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ORIGINAL ARTICLE

Magnetic Resonance Appearance and Clinical Significance of Anterior Suprapatellar Fat Pad in Anterior Knee Pain

Anterior Diz Ağrısında Anterior Suprapatellar Yağ Yastığının Manyetik Rezonans Görünümü ve Klinik Önemi

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Abstract

Introduction: Anterior knee pain (AKP) is a very common problem in the community and negatively affects the patient's quality of life. Many pathologies can be the cause of this problem. The principal aim of our study was to evaluate the relationship between the magnetic resonance appearance of the suprapatellar fat pad and AKP.

Methods: Knee magnetic resonance imaging of patients with and without AKP was analyzed retrospectively by two independent radiologists in two groups. The morphological structure of the anterior suprapatellar fat pad and other pathologies that may cause AKP in the knee joint were evaluated. Statistical relationships of the findings were investigated.

Results: Of the 395 patients who participated in the study, 195 (49.4%) were females and 200 (50.6%) were males. The median age of the patients was 43 (18–90) years. There was no significant difference in mean age between individuals with and without AKP (p=0.376). A significant difference was found between patients with and without AKP in terms of posterior contour bowing of the anterior suprapatellar fat pad, edema, and diameter increase in the fat pad, patellar tendinopathy, quadriceps tendinopathy, and medial patellar retinaculum damage (p<0.001, p<0.001, p<0.001, p=0.015, p=0.002, and p=0.032, respectively).

Discussion and Conclusion: Anterior suprapatellar fat pad syndrome is one of the pathologies involved in the etiology of AKP. The close relationship between the posterior contour bowing of the anterior suprapatellar fat pad we defined and AKP should be considered in radiological reporting.

Keywords: Anterior knee pain; Suprapatellar; Quadriceps; Fat pad

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Anterior knee pain (AKP) is a condition that negatively affects the patient's quality of life. The most common causes of AKP are anterior tendon pathologies, chondromalacia patella, plica syndromes, and fat pad impingement syndrome.[1–5]

There are three main fat pads in the anterior of the knee: infrapatellar, anterior suprapatellar (quadriceps), and posterior suprapatellar (prefemoral).[6] These are extrasynovial, intracapsular structures.[7,8]

Fat pad impingement syndromes are among the causes of traumatic and nontraumatic pain. Fat pad inflammation causing AKP was first described by Hoffa such as the infrapatellar fat pad (IPFP), which is the largest volume fat pad around the knee joint. It is also referred to as the "Hoffa's fat pad."[6] There are studies on IPFP pathologies caused by trauma, friction, or impingement that can cause AKP.[4,8–10] However, studies on the anterior suprapatellar fat pad (ASPFP) and the posterior suprapatellar fat pad are quite limited.[11]

Although the structures of the knee joint can be assessed with ultrasound and computed tomography, MR is the gold standard for assessing the soft tissues of the knee because it provides excellent resolution of the soft tissues.[12–16] ASPFP is in the shape of a triangle superior to the superior border of the patella, posterior to the quadriceps tendon (QT), anterior to the suprapatellar recess (SPR), and is seen in the intensity of adipose tissue on magnetic resonance imaging (MRI).[3,17] When the knee is in extension, the ASPFP is between the posterior insertion of the QT and the retropatellar cartilage that covers the proximal pole of the patellar base. It fills the space between the joint capsule and the synovium.[7] The importance of fat pads has been clearly understood, especially in patients whose fat pads have been surgically removed for pathological reasons, after the changes in the mechanics of the knee and the effects of friction become evident.[18]

In this study, we aimed to investigate the clinical reflections of the morphological changes observed in ASPFP in knee MRIs, as well as their relationships with neighboring structures in the knee joint.

Materials and Methods

Study Plan and Patient Selection Criteria

This study was planned as a cross-sectional study. This study was approved by the local ethics committee and complied with the Helsinki Declaration (Date: July 1, 2021, Decision No.: 28).

Examinations of 489 patients who underwent knee MRI at the imaging center between January 2020 and December 2020 were evaluated retrospectively. Thirty-eight patients who were under 18 years of age, 34 patients with previous surgery, mass, or appearance of synovitis, and 22 patients with L3, L4, L5 root compression or ipsilateral coxarthrosis were excluded from the study. Thus, 395 knee MRIs were accepted into the study. The patients were examined in two groups: the first group consisted of those who had AKP at rest or whose AKP appeared when they walked less than 100 m, and the second group consisted of those not experiencing AKP when they walked 500 m.

