Original Article

The Relationship between Disease Activity, Quality of Life, Functional Status, Spinal Mobility, Heel Enthesitis, and Cartilage Thickness in Patients with Axial Spondyloarthritis: A Cross-Sectional Study

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Abstract

Background: The aim of this study was to evaluate lower extremity cartilage thickness in axial spondyloarthritis (SpA) patients and healthy controls using ultrasound (US) and to determine the relationship between the indices, quality of life, enthesopathy, and cartilage thickness of patients with axial SpA.

Materials and Methods: This study included 73 axial SpA patients and 30 healthy controls. The patients with axial SpA were divided into two groups as with and without heel enthesitis. Demographic data, disease duration, and medical treatments of patients were recorded. The cartilage (hip, talar, and knee), plantar fascia, and Achilles tendon thicknesses of both healthy controls and axial SpA patients were measured by US. The Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), Bath Ankylosing Spondylitis Metrology Index (BASMI), Bath Ankylosing Spondylitis Functional Index (BASFI), patient global assessment (PGA), and Ankylosing Spondylitis Quality of Life (ASQoL) scores of patients were evaluated.

Results: There was no difference between the groups in terms of demographic data and body mass index. The axial SpA groups with and without heel enthesitis were similar in terms of medical treatment and disease duration. The axial SpA patients with heel enthesitis had thinner cartilages than those without heel enthesitis ($P < 0.05$). The axial SpA patients without heel enthesitis had thinner cartilage thicknesses than the healthy control group ($P < 0.05$). There were statistically significant differences between the two groups in terms of the BASDAI, BASFI, BASMI, and ASQoL scores. These indices were negatively correlated with cartilage thickness ($P < 0.05$; r: −0.420).

Conclusion: Lower extremity cartilage thickness is associated with disease activity, quality of life, and spinal mobility in patients with axial SpA.

Key Words: *Cartilage thickness, enthesopathy, spondyloarthritis, ultrasound*

Introduction

Spondyloarthritis (SpA) is a chronic, progressive, and inflammatory disease involving the axial skeleton, peripheral joints, entheses, and the synovium.[1] If untreated, peripheral joint deformities may develop due to cartilage destructive processes along with new bone formation.[2] Although this is an important indicator of disease activation and many hypotheses have been proposed regarding it, the exact cause remains unclear. Atagunduz *et al*. showed the critical role of cartilage-directed cellular autoimmunity in joint-specific tissue damage.[3] Kim *et al*. reported that serum cartilage markers (C-propeptide of type II collagen, proteoglycan aggrecan) were changed and associated with disease activity in patients with SpA.[4] It has also been shown that serum levels of collagen degradation fragments such as cartilaps (CTX-II) and C2M are increased in axial SpA patients.[5,6] Moreover, matrix-metalloproteinase-3 (MMP-3) is correlated with disease activity and plays an important role in cartilage degeneration.[7] MMPs have been shown to have a value in the prediction of joint cartilage degeneration in rheumatoid arthritis, osteoarthritis, and SpA patients.[8-10]

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Enthesopathy is a common condition and has a crucial role in the diagnosis of SpA. Achilles tendon and plantar fascia were the most affected entheseal regions in patients with SpA.[11,12] These entheses are fibrocartilaginous and the most involved regions in arthritis-related enthesopathy. Moreover, type 2 collagen is the common histological structure of both fibrocartilage and joint cartilage. The fact that cartilage thickness was affected by the severity of enthesopathy in a study may be the result of this common histology.[13] In the later stages of enthesitis, a proliferative process of cartilage metaplasia and endochondral ossification (induced by cytokines) leads to the formation of woven bone.

Musculoskeletal ultrasound (US) has become increasingly important for the assessment of joint cartilage owing to its noninvasive nature, low cost, portability, repeatability, dynamic real-time evaluation, and easy side-to-side comparison. It is also reliable and valid for diagnosing enthesopathy in patients with SpA.[14] However, there is only one cross-sectional study that evaluated the femoral cartilage thickness in patients with axial SpA using US.[15] This study reported that the cartilage of axial SpA patients was thicker due to the use of tumor necrosis factor (TNF)-alpha inhibitors.

