Original article

Body composition and bone mineral density in patients with spondyloarthritis: A case-control study

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Abstract

Objectives: This study aimed to investigate the relationship between bone mineral density (BMD) and various body composition parameters in Vietnamese patients with SpA.

Methods: A case-control study was conducted at Hue University of Medicine and Pharmacy University Hospital, Vietnam, involving 43 patients with SpA and 71 age-, sex-, and BMI-matched healthy controls. Dual-energy X-ray absorptiometry (DXA) was used to assess body composition, measuring BMD and Z-scores at the lumbar spine, femoral neck, and total hip. Additional parameters included muscle mass index (appendicular skeletal muscle mass (ASM), appendicular skeletal muscle mass index (ASMI), body fat percentage (BF%), and fat mass index (FMI). Multivariable linear regression models were used to analyze the associations between BMD and body composition variables, with mean differences and 95% confidence intervals calculated.

Results: SpA patients had significantly reduced BMD in the lumbar spine and total hip compared to the healthy control group. Furthermore, significant positive correlations were observed between femoral neck BMD and both ASM ($r = 0.304$, $p = 0.047$) and ASMI ($r = 0.319$, $p = 0.037$), as well as between total hip BMD and ASMI ($r = 0.307$, $p = 0.045$).

Conclusions: The findings highlight the importance of monitoring BMD in patients with SpA due to the observed bone loss. Further research is warranted to fully elucidate the complex interplay between inflammation, body composition, and bone health in SpA.

Keywords: *Body composition, bone mineral density, Spondyloarthritis, Vietnam.*

1. INTRODUCTION

Spondyloarthritis (SpA) encompasses a diverse group of chronic inflammatory rheumatic diseases that primarily target the axial skeleton, peripheral joints, and entheses - the points where tendons or ligaments attach to bone. Majorsubtypes within this spectrum include ankylosing spondylitis (AS), psoriatic arthritis (PsA), reactive arthritis, and SpA linked to inflammatory bowel disease. Despite their clinical differences, these conditions share underlying genetic and immunological features, including a strong link to HLA-B27 and elevated levels of pro-inflammatory cytokines [1].

In recent years, attention has shifted beyond joint and entheseal manifestations toward systemic consequences of SpA, including notable alterations in body composition and bone metabolism [2, 3].

One of the notable complications in SpA is reduced BMD, which predisposes patients to osteopenia and osteoporosis at a younger age compared to the general population [4, 5, 6]. These skeletal abnormalities have been observed even in early disease stages, with low BMD affecting up to 47% of patients in the lumbar spine and hip regions [7], suggesting that bone loss may begin early in the disease course. In parallel, patients with SpA frequently exhibit disruptions in muscle and fat compartments, often manifesting as sarcopenia and excess adiposity [3, 8]. These alterations likely stem from the combined effects of chronic systemic inflammation, immobility due to pain and stiffness, and the catabolic actions of key cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and IL-6 [9]. Emerging research also suggests that abnormal fat distribution, particularly visceral adiposity, may further exacerbate inflammatory activity and worsen disease outcomes [8, 10].

Although previous studies have described altered muscle and fat parameters in SpA, findings on the interrelationship between bone density, lean mass, and fat mass remain inconsistent. Several studies have demonstrated a marked reduction in appendicular muscle mass among patients with long-standing AS [2, 11], whereas others have reported body composition comparable to that of healthy individuals [12, 13]. These discrepancies highlight

the complexity of body composition alterations in SpA and suggest that multiple interacting factors, such as disease subtype, duration, inflammation level, and physical activity, may play important roles. Consequently, further investigation is warranted to clarify the interrelationship between bone, muscle, and fat compartments in SpA and to better define their clinical relevance.

Despite growing international interest in these associations, research focusing on Southeast Asian populations remains limited [6, 14]. In Vietnam, only limited data are available regarding the skeletal implications of SpA. Preliminary findings indicate a high prevalence of sarcopenia among Vietnamese SpA patients [15], yet comprehensive assessments that link bone density with muscle and fat indices are lacking.

This study aimed to assess bone mineral density (BMD) and its related factors in Vietnamese patients with SpA using dual-energy X-ray absorptiometry (DXA). Enhancing understanding of these interactions may provide valuable insights for improving clinical management and musculoskeletal outcomes in this population.

