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Research Article

Association of Infrapatellar Fat Pad Morphology and Signal Changes with Patellar Chondromalacia

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ABSTRACT

Objective: To evaluate the association between infrapatellar fat pad (IPFP) maximal area, depth, and signal intensity alterations and the presence and severity of patellar chondromalacia on magnetic resonance imaging (MRI).

Methods: In this single-center retrospective study, 172 knee MRI examinations with patellar chondromalacia and 78 control examinations were analyzed between January 1, 2024, and January 1, 2025. MRI scans were acquired without contrast using a 1.5-Tesla system (Ingenia Evolution, Philips Medical System, Best, the Netherlands). Alterations in IPFP maximal area, depth, and signal intensity (graded 0-3) were measured on fat-suppressed proton density-weighted images. Chondromalacia was staged using the MRI-adapted Outerbridge classification. Demographic characteristics, medial subcutaneous fat thickness, and IPFP measurements were compared between groups. Correlation analyses were performed to assess associations between IPFP metrics and chondromalacia severity. Parametric and nonparametric tests were applied; $P < 0$ .05 was considered statistically significant.

Results: Patients with chondromalacia were older than controls (50.8 ± 10.1 vs. 38.7 ± 12.7 years; $P < 0.001$) and included a higher proportion of females ($P = 0.009$). Medial subcutaneous fat thickness was greater in the chondromalacia group ($P < 0.001$). Male sex was correlated with larger maximal IPFP area and depth ($P < 0.001$). No significant correlation was observed between medial subcutaneous fat thickness and IPFP size. The IPFP signal intensity score was positively correlated with age (r = 0.50) and with chondromalacia grade (r = 0.377; $P < 0.001$ for both). IPFP maximal area and depth showed no significant association with chondromalacia grade, lesion location, or signal intensity score.

Conclusion: MRI signal intensity alterations of the IPFP are strongly associated with patellar chondromalacia severity. In contrast, dimensional measurements alone (area or depth) do not reflect disease severity, highlighting the superior diagnostic value of qualitative signal assessment.

Keywords: Patellar chondromalacia, infrapatellar fat pad, knee magnetic resonance imaging, signal intensity alteration, cartilage degeneration

INTRODUCTION

Hoffa's fat pad, also known as the infrapatellar fat pad (IPFP), is an intracapsular, yet extrasynovial structure located beneath the patella within the knee joint. Anteriorly, it is bordered by the patellar tendon; posteriorly, by the synovium-lined knee joint. Superiorly, it attaches to the inferior pole of the patella; inferiorly, it extends to the periosteum of the tibia.[1]

Structurally, the IPFP shares characteristics with subcutaneous adipose tissue, yet it also serves important biomechanical functions such as absorbing mechanical loads transmitted through the knee and attenuating shock. In addition, it has been shown to play a role in joint metabolic and inflammatory processes. The IPFP is known to be involved in the production of adipokines such as interleukin-6, tumor necrosis factor-α, and leptin, thereby influencing the biological environment of the knee joint.

Magnetic resonance imagin (MRI) studies conducted in patients with osteoarthritis (OA) have demonstrated that the IPFP exhibits increased signal intensity, suggesting that it may serve as a biomarker in knee OA.[2-4] Therefore, standardized,

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reproducible measurements of IPFP dimensions are important for evaluating metabolic and degenerative joint disorders.[1]

MRI studies in patients with OA have shown increased signal intensity of the IPFP, suggesting its potential as a biomarker for knee OA.[4] Alterations within the IPFP—such as inflammation or fibrosis—can be visualized on sagittal MRI sections by characteristic variations in signal intensity.[5]

Patellar chondromalacia is characterized by anterior knee pain and softening of the articular cartilage on the patellar surface, often accompanied by fibrillation or ulceration.[6] In addition to patient history and physical examination, radiological imaging methods are widely used in the diagnostic process. MRI is considered one of the most valuable imaging modalities for evaluating patellar chondromalacia and other cartilage pathologies.[7] Several studies have suggested that IPFP dimensions may be associated with patellar chondromalacia, implying that changes in IPFP size could alter the mechanical load distribution across the patellofemoral joint (PFJ), thereby contributing to cartilage degeneration.[5]

In this study, we aimed to investigate the association of maximal IPFP area, depth, and signal intensity alterations with the presence of patellar chondromalacia, considering the close anatomical relationship between the IPFP, patella, and PFJ.