To the best of our knowledge, US measurements of the hip, femoral, and talar cartilage thicknesses and factors affecting cartilage thickness in patients with axial SpA have not been previously studied. We hypothesized that cartilage thickness may be altered in patients with axial SpA due to disease activity, enthesopathy, functional disability, and poor quality of life.

Materials and Methods

Patients

We conducted a cross-sectional study between February and March 2020 including patients with axial SpA who were classified by the Assessment in SpondyloArthritis International Society criteria and 30 age- and sex-matched healthy controls.[16] The study recruited patients from the rheumatology outpatient clinics of the university and healthy controls who were blood donors from the blood bank of the institution or who were university staff and their family members. Those with a history of inflammatory arthritis (rheumatoid arthritis, juvenile rheumatoid arthritis, gout), previous ankle, knee, or hip surgery, trauma, arthritis, and use of steroid injection were excluded from the study.

Enthesopathy of the foot

The structural lesions used for diagnosing enthesopathy have proven to be reliable, valid, and highly sensitive and specific symptoms in the diagnosis.[17] Ultrasonographic images of entheseal lesions such as heterogeneous hypoechogenicity of the enthesis, intratendinous focal changes, thickening of the insertional tract of the tendon, and peritendinous/perientheseal thickening as well as subentheseal lesions such as irregularity of the bone surface, erosion, and enthesophytosis were taken into consideration when diagnosing enthesopathy. However, those with only bilateral heel enthesitis (Achilles tendon and/or plantar fascia involvement) were included in the study.

Clinical evaluation

Data including age, sex, body mass index (BMI), weight, and height were obtained for each participant. Disease duration and laboratory findings of patients with axial SpA were recorded. Furthermore, anti-inflammatory drugs, synthetic disease-modifying antirheumatic drugs (DMARDs), and biological DMARDs (TNF-alpha and interleukin [IL]-17 inhibitors) that were initiated for treatment according to the American College of Rheumatology recommendations were questioned.[18] A rheumatologist also administered the questionnaires of Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), Bath Ankylosing Spondylitis Functional Index (BASFI), patient global assessment (PGA), and AS Health-Related Quality of Life (ASQoL) to the patients.[19-21] Bath Ankylosing Spondylitis Metrology Index (BASMI) scores of patients were determined by measuring their spinal mobility.[22]

Ultrasonographic evaluation

These evaluations were carried out by a rheumatologist with 10 years of experience in the musculoskeletal ultrasonographic examination who was blinded to the study group and their clinical evaluation findings. A MyLab60 Xvision (Esaote Biomedica, Genova, Italy) equipped with a linear transducer with a frequency of 6–18 MHz was used for the evaluations. The cartilage thickness has diurnal variations, particularly in weight-bearing joints. Therefore, all measurements were performed at 08:00 a. m. Accordingly, attention was paid to match the BMI, age, and gender distribution of patients between the groups. The hip, knee, and talar cartilage thicknesses along with Achilles tendon and plantar fascia thicknesses were evaluated.

Hip cartilage thickness

During the US examination, patients were placed on the examination table in the supine position with the lower limb externally rotated (heels together, toes apart). The hip joint was scanned in both longitudinal and transverse planes. From proximal to distal, the acetabulum cortex and femoral head and neck were visualized as hyperechoic images, whereas the hyaline cartilage was visualized as a thin echoic layer.[23] The major disadvantage of US in hip evaluation is its partial accessibility to the inner joint structures. This is due to the inability of the US beam to penetrate the bony cortex, resulting in frequent difficulties in the complete visualization of the hyaline cartilage.

Knee cartilage thickness

Participants were seated on a plinth in a long-sitting position with their knees fully extended for 45 min to unload the femoral articular cartilage and to minimize the effects of preceding activity on the cartilage. US images of the femoral cartilage of both limbs were acquired immediately after 45 min of resting. The knee joint was flexed (as much as possible) to visualize the trochlea of the femur and the cartilage covering it in the transverse plane. In the depth of the quadriceps tendon, the cartilage was visualized as a well-defined, smooth, hypoechoic/anechoic line covering the highly reflective subchondral bone [Figure 1]. The measurements were performed at three levels: lateral femoral condyle (LFC), intercondylar area (ICA), and medial femoral condyle (MFC).[24] Although ultrasonographic evaluation of femoral cartilage thickness is practical, limitations such as operator dependency, lack of standardized definitions, and scoring systems are points to take into consideration.