2. MATERIALS AND METHODS

2.1. Study design and participants

This case–control study was conducted at the Department of General Internal Medicine - Endocrinology - Rheumatology and the Functional Exploration Unit of Hue University of Medicine and Pharmacy Hospital in Vietnam, from February 2023 to September 2024.

The study population comprised 43 patients with a confirmed diagnosis of SpA and 71 body mass index (BMI), sex, and age-matched healthy controls to minimize confounding effects on body composition variables (Fig. 1). Eligible SpA participants were aged 17 and 50 years and met the Assessment of SpA International Society (ASAS) classification criteria [1]. Control subjects were recruited from individuals attending routine health check-ups at the same hospital and had no history of spinal disorders or chronic lower back pain. Matching for age and BMI aimed to reduce confounding influences on body composition parameters. Participants were excluded if they had any conditions potentially affecting body composition, such as endocrine disorders (including diabetes mellitus, hyperthyroidism, hyperparathyroidism, Cushing's syndrome), hepatic cirrhosis, history of gastrectomy, severely underweight (BMI < 16.5 kg/m^2) or obese (BMI ≥ 30 kg/m^2) [16], chronic systemic diseases, pulmonary disorders, gout, or rheumatoid arthritis. Individuals were also excluded if they had contraindications to dual-energy X-ray absorptiometry (DXA), including recent radiological contrast or radioisotope exposure, pregnancy, presence of metallic implants in the scan area, or inability to maintain the scanning position. Those who declined or were unable to provide informed consent were excluded.

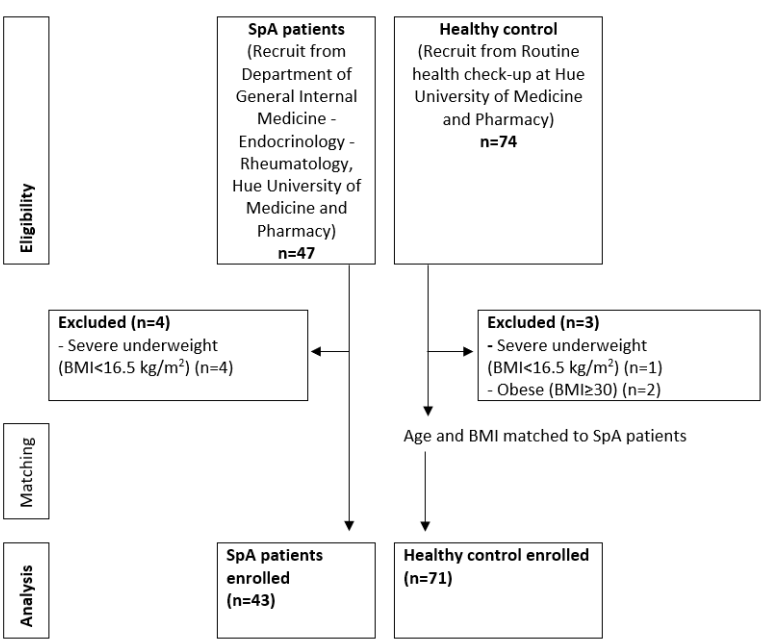

Figure 1. Study flowchart

2.2. Variables

Body composition and BMD were assessed using DXA (Medix DR, MEDILINK), with participants in a supine position and limbs extended, following standardized scanning procedures. All measurements were conducted by a single trained technician to ensure measurement consistency.

BMD values (g/cm²) and corresponding age- and sex-adjusted Z-scores were obtained at the lumbar vertebrae (L1–L4), total hip, and femoral neck of the non-dominant leg. Low BMD corresponded to a Z-score ≤ −2.0 at any site, in accordance with International Society for Clinical Densitometry guidelines [17].

Muscle mass parameters included total body muscle mass (kg) and muscle mass index (MMI, kg/m²). Appendicular skeletal muscle mass (ASM) was obtained by summing the lean mass of the arms and legs, and the appendicular skeletal muscle mass index (ASMI, kg/m²) was subsequently calculated. Fat mass was assessed as total fat mass (kg), body fat percentage (BF%; total fat mass divided by total body weight × 100), and fat mass index (FMI, kg/m²).

Demographic and clinical variables included age and sex, BMI (kg/m²), waist-to-hip ratio (WHR), and waist circumference (WC, cm). Disease duration was recorded from the time of initial SpA diagnosis. Disease activity was evaluated using the Ankylosing Spondylitis Disease Activity Score incorporating C-reactive protein (ASDAS-CRP) [18]. Medication use was documented, including non-steroidal anti-inflammatory drugs (NSAIDs), conventional synthetic disease-modifying antirheumatic drugs (csDMARDs), and biologic agents. Inflammatory markers included erythrocyte sedimentation rate (ESR, mm/h) and serum C-reactive protein (CRP, mg/L).