MRI examinations were performed using 1.5 T (SIGNA Explorer, GE Healthcare, USA) MRI scanners. Routine sequences included in the standard protocol were taken in the MRI examinations.

In the evaluation of the images, the presence of convexity (bowing) in the posterior contour of the ASPFP and the presence of edema in the ASPFP were investigated (Fig. 1). There was no objective criterion for this condition, which is defined as the mass effect, bowing, or posterior convexity in the literature. The criteria we set for bowing was to consider a prolapse of more than 1 mm posterior to the line drawn between the posterior superior and posterior inferior corners of the ASPFP as pathological. For the diagnosis of edema, ASPFP intensity was compared with the intensity of subcutaneous adipose tissue, and hyperintensity observed in fat-suppressed sequences was noted as edema. In addition, the longest anterior–posterior diameter of ASPFP (ASPFP APD) was measured in sagittal sections.

Additionally, SPR effusion, quadriceps tendon pathology (QTp), patellar tendon pathology (PTp), patellofemoral chondromalacia (PCM), medial meniscus degeneration, lateral meniscus degeneration, anterior cruciate ligament injury, posterior cruciate ligament injury, medial collateral ligament injury, lateral collateral ligament injury, medial patellar retinaculum injury (MPRI) and lateral patellar retinaculum injury (LPRI) were also evaluated. Images in the collection media of the imaging center were analyzed separately by two radiologists, each with 22 years and 19 years of experience, using the Picture Archiving and Communication System (PACS). The average of the measurements was taken. The consent of both radiologists was required for the presence of pathologies.

Statistical Analysis

Parametric test assumptions about whether there is a significant difference between patient groups with and without AKP in terms of numerical variables were determined

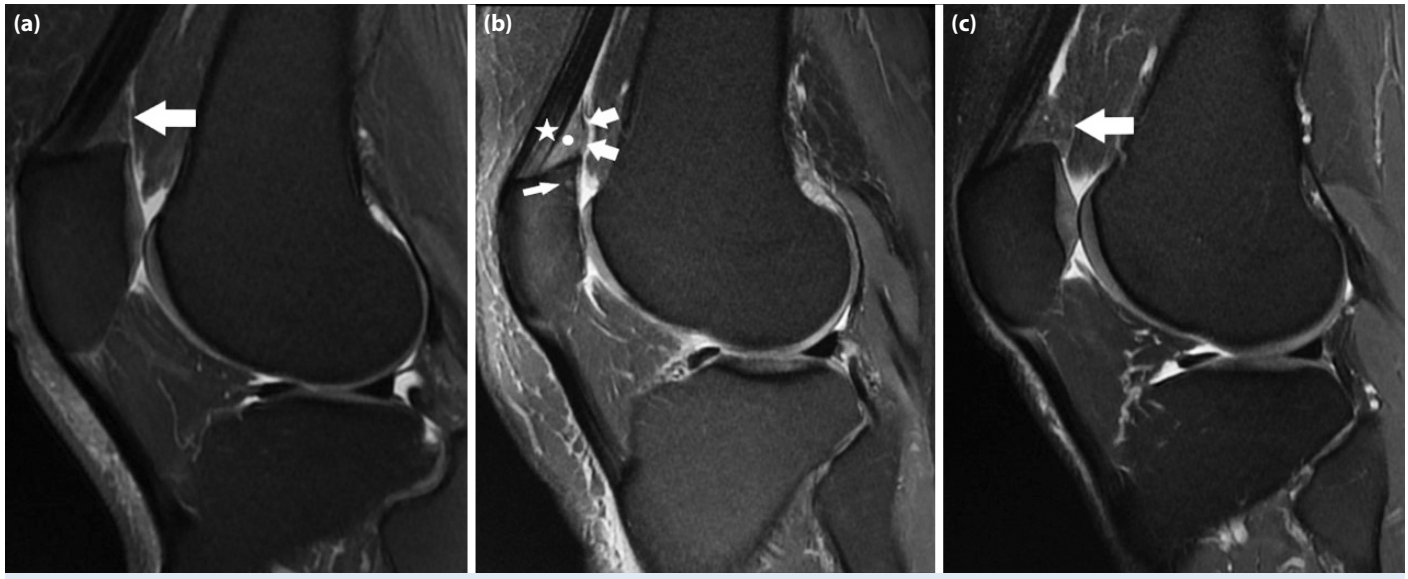

Figure 1. ASPFP (white arrow) is seen at normal thickness and intensity. The posterior contour is smooth **(a)**. Edematous ASPFP (white spot), bowing in its posterior contour (thick white arrows). The same patient also has signs of quadriceps tendinopathy (asterisc), chondromalacia (thin white arrow) **(b)**. The prominent thick ASPFP (white arrow) shows prominent bowing in its posterior contour, but there is no obvious edema **(c)**.