Talar cartilage thickness

Talar cartilage was measured while patients were lying in the supine position with their knees flexed at 90° and their feet placed flat on the examination table. The transducer was placed on a longitudinal view, medial to the tibialis anterior tendon [Figure 2]. The thickness of the anechoic hyaline cartilage was measured from the articular side of the talus.[25] Since the inner structures of the talar joint are easily accessible, the thickness of the cartilage can be measured more objectively than other lower extremity joints. However, it should be taken into consideration that US is an operator-dependent technique.

Achilles tendon

The Achilles tendon was scanned in the short axis (transverse to the tendon) from the musculotendinous junction to the insertion at the calcaneus. The tendon was also scanned in the long axis (parallel to the tendon) over the course of the tendon. Tendon thickness and cross-sectional area (CSA) measurements were obtained transversely to the tendon at the level of the medial malleolus in line with the previous work.[26] The thickness was measured at the level of the medial malleolus with the greatest anteroposterior diameter. CSA was measured by a trace ellipse method with the trace on the edge of the echogenic border.

Plantar fascia

Participants were placed in the prone position with the examined foot over the edge of the examination table and the ankle in a neutral position. The transducer was positioned over the plantar surface of the heel approximately 0.5 cm medial to the midline longitudinal axis of the foot to visualize the plantar fascia in a longitudinal view. The thickness of the plantar fascia was then measured at the anterior margin of the calcaneus.[27]

Statistical analysis

The minimum sample size was calculated based on 85% power and a two-sided significance level of 0.05 using statistical software. We targeted a sample size based on discerning differences in cartilage thickness among groups as the primary outcome. The sample size capable of detecting a change of the difference between groups was estimated using the mean and expected standard deviation of change in cartilage thickness data obtained from a previous study (cartilage thickness for Group 1: 1.69 ± 0.36 vs. Group 2: 1.99 ± 0.29). The critical sample size was estimated to be 20 patients per group.

The Statistical Package for the Social Sciences Statistics V22.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for all statistical analyses. Demographic characteristics were presented as descriptive statistics. The Kolmogorov–Smirnov test was used to check the normality assumption of the data. Nonparametric tests were used for statistical analyses of nonnormally distributed data. The significance of differences for continuous variables was analyzed by the Kruskal–Wallis variance analysis, while the Chi-square

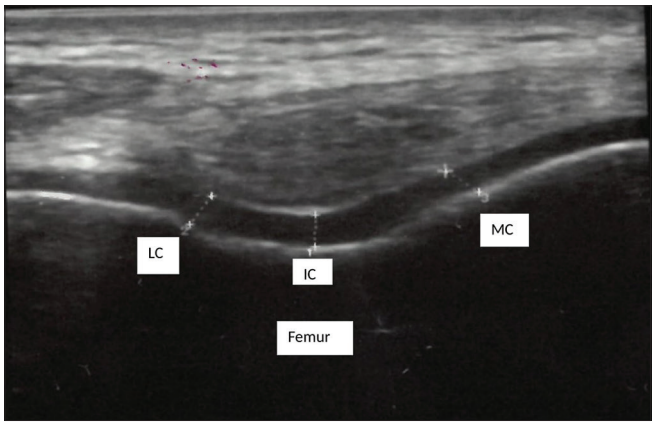

Figure 1: Ultrasonographic image of the femoral cartilage in the axial plane showing automatically generated cartilage thickness in a healthy control