2.3. Statistical analysis

Continuous variables were expressed as means (95% CIs) or medians (Q1–Q3), as appropriate, while categorical variables were presented as frequencies and percentages. Group comparisons used Student's t-test, Mann–Whitney U test, or chi-square test. Spearman's correlation assessed associations between continuous variables. Multivariable linear regression was performed to identify factors associated with BMD, with results reported as mean differences and 95% CIs. Statistical analyses were conducted using SPSS version 16, with $p < 0.05$ considered significant.

2.4. Ethics Statement

The study protocol was approved by the Institutional Review Board of the Hue University of Medicine and Pharmacy (No. H2023/226 dated 24th May 2023) of Vietnam. Informed consent was confirmed by the Institutional Review Board.

3. RESULTS

3.1. Study participants

Our study initially collected a total of 47 subjects in the patient group and 74 subjects in the control group. After applying the exclusion criteria, 43 patients with SpA and 71 control subjects remained eligible. Finally, 43 SpA patients and 71 healthy controls were included in the analysis.

No significant differences were observed between the SpA patients and healthy controls with respect to sex, age, or BMI (Table 1). WC and WHR were significantly elevated in SpA patients compared with controls (WC: 78 [70 - 83] vs. 71 [65 - 74] cm, $p < 0.001$; WHR: 0.86 ± 0.05 vs. 0.82 ± 0.05, $p < 0.001$). The median CRP level among SpA patients was 11.73 mg/L (IQR: 2.29 - 41.08), and ESR was 20.00 mm/h (IQR: 10.00 - 35.00). Median disease duration was 4.00 years (IQR: 2.00 - 7.00), with 88.37% of patients classified as having active disease. Regarding treatment history, 65.12% of patients had not received DMARDs therapy.

Table 1. Comparison of characteristics between the patients and the control group

Characteristic		Patients (n = 43)	Controls (n = 71)	*p*
Sex, n (%)	Male	24 (55.81)	27 (38.00)	0.064
	Female	19 (44.19)	44 (62.00)	
Age (years)		29 (22 - 36)	30 (21 - 37)	0.704
Age group, n (%)	≤ 30	24 (55.81)	36 (50.70)	0.596
	> 30	19 (44.19)	35 (49.30)	
BMI (kg/m²)		21.26 (19.38 - 22.68)	21.45 (19.56 - 23.05)	0.559
WC (cm)		78 (70 - 83)	71 (65 - 74)	< 0.001
WHR, mean (SD)		0.86 (0.05)	0.82 (0.05)	< 0.001

CRP (mg/l)		11.73 (2.29 - 41.08)	-
ERS (mm/h)		20.00 (10.00 - 35.00)	-
Disease duration (years)		4.00 (2.00 - 7.00)	-
Disease activity, n (%)	Inactive	5 (11.63)	-
	Active	38 (88.37)	-
Used DMARDs, n (%)	No	28 (65.12)	-
	Yes	15 (34.88)	-

Values are reported as the median (interquartile range) or n (%) [unless otherwise indicated].

BMI, body mass index; CRP, C-reactive protein; DMARDs, disease-modifying antirheumatic drugs; ESR, erythrocyte sedimentation rate; SD, standard deviation; WC, waist circumference; WHR, waist-to-hip ratio.

3.2. Comparison of Bone Mineral Density Between Groups

Table 2. Comparison of bone mass in the patients and control group

Characteristic		**Patients (n = 43)**	**Controls (n = 71)**	***p***
Lumbar spine	BMD	0.909 ± 0.154	1.036 ± 0.133	< 0.001
	Z-score	-0.651 ± 1.540	0.623 ± 1.276	< 0.001
Femoral neck	BMD	0.897 ± 0.147	0.946 ± 0.137	0.081
	Z-score	0.507 ± 1.301	1.051 ± 1.200	0.029
Total hip	BMD	0.976 ± 0.143	1.054 ± 0.137	0.005
	Z-score	-0.035 ± 1.117	0.559 ± 1.012	0.006

Values are reported as mean ± SD; BMD: bone mineral density.