by Student's t test, and if not, by the Mann–Whitney U Test. Pearson's chi-squared test or Fisher's exact test was used depending on the assumptions in the evaluation of categorical data. The prevalence odds ratio (POR) analysis was used to determine the likelihood factors for AKP, and the POR was given. The analysis of the study was made using the SPSS v22 program (IBM, Armonk, NY, USA), and $p<0.05$ was considered statistically significant.

Results

Of the 395 patients included in the study, 195 (49.4%) were females and 200 (50.6%) were males. The median age of the patients was 43 (18–90) years. While 177 (44.8%) of the patients had AKP, 218 (55.2%) did not have AKP. The median age of patients without AKP was 42 (18–80) years, while those with AKP was 45 (18–90) years. Table 1 presents the rates and statistical values of the presence or absence of AKP in the parameters we looked at.

For the ASPFP diameter, which is a likelihood factor for AKP, a 1-unit increase in diameter increases the likelihood of AKP by 2.32 times ($p<0.001$). Patients with positive PTp have a 1.7 times higher likelihood of AKP than those with negative PTp (OR=1.722; 95% CI: 1.108–2.676; p=0.016). Patients with positive QTp have a 1.9 times higher likelihood of AKP than those with negative QTp (OR=1.862; 95% CI: 1.246-2.784; p=0.002). In the group of patients with positive MPRI, there is a 1.8 times higher likelihood of AKP than those with negative MPRI (OR=1.820; 95% CI: 1.048–3.161; p=0.033). The likelihood of developing AKP is 90.8 times

Table 1. Comparison of AKP in terms of parameters

	AKP		p
	Negative	**Positive**	
Age	42 (18–80)	45 (18–90)	0.376[a]
Gender			0.058[b]
Female	117 (60.0)	78 (40.0)	
Male	101 (55.5)	99 (49.5)	
Bowing	10 (6.5)	144 (93.5)	<**0.001**[b]
Edema	52 (26.7)	143 (73.3)	<**0.001**[b]
SPE	102 (59.3)	70 (40.7)	0.149[b]
PTp	51 (45.5)	61 (54.5)	**0.015**[b]
QTp	97 (47.8)	106 (52.2)	**0.002**[b]
MMp	98 (55.7)	78 (44.3)	0.860[b]
LMp	41 (54.7)	34 (45.3)	0.919[b]
ACLI	62 (55.9)	49 (44.1)	0.868[b]
PCLI	4 (66.7)	2 (33.3)	0.695[c]
MCLI	12 (63.2)	7 (36.8)	0.474[b]
LCLI	32 (57.1)	24 (42.9)	0.751[b]
MPRI	26 (42.6)	35 (57.4)	**0.032**[b]
LPRI	28 (53.8)	24 (46.2)	0.834[b]
PCM	54 (51.9)	50 (48.1)	0.435[b]
ASPFP APD (mm)	6.05 (3.30–9.70)	8.4 (3.90–16.20)	<**0.001**[a]

a: Mann–Whitney U test, median (minimum–maximum); b: Pearson's chi-squared test, n (%); c: Fisher's exact test, n (%); SPE: Suprapatellar recess effusion; QTp: Quadriceps tendon pathology; PTp: Patellar tendon pathology; PCM: Patellofemoral chondromalacia; MMp: Medial meniscus degeneration; LMp: Lateral meniscus degeneration; ACLI: Anterior cruciate ligament injury; PCLI: Posterior cruciate ligament injury; MCLI: Medial collateral ligament injury; LCLI: Lateral collateral ligament injury; MPRI: Medial patellar retinaculum injury; LPRI: Lateral patellar retinaculum injury.