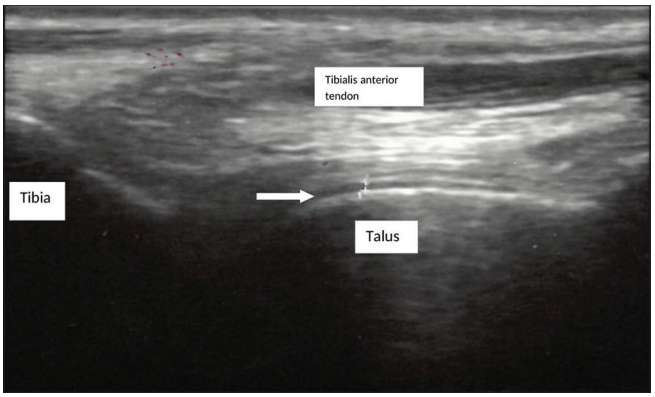

Figure 2: Ultrasonographic image of the ankle cartilage in the longitudinal plane showing automatically generated cartilage thickness in a healthy control

test was used to analyze categorical variables at baseline. The correlation between nonparametric variables was evaluated by Spearman's correlation analysis. The *post hoc* Bonferroni correction (Mann–Whitney U-test) and the Kruskal–Wallis variance analysis were used for intergroup comparisons. $P < 0.05$ was considered statistically significant in all other analyses.

Ethics

The participants were informed about the content of the study. Their written informed consent was obtained. The study was designed in accordance with the principles of the Helsinki Declaration, and the approval for the study was obtained from the Pamukkale Unıversity local ethics committee (approval number: 60116787-020/16182). Approval Date was: 24.02.2020.

Results

A total of 87 patients with axial SpA were evaluated for eligibility. Of these patients, 14 were excluded from the study. Thus, the study included 73 patients with axial SpAand 30 healthy controls. Thirty-nine of the patients with heel enthesitis were assigned to Group 1, while 34 without heel enthesitis were assigned to Group 2 [Figure 3].

The mean age of patients with axial SpA was 43.3 ± 5.5 years, while the mean age of healthy controls was 42.9 ± 8.2 years. Moreover, 63% of SpA patients and 47% of healthy controls were male. There were no significant differences with regard to demographic characteristics of patients with axial SpA and healthy controls ($P > 0.05$).

There was a significant difference between the axial SpA groups in terms of BASDAI, BASFI, BASMI, and ASQoL scores ($P < 0.001$). Moreover, axial SpA patients with enthesitis had significantly thinner cartilage thicknesses than those without enthesitis [Figure 4a and b] ($P < 0.05$) [Table 1].

The measurements of cartilage, Achilles tendon, and plantar fascia thicknesses were correlated with the BASDAI, BASFI, BASMI, and ASQoL scores ($P < 0.05$) [Table 2].

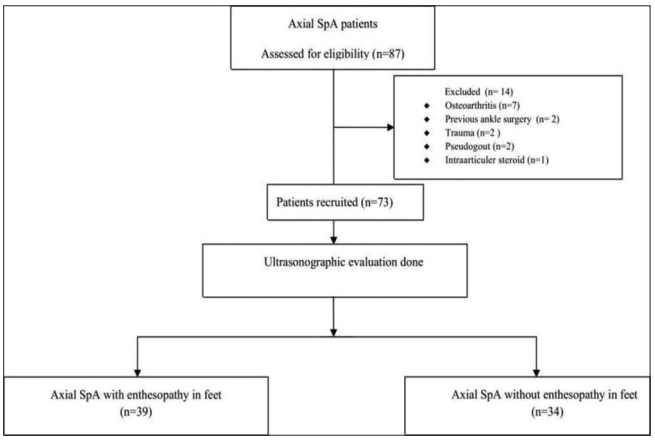

Figure 3: Flow chart of patients recruited in the study

Discussion

The present study demonstrated that patients with axial SpA had a thinner cartilage thickness and thicker plantar fascia and Achilles tendon thicknesses than healthy controls. Likewise, axial SpA patients with heel enthesitis had a thinner cartilage thickness but thicker plantar fascia and Achilles tendon thicknesses than axial SpA patients without heel enthesitis. Furthermore, there was a negative correlation between cartilage thickness and disease activity, spinal mobility, and quality of life in patients with axial SpA.