MATERIAL AND METHODS

Study Approval

This retrospective study was approved by the Clinical Research Ethics Committee of the Ministry of Health University of Health Sciences Türkiye, Ankara Etlik City Hospital (approval no: 2025/0231, date: 26.02 2025). In this retrospective study, the requirement for informed consent was waived. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Study Population

This retrospective study evaluated patients who underwent non-contrast knee MRI between January 1, 2024, and January 1, 2025. The inclusion criteria were as follows:

1. Age ≥18 years

2. Availability of knee MRI examinations in our radiology database

The exclusion criteria were:

1. History of trauma to the knee

2. Prior open or arthroscopic knee surgery

3. Presence of rheumatologic disease or synovitis

4. Anatomical variants of the patella (e.g., bipartite patella)

5. Evidence of significant joint effusion and/or hemarthrosis

6. Presence of any intra-articular space-occupying lesion

7. Poor image quality preventing adequate evaluation

The study included 172 knee MRI examinations from 150 patients who met the inclusion criteria and had been diagnosed with patellar chondromalacia. The control group included 78 knee MRI examinations from 68 patients without patellar chondromalacia. In total, 250 knee MRI examinations were analyzed.

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Anthropometric Measurements

Body weight and height were measured in kilograms and centimeters, respectively. Body mass index (BMI) was calculated using the following formula:

BMI = kg/m^2, where kg represents the patient's body weight and m^2 represents the square of the patient's height.

Magnetic Resonance Imaging Assessment

All knee MRI examinations were performed on a 1.5-Tesla MR system (Ingenia Evolution, Philips Medical System, Best, the Netherlands) using a standard knee protocol. Coronal T1-weighted images were acquired with TR 622 ms, TE 8 ms, FOV 18 cm, slice thickness 3 mm, and a matrix of 360×204 pixels. Sagittal proton density SPAIR (PD SPAIR) images were obtained with TR 3466 ms, TE 30 ms, FOV 18 cm, slice thickness 3 mm, and a matrix of 292×272 pixels. Axial PD SPAIR images were acquired with TR 3333 ms, TE 30 ms, FOV 18 cm, slice thickness 3 mm, and a matrix of 360×263 pixels, while coronal PD SPAIR images were obtained with TR 622 ms, TE 8 ms, FOV 18 cm, slice thickness 3 mm, and a matrix of 360×304 pixels.

IPFP Measurements

The maximal cross-sectional area of the IPFP was calculated on sagittal T2-weighted MR images using our institutional workstation (IntelliSpace Portal v12.1, Philips, Best, the Netherlands) after manually delineating its borders. IPFP depth was defined as the distance from the anterior to the posterior surface, measured along a line perpendicular to the patellar tendon (Figure 1).

Medial Subcutaneous Fat Thickness

Medial subcutaneous fat (SCF) thickness of the knee joint region was measured as an indicator of body adiposity. The measurement was performed on axial PD-weighted images and extended from the skin surface to the posteromedial aspect of the femoral condyle cortex, as illustrated in Figure 2.

MAIN POINTS

- Infrapatellar fat pad (IPFP) signal changes on magnetic resonance imaging (MRI) strongly reflect the severity of patellar chondromalacia.
- IPFP size (area or depth) does not correlate with lesion severity or location.
- Qualitative MRI assessment of the IPFP has greater diagnostic value than dimensional measures.

Patellar Chondromalacia Assessment

Patellar chondromalacia was graded from 0 to 4 on fat-suppressed proton density-weighted axial MRI images according to the MRI-adapted Outerbridge classification.

Assessment of IPFP Signal Intensity Changes

Changes in IPFP signal intensity were evaluated on sagittal fat-suppressed proton density-weighted images (Figure 3). The sagittal plane demonstrating the most prominent signal increase was selected, and edema was defined as areas of high signal on the fat-suppressed sequence. Signal intensity alterations of the IPFP were classified as follows: grade 0 = no change; grade 1 = ≤ 10% of the area; grade 2 = 10-20% of the area; grade 3 = ≥ 20% of the area. Two observers (EZ, BA) independently performed the assessments, and a random cross-check method was used to compare the observers' assessments. Intra-observer reliability, assessed using the intraclass correlation coefficient, was 0.90, while inter-observer reliability was 0.88.

