TBS over time. This lack of longitudinal data constrained our understanding of the dynamic interaction between BPH and bone health, making it challenging to draw definitive conclusions about the long-term effects of BPH on bone quality and density.

This study showed the complex relationship between BMI, BPH, and bone health. These strategies had to address not only the direct symptoms and challenges posed by BPH but also explore its long-term effects on bone health. It was crucial to consider a broader context to devise effective solutions that encompassed everything from pharmacological treatments to modifications in lifestyle habits. As a result, this study pointed out the importance of future research aimed at conducting more thorough lifestyle evaluations and extending follow-up periods. Such efforts were crucial for a comprehensive understanding of the relationship between BPH, lifestyle choices, and bone health, ultimately leading to better and more informed treatment plans for individuals facing these intertwined health concerns. Integrated strategies are essential to address lower TBS in individuals with BPH, especially those with higher BMI.

5 | Conclusion

In conclusion, managing aging-related health conditions, such as BPH and osteoporosis, requires a comprehensive multifaceted approach. The interconnectedness of BMI, BPH, bone health, and overall metabolic health underscores the need for integrated care strategies. These strategies should address the direct symptoms and complications associated with each condition while also considering the broader implications for metabolic health and lifestyle. Tailored interventions that incorporate both pharmacological and non-pharmacological approaches are essential for optimizing patient outcomes, highlighting the importance of a holistic perspective in the treatment and management of these complex health issues.

Author Contributions

All authors made substantial contributions to the conception and design of the study. Image acquisition and data collection were carried out by Tzyy-Ling Chuang and Pao-Liang Chen, while Malcolm Koo provided statistical support and interpretation. The initial draft of the manuscript was prepared by Tzyy-Ling Chuang and Yuh-Feng Wang, with Malcolm Koo and Mei-Hua Chuang supervising the writing process. Mei-Hua Chuang and Yuh-Feng Wang further revised the manuscript and finalized the submission. All authors reviewed and approved the final manuscript.

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Conflicts of Interest Statement

The authors declare no conflicts of interest.

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