Although there are valid molecular studies on cartilage thickness in patients with axial SpA in the literature, the number of studies on mechanical factors affecting cartilage is limited. It has been reported that immobilization causes severe degeneration in the joint cartilage, resulting in contraction.[28,29] Tunç *et al*. also measured the cartilage thickness of patients with hemiplegia using US.[30] They reported that the cartilage thickness on the nonparetic side was thicker due to mobilization. Another study evaluated the femoral cartilage thickness by US in patients with spinal cord injury and found a thinner cartilage thickness compared to healthy controls.[31] In our study, more pain felt in the heel, poor quality of life, higher disease activity, and functional disability of axial SpA patients with heel enthesitis may have resulted in immobilization, suggesting the presence of a relationship between pain, immobilization, and cartilage thickness. The differences in the cartilage thicknesses of axial SpA patients with and without enthesitis may be the result of immobilization secondary to pain and disease activity. Nonetheless, since it seems difficult to draw such a conclusion due to the cross-sectional nature of our study, there is a need for prospective studies to verify this.

There are studies evaluating cartilage thickness in rheumatic diseases using US. However, all these studies have evaluated femoral cartilage thickness. Batmaz *et al*. stated that the cartilage of patients with axial SpA was thicker compared to healthy controls since the use of anti-TNF reduced type 2 cartilage degradation and increased the aggregate turnover.[15] On the other hand, there is a study reporting decreased femoral cartilage thickness in patients with axial SpA compared to healthy

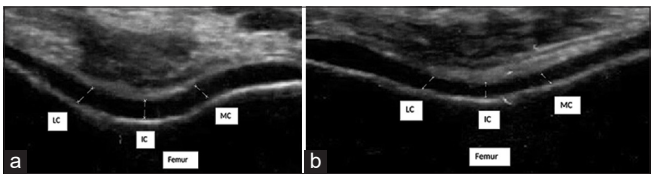

Figure 4: (a) Ultrasonographic image of the femoral cartilage in the axial plane showing automatically generated cartilage thickness in a patient with axial spondyloarthritis without heel enthesitis. (b) Ultrasonographic image of the femoral cartilage in the axial plane showing automatically generated cartilage thickness in a patient with axial spondyloarthritis with heel enthesitis

Table 1: Comparison of demographic characteristics and clinical and ultrasonographic measurement of thickness of cartilage and enthesesis

	Group 1 (*n*=39) Axial SpA with heel enthesitis	Group 2 (*n*=34) Axial SpA without heel enthesitis	Group 3 (*n*=30) Healthy controls	*P*	Mann–Whitney U-test with Bonferroni correction
Gender					
Male, *n* (%)	25 (64)	21 (44)	14 (47)	0.311	
Age (years), median (IQR)	45 (6)	42.5 (6)	44 (5)	0.182	
BMI (kg/m^2), median (IQR)	26.7 (3.8)	24.5 (5.1)	25.6 (5.6)	0.052	
HLA positivity, *n* (%)	34 (87)	31 (88)	-	0.250	
Disease duration (years), median (IQR)	10 (12)	11 (10)	-	0.378	
Laboratory findings, median (IQR)					
CRP (mg/dl), 0-1	4 (4)	4.5 (5)	-	0.570	
Sedimentation (mm/h)	19 (15)	22.5 (16)	-	0.293	
Bath indices, median (IQR)					
PGA	6 (2)	5 (2)	-	0.345	
BASDAI	5.5 (1.4)	3.6 (4.1)	-	<0.001*	
BASFI	5.7 (2)	1.3 (2.4)	-	<0.001*	
BASMI	4.8 (1.4)	2 (1.8)	-	<0.001*	
ASQoL	13 (5)	6 (7.5)	-	<0.001*	
Medical treatment, *n* (%)					
NSAID	4 (10)	5 (15)	-	0.247	
NSAID + SLZ	2 (5)	2 (6)			
Biologic DMARDs	33 (85)	27 (79)			
Right (mm), median (IQR)					
Hip	0.06 (0.02)	0.07 (0.03)	0.09 (0.02)	<0.001*	Group 1<Group 2, *P*<0.001 Group 2<Group 3, *P*<0.001 Group 1<Group 3, *P*<0.001
Knee					
MFC	0.16 (0.04)	0.19 (0.05)	0.22 (0.07)	<0.001*	Group 1<Group 2, *P*<0.001 Group 2<Group 3, *P*=0.020 Group 1<Group 3, *P*<0.001
ICA	0.16 (0.04)	0.20 (0.05)	0.23 (0.07)	<0.001*	Group 1<Group 2, *P*<0.001 Group 2<Group 3, *P*=0.037 Group 1<Group 3, *P*<0.001
LFC	0.17 (0.04)	0.19 (0.04)	0.22 (0.08)	<0.001*	Group 1<Group 2, *P*<0.001 Group 2<Group 3, *P*=0.001 Group 1<Group 3, *P*<0.001
Talar	0.06 (0.01)	0.07 (0.03)	0.09 (0.02)	<0.001*	Group 1<Group 2, *P*<0.001 Group 2<Group 3, *P*<0.001 Group 1<Group 3, *P*<0.001
Achilles tendon	0.46 (0.07)	0.38 (0.08)	0.34 (0.70)	<0.001*	Group 1>Group 2, *P*<0.001 Group 2>Group 3, *P*=0.009 Group 1>Group 3, *P*<0.001
Plantar fascia	0.46 (0.07)	0.38 (0.08)	0.23 (0.10)	<0.001*	Group 1>Group 2, *P*<0.001 Group 2>Group 3, *P*=0.001 Group 1>Group 3, *P*<0.001
Left (mm), median (IQR)					
Hip	0.05 (0.03)	0.07 (0.03)	0.09 (0.02)	<0.001*	Group 1<Group 2, *P*<0.001 Group 2<Group 3, *P*<0.001 Group 1<Group 3, *P*<0.001