RESULTS

A total of 250 knee MRI examinations were analyzed, including 78 in the control group and 172 in the patellar chondromalacia group. The mean age of the control group was 38.74 ± 12.72 years, whereas that of the patellar chondromalacia group was 50.83 ± 10.09 years, a difference that was statistically significant (P < 0.001). In the control group, 48.5% (n=33) were female and 51.5% (n=35) were male; in the patient group, 72.7% (n=109) were female and 27.3% (n=41) were male (P = 0.009). Medial SCF thickness was significantly higher in the patient group (23.57 ± 10.36 mm) compared to the control group (17.55 ± 7.31 mm; P < 0.001). Baseline characteristics, including sex, weight, and BMI, are presented in Table 1.

Correlation analysis revealed a significant relationship between sex and both the maximal IPFP cross-sectional area (r =-0.364; P < 0.001) and the IPFP depth (r = -0.338; P < 0.001), indicating that males had significantly greater maximal IPFP area and depth than females. Subgroup analyses supported this finding, showing that males had a significantly greater maximal IPFP area (667.78 ± 134.59 mm^2 vs. 533.02 ± 109.79 mm^2, P < 0.001) and depth (29.50 ± 3.49 mm vs. 25.78 ± 3.44 mm, P < 0.001) than females (Tables 2, 3).

Medial SCF thickness was not significantly correlated with IPFP area or depth. The IPFP signal intensity score demonstrated a significant positive correlation with age (r = 0.50; P < 0.001) and chondromalacia grade (r = 0.377; P < 0.001), indicating that increases in age and chondromalacia severity were associated with higher IPFP signal intensity scores. No significant correlations were observed with other variables (P > 0.05; Table 2).

The maximal IPFP cross-sectional area was significantly greater in males (667.78 ± 134.59 mm^2) than in females (533.02 ± 109.79 mm^2; P < 0.001). Comparisons according to chondromalacia grade revealed no significant differences (Grade 1: 558.66 ± 110.73 mm^2, Grade 2: 574.72 ± 129.33 mm^2,

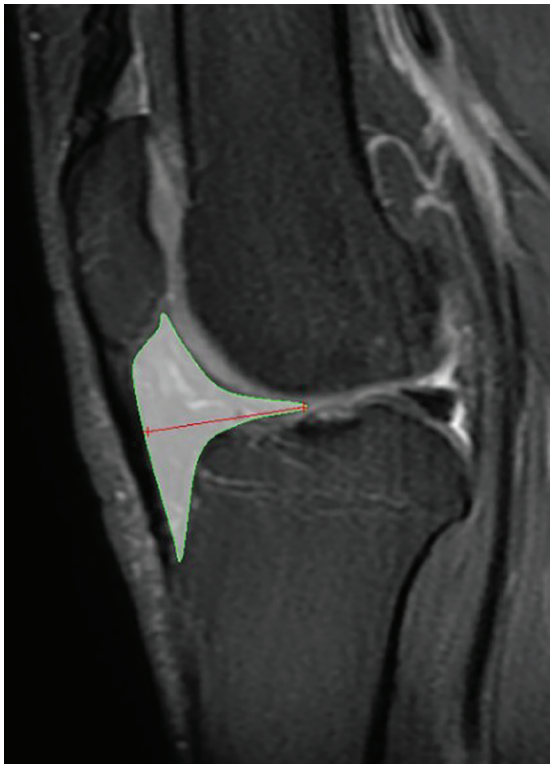


Figure 1. Sagittal T2-weighted MRI demonstrating the maximal cross-sectional area and depth of the infrapatellar fat pad (IPFP). The white shaded region represents the maximal cross-sectional area of the IPFP. The red line, drawn perpendicular to the patellar tendon from the anterior to the posterior surface, indicates the IPFP depth