Table 2. Prevalence odds ratio analysis results in the evaluation of bowing and edema together

	Odds ratio	95% CI	
		Lower	Upper
Bowing P/edema N	10.863	3.826	30.847
Bowing P/edema P	217.233	58.388	822.299
Edema P/bowing N	1.411	0.629	3.167

P: Positive; N: Negative; CI: Confidence interval.

higher in patients with positive bowing than in patients without (OR=90.764; 95% CI: 43.360–189.990; p<0.001). The likelihood of developing AKP is 13.4 times higher in patients with edema than in patients without edema (OR=13.426; 95% CI: 8.253–21.844; p<0.001).

The frequency of bowing was 93.5% in patients with AKP and 6.5% in patients without AKP (p<0.001), and the frequency of edema was 73.3% in AKP patients and 26.7% in the other group (p<0.001).

The diameter was found to be significantly higher in patients with bowing (p<0.001). While the mean ASPFP APD was 8.91±1.97 mm in patients with bowing, it was 6.17±1.36 mm in patients without bowing.

When bowing and edema parameters, which are likelihood factors for the appearance of AKP, were evaluated together, there was a significant interaction (bowing vs edema) between bowing and edema (p=0.001). The likelihood of occurrence of AKP (odds ratio) according to the coexistence of bowing and edema is given in Table 2.

Discussion

Our study reveals a strong association of morphological changes in ASPFP with AKP. Regardless of whether the etiology is traumatic or nontraumatic, ASPFP is hyperintense on T2W sequences in acute inflammation. At the same time, convexity (bowing) occurs in its posterior contour.[4,19] Jacobson et al.[4] state that inflammation in IPFP shows a mass effect by creating bowing in the patellar tendon. The term bowing was first used by Shabshin et al.[18] for ASPFP although researchers stated that inflammation in ASPFP causes pressure on both the SPR and QT. In other studies, the term bowing was not preferred; the term mass effect was used. However, in some studies examining ASPFP, there is no information about where the mass effect is.[20–22] Some state that it is a mass effect on SPR.[3,11,19]

Can et al.[20] found the ASPFP thickness in patients with mass effect positive to be higher than in other studies (10.2 mm). However, they did not detect a relationship between mass effect and size. In this study, the relationship between edema and convexity was prominent. In our study, the diameter was found to be significantly higher in patients with bowing (p<0.001). There was also a significant difference in edema between patients with and without bowing (p<0.001).

Wang et al.[7] reported that AKP is common in patients with ASPFP mass effect and changes in signal structure. In our study, we selected to use the term bowing instead of mass effect. For this expression, which is used in the literature with a similar meaning, our study completely and definitively supports this. According to our findings, there was a significant difference in AKP in patients with both bowing and edema (p<0.001). On the other hand, Shabshin et al. stated ASPFP to have both signal alteration and bowing appearance to call it pathological; the mass effect rate was 4.2%. Although most of our findings were in line with previous studies, the frequency of bowing was particularly high.[3,18]

In our study, a higher likelihood for AKP was found between AKP and increased MPR, QT, PT, and diameter although lower than bowing. In QTp and SPR in particular, effusion is one of the anatomically expected results in ASPFP inflammation.[18] It was stated in their concomitant findings that there was no relationship between normal and mass effect ASPFPs in terms of QTp and PTp.[18]

Staeubli et al.[17] found the ASPFP thickness to be different in men and women (6±2 mm in women and 7±2 mm in men). In our group, the thickness of ASPFP was 5.68±1.21 mm in women and 6.58±1.39 mm in men in the group with both bowing and edema negative and which we considered normal. The result is in parallel with the aforementioned study, and the difference between genders is significant (p<0.001).

Defining the convexity in the posterior contour of the ASPFP as the mass effect, Roth et al.[3] revealed that this image is accompanied by an anteroposterior thickness increase and signal increase. We obtained similar results in our study, and in addition, the association between bowing and edema was also evident. The mean ASPFP thickness (6.05 mm) found in patients without AKP is consistent with the findings of the previous study.[3,17] Wang et al.[7] found both mass effect and signal intensity alteration separately and significantly, as we did. Again, similar to ours, their relationship with the AKP was also significant.

In our study, physical examination findings were not taken into account; however, morphological changes (thickness, intensity, and contour convexity) in ASPFP were found to be significant in patients whose reason for applying to the

health center was AKP compared with patients without AKP complaint (p<0.001, p<0.001, and p<0.001, respectively).

Roth et al.[3] found that AKP and MCL abnormalities were also statistically associated in their study, but they reported that bowing, increased thickness, and edema in ASPFP were not associated with lesions in MCL. In our study, MCL lesions did not affect AKP. There was no difference between MCL lesions in terms of bowing, increased thickness, and edema in ASPFP (p=0.474, p=0.844, p=0.140, and p=0.858, respectively).