Contd...

Table 1: Contd...

	Group 1 (*n*=39) Axial SpA with heel enthesitis	Group 2 (*n*=34) Axial SpA without heel enthesitis	Group 3 (*n*=30) Healthy controls	*P*	Mann–Whitney U-test with Bonferroni correction
Knee					
MFC	0.16 (0.04)	0.20 (0.04)	0.22 (0.05)	<0.001*	Group 1<Group 2, *P*<0.001
					Group 2<Group 3, *P*=0.012
					Group 1<Group 3, *P*<0.001
ICA	0.16 (0.03)	0.20 (0.05)	0.22 (0.06)	<0.001*	Group 1<Group 2, *P*<0.001
					Group 2<Group 3, *P*=0.041
					Group 1<Group 3, *P*<0.001
LFC	0.17 (0.04)	0.21 (0.06)	0.23 (0.07)	<0.001*	Group 1<Group 2, *P*<0.001
					Group 2<Group 3, *P*=0.035
					Group 1<Group 3, *P*<0.001
Talar	0.05 (0.01)	0.07 (0.03)	0.09 (0.01)	<0.001*	Group 1<Group 2, *P*<0.001
					Group 2<Group 3, *P*=0.001
					Group 1<Group 3, *P*<0.001
Achilles tendon	0.47 (0.10)	0.37 (0.08)	0.34 (0.60)	<0.001*	Group 1>Group 2, *P*<0.001
					Group 2>Group 3, *P*=0.009
					Group 1>Group 3, *P*<0.001
Plantar fascia	0.33 (0.08)	0.27 (0.08)	0.24 (0.05)	<0.001*	Group 1>Group 2, *P*<0.001
					Group 2>Group 3, *P*=0.008
					Group 1>Group 3, *P*<0.001

**P*<0.05 statistically significant. The Kruskal–Wallis test was used. IQR: Interquartile range, SpA: Spondyloarthritis, PGA: Patient global assessment, ASQoL: Ankylosing Spondylitis Quality Of Life Questionnaire, BASDAI: Bath Ankylosing Spondylitis Disease Activity Index, BASFI: Bath Ankylosing Spondylitis Functional Index, BASMI: Bath Ankylosing Spondylitis Metrology Index, NSAID: Nonsteroid anti-inflammatory drug, SLZ: Sulfasalazine, HLA: Human leukocyte antigen, CRP: C-reactive protein, MFC: Medial femoral condyle, LFC: Lateral femoral condyle, BMI: Body mass index, DMARDS: Disease-modifying antirheumatic drugs, ICA: Intercondyler area

Table 2: The relationship between disease activity, quality of life, functional status, spinal mobility, enthesopathy, and cartilage thickness