MRI: Magnetic resonance imaging

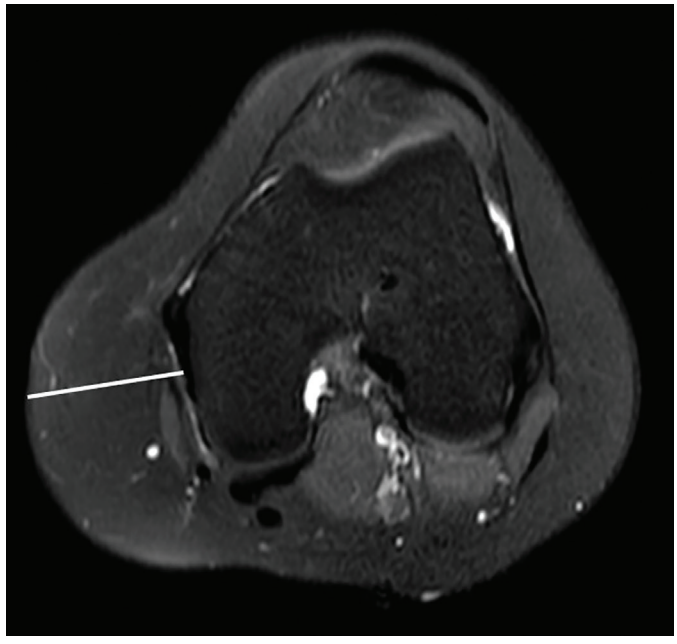


Figure 2. Measurement of subcutaneous fat thickness. Axial T2-weighted MRI of the knee demonstrating measurement of the subcutaneous fat thickness on the medial aspect of the knee joint at the level of the medial femoral condyle

MRI: Magnetic resonance imaging

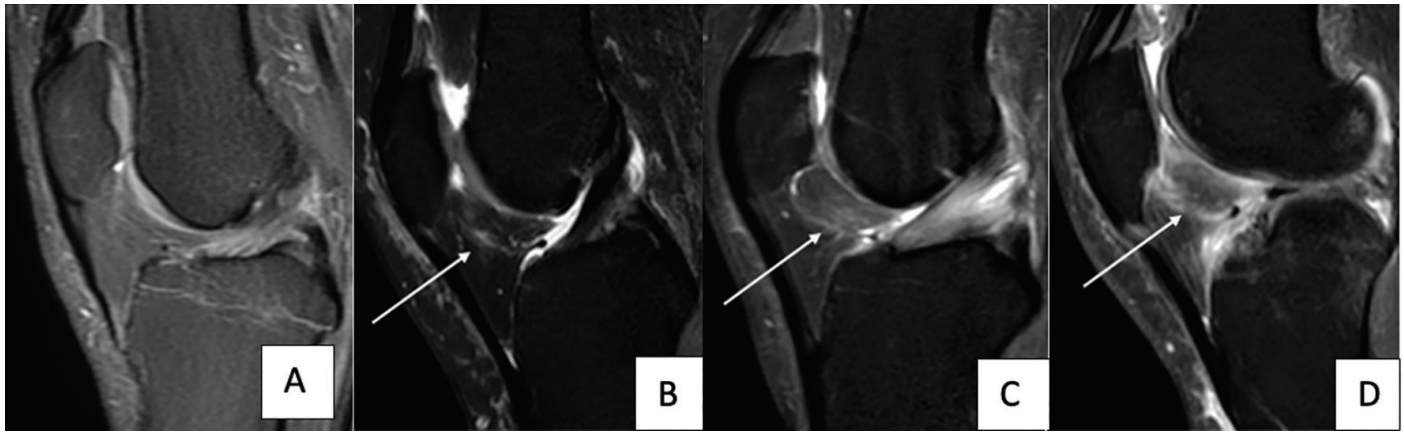


Figure 3. Classification of infrapatellar fat pad (IPFP) signal intensity alterations on MRI. The white arrows indicate regions of increased signal intensity within the IPFP. Each grade represents the percentage of IPFP involvement: Grade 0, no alteration; Grade 1, ≤ 10%; Grade 2, 10-20%; Grade 3, ≥ 20%

A) – IPFP Grade 0: No signal alteration, B) – IPFP Grade 1: Involvement of ≤10% of the IPFP area (white arrow indicates region of increased signal), C) – IPFP Grade 2: Involvement of 10-20% of the IPFP area (white arrow indicates region of increased signal), D) – IPFP Grade 3: Involvement of ≥20% of the IPFP area (white arrow indicates region of increased signal)