Tsavalas and Karantanas[19] stated that despite the 13.8% mass effect rate they found, there was no statistical relationship between knee pain and ASPFP mass effect. In our study, the frequency of bowing was 81.4 in patients with AKP and 4.6 in patients without AKP (p<0.001). It can be thought that the difference between the literature findings, those with AKP in particular, is due to the objective criteria we have determined for the definition of bowing.[3,7,19] Therefore, we accept that bowing is important in the etiology of patients with knee pain.

In previous studies, signal alteration (i.e., edema) was found to be 42%–54%.[3,7] In our study, it was 80.8% in AKP patients and 23.9% in the other group (p<0.001).

While Shabshin et al.[18] used both bowing and edema together for the diagnosis of impingement, Roth et al.[3] and Wang et al.[7] stated contour convexity as the only valid condition for mass effect. We accept that all these approaches are correct.

Wang et al.[7] considered the presence of either mass effect or signal alteration sufficient to be considered ASPFP pathology. Although our findings are similar, we think that we have revealed clearer values as this was the focus of our study. Wang et al.[7] emphasized the inconsistency of their findings with previous studies and argued that this may be due to the advanced average age in their patient groups.[7] Our findings were generally in line with previous studies, and the mean age of our patients was 43 years. We suspect the reason for this might be the availability and prevalence of MRI scans in Turkey, the low average age of the population, and those able to access the MRI devices. The prevalence of some diseases, especially chondromalacia and degenerative osteoarthritis, is high in advanced ages, and this may point to the source of some of the differences in the results.

In previous studies, there was no association between ASPFP mass effect and PF cartilage lesion and clinical worsening.[22] In a study investigating the relationship between knee FPs and normal, moderate, and end-stage osteoarthritis, no relationship was found with ASPFP, while there was a relationship with IPFP.[23] In another study, it was reported that specific cytokines in IPFP may contribute to paracrine inflammation and progressive cartilage damage in obese patients.[24] This may also apply to ASPFP although we only looked at the patellofemoral joint. We did find a relationship between chondromalacia and AKP and mass effect; in our study, all patients with bowing and PCM had AKP.

The close relationship between pain and fat pads is not surprising, as we know that fat pads are highly innervated.[25] Since the substance P-immunoreactive nerve fibers in ASPFP is a potential source of nociceptive output responsible for AKP, it is natural to have these results.[26]

In the literature, it is stated that ASPFP syndrome is a clinical diagnosis and that edema on MRI does not have to be associated with AKP.[20] In our study, we found that edema solely, without an increase in diameter or bowing, did not explain AKP. In the same study by Can et al.,[20] patellofemoral morphology measurements were made to explain the etiology of ASPFP syndrome, and no significant relationship was found, except for the tibial tubercle–trochlear groove (TT–TG) distance and patellar tilt.

The exact cause of ASPFP syndrome is not known. Among the proposed theories is hyperflexion of the knee joint; it is thought that it may develop secondary to other knee pathologies such as synovitis and chondromalacia. It is also speculated that it may occur primarily due to intrinsic reasons. According to another theory, it has been suggested that the habit of excessive knee flexion may predispose to ASPFP impingement syndrome.[3] Although there is no title related to these subjects in our study, we think that this subject can be better understood with other studies focusing on personal characteristics.

ASPFP is located anterior to the SPR and posterior to the QT. Therefore, it is expected that ASPFP inflammation affects these two structures or is affected by the pathologies of these structures.[3]

Our study was not aimed at finding the etiology of ASPFP pathologies; however, the frequency of tendinosis accompanying QT is statistically significant. Therefore, edema and thickening of ASPFP occurring for any reason may affect QT. It can be thought that the thickening in the QT may increase the inflammation in the ASPFP with the effect of friction and cause the problem to become chronic.

In our study, not getting clinical information other than pain, not following the treatment process, and not performing histopathological evaluation can all be seen as a deficiency.

Conclusion

Today, overdetection and overdefinition is an important problem in musculoskeletal imaging. It is often discussed how the pathologies written in MRI reports are related to the patient's complaints.[27] In overcoming these problems, it is important to report the radiological findings most closely related to the patient's clinic. We think that the close relationship between the impingement criteria we defined and AKP should be taken into account in radiological reporting.

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