	BASDAI		BASFI		BASMI		ASQoL	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
Hip								
Cartilage thickness	−0.340	0.040*	−0.486	<0.001*	−0.519	<0.001*	−0.420	<0.001*
Knee								
MFC cartilage thickness	−0.314	0.007*	−0.486	<0.001*	−0.463	<0.001*	−0.241	0.044*
ICA cartilage thickness	−0.241	0.003*	−0.387	0.001*	−0.387	<0.001*	−0.211	0.018*
LFC cartilage thickness	−0.271	0.021*	−0.569	<0.001*	−0.501	<0.001*	−0.367	0.001*
Talar								
Cartilage thickness	−0.291	0.011*	−0.478	<0.001*	−0.480	<0.001*	−0.433	<0.001*
Plantar								
Fascia thickness	0.416	0.046*	0.285	0.015*	0.417	<0.001*	0.236	0.044*
Achilles								
Tendon thickness	0.320	0.044*	0.483	<0.001*	0.613	<0.001*	0.295	0.011*

**P*<0.05 statistically significant. LFC: Lateral femoral condyle, MFC: Medial femoral condyle, ASQoL: Ankylosing Spondylitis Quality of Life, BASDAI: Bath Ankylosing Spondylitis Disease Activity Index, BASFI: Bath Ankylosing Spondylitis Functional Index, BASMI: Bath Ankylosing Spondylitis Metrology Index, ICA: Intercondyler area

controls.[32] Another study reported that the cartilage thickness of patients with psoriatic arthritis was not different from that of healthy controls.[13] Moreover, the femoral cartilage thickness of patients with systemic lupus erythematosus has been found to be thicker compared to healthy controls. This has been explained by the chondrogenesis effect of steroids.[33] The cartilage thickness of patients with Behcet's disease has been found to be thinner compared to healthy controls. It has been stated that this may be due to subclinical inflammation.[34]

Subclinical inflammation has also been found to be effective in disease activity in patients with axial SpA.[35] Although there was no peripheral joint involvement in the patients included in our study, thinner cartilage of patients with axial SpA compared to healthy controls may be related to subclinical inflammation.

There are studies in the literature reporting that cartilage thickness is affected by disease activity. A study showed different cartilage thicknesses even among the subtypes of juvenile idiopathic arthritis.[36] This was explained by the difference in the severity of disease activity. Another study demonstrated the correlation of molecules such as C-propeptide of type II collagen, a biosynthesis marker, and the Col2-3/4 long mono levels, excessive and progressive cleavage of type II collagen with disease activity in patients with axial SpA.[4] MMPs have been shown to correlate with disease activity, cause cartilage degeneration, and play a role in radiographic progression.[5-7] IL-12/23/17 cascade is also effective in cartilage destruction along with enthesopathy in SpA patients.In particular, IL-17 and IL-23 cytokines released by this cascade have been shown to correlate with disease activity.[37-41] Therefore, the reason for the difference in joint cartilage thickness between the axial SpA groups in our study can be explained by serum levels of inflammatory markers, cytokines, and disease activity. However, there is also a study showing no correlation between cartilage molecules such as MMP-3, cross-linked C-terminal telopeptide of type I collagen, urinary deoxypyridinoline, and disease activity.[42] Therefore, this needs to be further investigated to verify these results.

Our study has three potential limitations. First of all, evaluating only the lower extremity major joint cartilage thickness and not evaluating the other entheseal regions involved in SpA is an important limitation. Moreover, not evaluating disease activity with Ankylosing Spondylitis Disease Activity Score is another limitation of our study. Because BASDAI is a patient-reported outcome measure, its score might be affected by conditions such as fibromyalgia, and depression. However, our most important and potential limitation is the lack of intraobserver and interobserver evaluation of tendon cartilage thickness measurement since US is an operator-dependent modality. Therefore, the use of three-dimensional magnetic resonance imaging, which evaluates the cartilage volume more objectively than US, could increase the quality of the study.

Conclusion

High disease activity, poor quality of life, spinal mobility, and enthesopathy are associated with a thinner cartilage thickness in patients with axial SpA. Therefore, it is emphasized that the presence of these associated factors may be a risk factor for the development of cartilage degeneration in patients with axial SpA. Accordingly, this association may be useful to identify targeted prevention and potential treatment strategies for early osteoarthritis. Further prospective studies with larger populations are needed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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