MRI: Magnetic resonance imaging

Grade 3: 580.00±132.66 mm^2, Grade 4: 559.91 ± 122.11 mm^2; *P* = 0.595). Similarly, no significant differences were observed in maximal IPFP area based on chondromalacia location (medial: 575.48 ± 110.15 mm^2, lateral: 553.34±152.01 mm^2, central: 602.33 ± 161.06 mm^2; *P* = 0.537) (Table 3). Regarding IPFP depth, males (29.50 ± 3.49 mm) had significantly greater values than females (25.78 ± 3.44 mm; *P* < 0.001). However, IPFP depth did not differ significantly according to chondromalacia grade (Grade 1: 27.00 ± 3.16 mm, Grade 2: 26.00 ± 3.66 mm, Grade 3: 27.45 ± 3.82 mm, Grade 4: 27.10 ± 4.14 mm; *P* = 0.512),chondromalacia location (medial: 26.83 ± 3.83 mm, lateral: 26.43 ± 3.84 mm, central: 28.00 ± 3.77 mm; *P* = 0.399), or IPFP signal intensity score (Grade 0: 26.08 ± 3.72 mm, Grade 1: 26.84 ± 4.19 mm, Grade 2: 27.47 ± 3.82 mm, Grade 3: 27.40 ± 3.15 mm; *P*=0.793) (Table 3).

DISCUSSION

Knee OA is the most common degenerative joint disease among middle-aged and elderly individuals, characterized by joint swelling, pain, and limited range of motion.[8] Depending on the location of the lesion, OA can be classified as tibiofemoral joint OA or patellofemoral joint osteoarthritis (PFOA). Studies have shown that approximately 60% of patients with symptomatic knee OA exhibit PFJ abnormalities.[9]

Several risk factors have been identified for OA, with advanced age recognized as a major contributor.[10] In the present study, PFJ degeneration was found to increase with age. This phenomenon may be attributed to the natural aging process of the PFJ and, potentially, to the effects of osteoporosis.[11] Recent studies suggest that the inflammatory milieu created by senescence-associated secretory phenotype factors associated with aging contributes to cartilage degeneration and subchondral bone remodeling, ultimately leading to cartilage loss and progression of OA.[12] In our study, we observed a moderate positive correlation between changes in IPFP signal intensity and age, indicating that IPFP alterations are closely associated with the aging process.

Consistent with previous findings, our study demonstrated a higher incidence of PFOA in females than in males.[13] Many researchers attribute this difference to estrogen levels in women. While some studies suggest that estrogen plays a protective role in OA, others report that it may contribute to the pathogenesis of the disease. This indicates that sex differences cannot be explained solely by estrogen and that other factors, such as muscle strength, lifestyle habits, and bone density, may also play important roles.[2]

Additionally, Wang and He reported that obesity is one of the most influential and modifiable risk factors for OA. In our study, patients with PFOA had a higher BMI than those in the control group. The increased load associated with overweight and obesity accelerates the progression of PFOA and induces abnormal changes in PFJ mechanics.[15] The relationship between obesity and joint degeneration is not only biomechanical but also involves adipose tissue, which can activate inflammatory processes. The IPFP plays a role in the initiation and progression of OA through the release and activation of pro-inflammatory mediators. Due to its anatomical location, the IPFP can directly influence the composition of synovial fluid, which in turn may affect other joint structures. Proinflammatory cytokines activate substance P-containing nerve fibers, which mediate anterior knee pain. These cytokines, in conjunction with adipose tissue, immune cells, and the nervous system, directly contribute to the production of chemokines, other cytokines, and growth factors. Collectively, these effects influence the metabolism and function of the synovial membrane and articular cartilage.[16]

In our study, a weak positive correlation was observed between BMI and changes in IPFP signal intensity (r=0.22; *P* < 0.02).