Table 2 presents the comparison of bone mineral density (BMD) between SpA patients and healthy controls. Patients exhibited significantly lower lumbar spine BMD (0.909 ± 0.154 vs. 1.036 ± 0.133, p < 0.001) and Z-scores (−0.651 ± 1.540 vs. 0.623 ± 1.276, p < 0.001) compared with controls. At the femoral neck, mean BMD was slightly lower in patients than in controls (0.897 ± 0.147 vs. 0.946 ± 0.137), although this difference was not significant (p = 0.081). In contrast, femoral neck Z-scores were significantly reduced in the patient group (0.507 ± 1.301 vs. 1.051 ± 1.200, p = 0.029).

3.3. Correlations Between BMD and Clinical or Body Composition Variables

Table 3. Correlation between bone mineral density and various factors in SpA patients (n=43)

BMD	**Lumbar spine**		**Femoral neck**		**Total hip**	
	r	**p**	**r**	**p**	**r**	**p**
Age	-0.066	0.673	0.097	0.537	0.048	0.758
BMI	0.054	0.731	0.219	0.158	0.277	0.072
WC	-0.002[a]	0.988	0.184[a]	0.238	0.208[a]	0.180
WHR	-0.117	0.453	0.095	0.545	0.049	0.755
Disease duration	-0.219[a]	0.157	-0.105[a]	0.501	-0.132[a]	0.400
LMI	-0.221	0.155	0.200	0.199	0.196	0.209
ASM	-0.026	0.869	0.304	0.047	0.251	0.104
ASMI	-0.085	0.588	0.319	0.037	0.307	0.045
BF%	0.199[a]	0.201	-0.021[a]	0.895	0.102[a]	0.513
FMI	0.113	0.471	0.034	0.827	0.155	0.322

Values represent Pearson correlation coefficients, except where indicated by [a], which denote Spearman correlation coefficients.

ASM, appendicular skeletal muscle mass; ASMI, appendicular skeletal muscle mass index; BMD, bone mineral density; BMI, body mass index; BF%, body fat percentage; FMI, fat mass index; MMI, muscle mass index; LMI, lean mass index; WC, waist circumference; WHR, waist-to-hip ratio.

Table 3 presents the correlations between BMD at key skeletal sites and clinical and body composition metrics among patients with SpA. At the femoral neck, BMD was significantly positively associated with ASM (r = 0.304, p = 0.047) and ASMI (r = 0.319, p = 0.037). Similarly, total hip BMD was significantly correlated with ASMI (r = 0.307, p = 0.045). Lumbar spine BMD showed no significant associations with the measured variables. Other variables, including age, BMI, WC, WHR, disease duration, LMI, BF%, and FMI, did not demonstrate significant correlations with BMD at any skeletal site.

3.4. Multivariate Analysis of BMD Determinants

Table 4. Some factors associated with bone mineral density in SpA patients (Multiple linear regression) (n = 43)

Variables	Lumbar spine BMD		Femoral neck BMD		Total hip BMD	
	Adjusted β (95% CI)	p	Adjusted β (95% CI)	p	Adjusted β (95% CI)	p
Age	-0.001 (-0.007; 0.005)	0.745	0.004 (-0.001; 0.009)	0.140	0.003 (0.238; 1.116)	0.259
BMI	0.000 (-0.035; 0.035)	0.980	0.006 (-0.025; 0.038)	0.697	0.001 (-0.029; 0.031)	0.971
ASDAS-CRP	-0.039 (-0.080; 0.001)	0.056	-0.035 (-0.071; 0.002)	0.061	-0.044 (-0.079; -0.009)	0.014
ASMI	-0.026 (-0.115; 0.062)	0.550	0.053 (-0.026; 0.133)	0.183	0.054 (-0.022; 0.129)	0.161
FMI	0.004 (-0.033; 0.041)	0.832	-0.011 (-0.045; 0.022)	0.491	0.000 (-0.032; 0.032)	0.983

ASMI, appendicular skeletal muscle mass index; BMD, bone mineral density; BMI, body mass index; CI, confidence interval; FMI, fat mass index.

In multivariate analysis, elevated ASDAS-CRP was independently linked to reduced total hip BMD (β = −0.044; 95% CI: −0.079, −0.009; p = 0.014), whereas other factors showed no significant independent effects (Table 4). No significant predictors of lumbar spine or femoral neck BMD were identified, and age, BMI, ASMI, and FMI were not independently associated with total hip BMD.