Table 1. Comparison of Demographic Characteristics and Infrapatellar Fat Pad (IPFP) Measurements Between Control and Patient Groups

		Control	Patient		
Variables		**Mean ± SD/median (min-max)/N (%)**	**Mean ± SD/median (min-max)/N (%)**	**t/ Z/ X^2**	***P***
Age (years)		38.74 ± 12.72	50.83 ± 10.09	-5.741*	**< 0.001**
Gender	Female	33 (48.5%)	109 (72.7)	6.805‡	**0.009**
	Male	35 (51.5%)	41 (27.3%)		
Side	Right	40 (55.5%)	87 (50.6%)	0.302‡	0.583
	Left	32 (44.5%)	85 (49.4%)		
Height (cm)		164.70 ± 6.44 165 (158-182)	165.66 ± 8.91 166 (150-183)	-0.726†	0.468
Weight (kg)		64.08 ± 9.91 67 (45-85)	82.05 ± 10.49 78 (58-103)	-8.326†	**< 0.001**
BMI (kg/m^2)		23.67 ± 3.52 23.2 (16.5-27.2)	29.99 ± 3.78 29.7 (21-41.6)	-8.077†	**< 0.001**
IPFP maximum CSA (mm^2)		544.49 ± 120.62	569.66 ± 131.03	-1.118*	0.265
IPFP depth (mm)		25.67 ± 3.99	26.79 ± 3.82	-1.637*	0.104
Medial subcutaneous fat thickness (mm)		17.55 ± 7.31 15 (7-41)	23.57 ± 10.36 21 (5-63)	-3,847†	**< 0.001**
Chondromalacia location	Medial	-	93 (54.4%)	-	-
	Lateral	-	63 (36.9%)	-	-
	Medial+lateral	-	16 (8.7%)	-	-
Chondromalacia grade	Grade 1	-	15 (8.8%)	-	-
	Grade 2	-	60 (35%)	-	-
	Grade 3	-	40 (23.2%)	-	-
	Grade 4	-	57 (33%)	-	-
IPFP signal intensity score	Grade 0	-	57 (33.1%)	-	-
	Grade 1	-	58 (33.8%)	-	-
	Grade 2	-	31 (18%)	-	-
	Grade 3	-	26 (15.1%)	-	-

*: t-value: Independent samples t-test, †: Z-value: Mann-Whitney U test, ‡: X^2-value Pearson chi-square test, BMI: Body mass index, IPFP: Infrapatellar fat pad, CSA: Cross-sectional area, IPSS: Infrapatellar fat pad signal intensity, SD: Standart deviation. *P* < 0.05 considered statistically significant (indicated in bold).

Table 2. Correlation Analysis Between İnfrapatellar Fat Pad Maximum Cross-Sectional Area, Depth, Signal Intensity Scores, and Other Variables

Variables	**IPFP maximum CSA**		**IPFP depth**		**IPFP signal intensity score**	
	r	***P***	**r**	***P***	**r**	***P***
Gender	-0.364	**< 0.001**	-0.338	**< 0.001**	0.033	0.738
Age	0.087	0.291	0.135	0.100	0.50	**< 0.001**
Side	0.058	0.481	0.141	0.086	0.03	0.766
Height	0.045	0.582	0.025	0.763	-0.096	0.335
Weight	0.150	0.066	0.158	0.054	0.106	0.287
BMI	0.097	0.237	0.107	0.192	0.229	**0.020**
Med. fat thickness	-0.150	0.067	-0.273	0.058	0.142	0.152
CM grade	-0.029	0.771	0.037	0.711	0.377	**< 0.001**
CM location	-0.052	0.599	-0.016	0.870	0.022	0.826

IPFP: Infrapatellar fat pad, CSA: Cross-sectional area, BMI: Body mass index, CM: Chondromalacia, med: medial, r: Spearman correlation coefficient; p: Significance level from Spearman correlation analysis (p<0.05 considered statistically significant)

Table 3. Comparison of Infrapatellar Fat Pad Maximum Cross-Sectional Area and Depth According to Gender, Chondromalacia Grades, Chondromalacia Locations, and IPFP Signal Intensity Scores

Variables		IPFP maximum CSA (mean ± SD)	*P* value	IPFP depth (mean ± SD)	*P* value
Gender					
	Male	667.78 ± 134.59	**< 0.001***	29.50 ± 3.49	**< 0.001***
	Female	533.02 ± 109.79		25.78 ± 3.44	
Chondromalacia grade					
	1	558.66 ± 110.73	0.595†	27.00 ± 3.16	0.512†
	2	574.72 ± 129.33		26.00 ± 3.66	
	3	580.00 ± 132.66		27.45 ± 3.82	
	4	559.91 ± 122.11		27.10 ± 4.14	
IPFP signal intensity score					
	0	569.32 ± 150.12	0.838‡	26.08 ± 3.72	0.793†
	1	573.80 ± 139.48		26.84 ± 4.19	
	2	583.57 ± 110.55		27.47 ± 3.82	
	3	543.13 ± 89.20		27.40 ± 3.15	
Chondromalacia location					
	Medial	575.48 ± 110.15	0.537‡	26.83 ± 3.83	0.399†
	Lateral	553.34 ± 152.01		26.43 ± 3.84	
	Medial+lateral	602.33 ± 161.06		28.00 ± 3.77	

*: Independent samples t-test; †: Kruskal-Wallis test, ‡: ANOVA test. $P < 0.05$ (considered statistically significant). IPFP: Infrapatellar Fat Pad, CSA: Cross-sectional area. SD: Standart deviation

This correlation suggests that as BMI increases, there may be a corresponding increase in IPFP inflammation or signal intensity. However, given the weakness of this relationship, BMI alone cannot fully explain the changes in IPFP signal, and other factors such as inflammation, mechanical stress, and synovial fluid composition are likely to contribute.

In our study, no significant relationship was found between BMI and IPFP area or depth. Similarly, Han et al.[17] reported no significant association between IPFP area and BMI. Recent studies from the United States and the Netherlands have reported that BMI is not significantly associated with IPFP volume in either the control or the OA groups.[18,19] The lack of a relationship between IPFP volume and obesity or body weight changes may be explained by the IPFP's anatomical location within the joint capsule, thereby isolating it from systemic circulation. Its limited capacity to absorb circulating free fatty acids and, consequently, its inability to increase in volume by accumulating fat have been proposed as potential mechanisms.[20]

BMI does not accurately assess fat distribution around the body and cannot distinguish adipose tissue from non-adipose body mass, which limits its utility. In contrast, measurements of SCF around the knee provide a more localized assessment of periarticular fat, offering additional information beyond BMI regarding the impact of adipose tissue changes on OA progression.[21] In our study, medial SCF thickness was significantly greater in the patient group with patellar chondromalacia than in the control group ($P < 0.001$).

Studies investigating the relationship between IPFP area and SCF thickness are limited. In our study, no significant associations were observed between medial SCF thickness and IPFP area or depth ($P = 0.067$ and $P = 0.058$, respectively). These findings are consistent with previous reports. Ragab and Serag[22] reported a weak negative correlation between SCF thickness and IPFP area ($r = 0.201$, $P < 0.01$, 95% confidence interval), although this association did not reach significance in multivariate analysis ($P = 0.14$). Similarly, Teichtahl et al.[23] found no significant relationship between body fat measures (subcutaneous fat, total body fat) and IPFP size. These findings suggest that the IPFP may be independent of systemic adiposity. Furthermore, Masaki et al.[20] reported that the IPFP is preserved even under fasting conditions. This observation supports the notion that the IPFP may represent a metabolically distinct fat depot with limited direct association with systemic fat accumulation.

In our study, the mean IPFP area for all patients was 5.69 cm^2 [±1.3 (standard deviation) SD], which is lower than the values reported by Ricatti et al.[1], 7.59 cm^2 (± 1.18 SD), and Ragab and Serag[22], 6.9 cm^2 (± 1.6 SD). This discrepancy may be attributable to differences in the demographic characteristics of the patient populations included in these studies.

In our study, males exhibited significantly greater IPFP maximum cross-sectional area ($r = -0.364$; $P < 0.001$) and depth ($r = -0.338$; $P < 0.001$) than females. This finding is consistent with previous literature. Duran et al.[24] reported a significant difference in mean IPFP area between males and females, with males having larger values ($P < 0.05$). Similarly, Han et al.[17] noted

a negative association between increased IPFP area and female sex. Although the underlying mechanisms linking sex and IPFP volume are not fully understood, sex hormones may play a role in this process. Prior studies have shown that sex hormones influence fat distribution across different anatomical regions.[25]

In our study, no significant relationship was observed between IPFP area or depth and patient age (P = 0.291 and P = 0.100, respectively). Ragab et al.[22] reported a negative correlation between IPFP area and age (r = -0.401, P < 0.001), whereas Han et al.[17] found a significant positive association between IPFP volume and age (P < 0.05). These contrasting findings suggest that the effect of age on IPFP measurements may be complex, and age-related changes in periarticular fat may vary according to individual and population-level factors.