4. DISCUSSION

This study showed that SpA patients have significantly lower BMD than age- and BMI-matched controls. BMD at the femoral neck and total hip was positively associated with muscle mass, while higher disease activity was linked to lower total hip BMD. These findings are consistent with previous reports of increased osteopenia and osteoporosis in SpA patients. For instance, El Maghraoui et al. [19] found osteoporosis in 16% of AS patients in Morocco versus 3% in controls, and Singh et al. [20] reported lumbar spine osteoporosis in 22% of Indian AS patients compared to 2.7% in controls. Similar trends have been documented in France, Korea, and China, underscoring the global relevance of impaired bone health in SpA populations [6, 21].

The study revealed significant positive associations between femoral neck and hip BMD and ASMI, suggesting that muscle mass contribute to the preservation of bone health in SpA patients. This aligns with prior studies linking sarcopenia to compromised bone integrity in the context of inflammation [19]. However, the lack of a significant association between lumbar spine BMD and muscle or fat mass in the study may reflect disease-specific pathological changes in the axial skeleton. In AS, syndesmophyte formation and calcification of spinal ligaments can lead to overestimated BMD on DXA, thereby masking underlying bone loss [22]. This phenomenon may explain the discrepancy in correlations observed at different skeletal sites. Notably, several previous studies reported conflicting results. In Morocco, it was shown by El Maghraoui et al. [19] that total hip BMD was lower in AS patients with sarcopenia compared to those without. In contrast, a study conducted in Tunisia found no significant associations between lumbar spine or femoral neck BMD, T-scores, or Z-scores and measures of muscle or fat mass [12]. Similarly, a study in Thailand reported that femoral neck BMD and T-score were not significantly correlated with decreased ASMI [14]. These inconsistencies likely stem from methodological heterogeneity, such as differences in body composition assessment (DXA vs. BIA), varied cut-offs for defining sarcopenia, and population characteristics including age, sex distribution, disease subtype (AS vs. PsA), and disease duration. A recent systematic review emphasized the influence of these factors in sarcopenia prevalence studies among SpA populations [3]. Our findings highlight a site-specific association between muscle mass and BMD, particularly at the femoral neck and total hip, which are less affected by spinal calcifications and may better reflect true bone status in SpA patients.

The multivariate analysis demonstrated a significant inverse association between ASDAS-CRP and total hip BMD, reinforcing the established link between systemic inflammation and dysregulated bone remodeling in SpA [9]. Similarly, Van Der Weijden et al. [7] found correlations between inflammatory markers, including CRP, and decreased bone mass at the femoral neck and lumbar spine. However, the relationship between inflammation and BMD in SpA is complex and influenced by factors such as timing of assessment, patient characteristics, disease duration, subtype, and prior treatments. Future studies should consider these variables to better clarify the link between inflammation and bone health.

In this study, although femoral-neck BMD showed a correlation with ASM and ASMI in univariate analysis, these associations were not significant after multivariate adjustment. Several factors may explain this finding. Most importantly, systemic inflammation - as reflected by ASDASCRP - may exert a dominant influence on bone loss; consistent with prior studies identifying CRP and functional capacity (BASFI/BASMI) as primary determinants of low BMD in early SpA [7]. Finally, inflammatory pathways specific to SpA, including dysregulation of Wnt/β-catenin signaling and altered expression of bone regulatory proteins such as DKK-1 and sclerostin [23], may fundamentally modify the typical muscle-bone relationship observed in non-inflammatory conditions. Together, these factors suggest that total hip BMD, with its larger measurement area and lower technical variability, may serve as a more robust indicator of muscle-related bone changes in SpA populations.

Several limitations should be noted. The small sample size may limit statistical power and generalizability. The inclusion of both treated and treatment-naive patients, including those on TNF inhibitors, may have introduced heterogeneity. Additionally, physical activity and dietary factors were not assessed. Despite these limitations, the study's strength lies in the comprehensive DXA-based evaluation of body composition and the inclusion of a control group, enabling reliable comparisons.

5. CONCLUSION

This study demonstrated significant associations between BMD, muscle mass, and disease activity in Vietnamese SpA patients. BMD was reduced, particularly at the total hip, and was positively associated with muscle mass, while higher disease activity was independently linked to lower total hip BMD. These findings highlight the need for integrated assessment and management of inflammation, muscle, and bone health. Further longitudinal studies are required to assess the impact of interventions on body composition.

Conflict of Interest

The authors have no conflicts of interest to declare.

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