The IPFP has long been considered a structural fat pad with minimal or no metabolic response. However, a meta-analysis by Clockaerts et al.[26] suggested that the IPFP is a complex structure containing nerve fibers, adipocytes, and immune cells, and may contribute to the progression of knee OA through the production and release of inflammatory mediators. In this context, a large-volume IPFP may induce pathological outcomes by altering cytokine release from adipocytes and other pro-arthritic mediators, or by affecting the distribution and magnitude of forces within the PFJ. Conversely, the molecular and local mechanical effects of OA developing in the PFJ may alter the local environment, potentially leading to IPFP enlargement.

Cowan et al.[27] reported that individuals with knee OA predominantly affecting the PFJ exhibited larger MRI-measured IPFP volumes compared to asymptomatic individuals, and greater IPFP volume was associated with more severe pain. In contrast, Han et al.[17] observed that individuals with larger IPFP areas had less radiographic evidence of OA. Similarly, Uludağ and Sirik found that increased IPFP dimensions were associated with a lower prevalence of patellar chondromalacia. Pan et al.[3] suggested that the IPFP may exert protective effects against mechanical and inflammatory knee pain, noting that reduction of the IPFP during knee surgery could lead to increased postoperative pain. Consequently, preservation of the normal IPFP during knee surgery has been emphasized.

These divergent findings in the literature suggest that the IPFP may play a physiologically beneficial role due to its biomechanical or anti-catabolic properties, while simultaneously potentially contributing pathologically through its pro-inflammatory or metabolic characteristics.[4]

The maximum cross-sectional area and depth of the IPFP are commonly used to define IPFP size on MRI.[1] In our study, no statistically significant differences in IPFP area or depth were observed between subjects with and without patellar chondromalacia. This finding suggests that assessing the influence of the IPFP on patellar chondromalacia based solely on dimensional parameters may be insufficient, and analysis of IPFP signal intensity changes reflecting inflammatory mediator release should also be considered. Furthermore, IPFP dimensions may be associated with multiple factors, including OA stage, subtype, and genetic predisposition. Therefore, more in-depth studies are warranted to better characterize the role of IPFP volume in OA progression.

In our study, changes in IPFP signal intensity showed a significant positive correlation with the degree of chondromalacia (r = 0.377; P < 0.001). This finding suggests that the IPFP is not merely a structural fat pad but may also be closely associated with degenerative processes in the knee joint. Our results, together with previous findings in the literature[1,28], indicate that changes in IPFP signal intensity could be considered clinically meaningful parameters, relevant not only for the diagnosis of OA but also for guiding treatment planning. The relationship between IPFP signal intensity changes on MRI and circulating inflammatory factor levels remains an important issue to be addressed in future studies.

Study Limitations

This study has several limitations. First, due to its retrospective design, the clinical information and physical examination findings for the patients were incomplete. Additionally, cartilage damage was assessed only qualitatively, which may have introduced some inaccuracies. Therefore, future studies are planned to include quantitative or semi-quantitative analyses. Due to study constraints, pathological evaluation of synovial inflammation could not be performed, potentially resulting in the overlooking of non-OA causes of synovitis.

CONCLUSION

This study demonstrated a significant relationship between IPFP signal intensity changes on MRI and the degree of patellar chondromalacia. However, the lack of a direct association of IPFP area and depth with chondromalacia suggests that the IPFP should not be evaluated solely based on dimensional measurements. Prospective studies, supported by clinical data and incorporating quantitative analyses, will help better understand the role of the IPFP in knee OA.

Ethics

Ethics Committee Approval: This retrospective study was approved by the Clinical Research Ethics Committee of the Ministry of Health University of Health Sciences Türkiye, Ankara Etlik City Hospital (approval no: 2025/0231, date: 26.02 2025).

Informed Consent: The requirement for informed consent was waived due to the retrospective nature of the study.

Footnotes

Author Contributions

Concept Design – E.Z., B.A., İ.S.D., Data Collection or Processing – E.Z., B.A., İ.S.D., Analysis or Interpretation – E.Z., B.A., Literature Review – E.Z., B.A., Writing, Reviewing and Editing – E.Z., B.A.

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