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Original Article

Three-dimensional volume and shape of the infrapatellar fat pad during quasi-static knee extension from 30° to 0°: comparisons of patients with osteoarthritic knees and young, healthy individuals

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Abstract. [Purpose] Previous studies suggest that the infrapatellar-fat-pad is affected by aging or knee osteoarthritis, and that the infrapatellar-fat-pad in knee osteoarthritis cases may be associated with limited mobility during knee movement. This study aimed to determine changes in the shape and volume of the infrapatellar-fat-pad between 30° and 0° of knee extension in knee osteoarthritis cases and in young, healthy individuals, and to characterize differences in patellar mobility, patellar tendon mobility, and length between the groups. [Participants and Methods] We created 3D models of the infrapatellar-fat-pad, the patellar tendon, and bones using sagittal MRI with the knee at 30° and 0°. The following four parameters were determined: (1) movement of the infrapatellar-fat-pad; (2) infrapatellar-fat-pad volume; (3) angle and surface length of the patellar tendon; and (4) patellar movement. [Results] Compared with the knee osteoarthritis group, the healthy group showed (1) reduced anterior movement of the infrapatellar-fat-pad; (2) smaller volume changes only in the infero-postero-lateral portion; and (3) no changes in the angle of the patellar tendon to the tibial plateau between 30° to 0°. [Conclusion] In conclusion, between 30° and 0°, (1) the infrapatellar-fat-pad in patients with knee osteoarthritis exhibited less anterior movement, and (2) the patellar tendon angle was diminished in patients with knee osteoarthritis compared with those of young-healthy knees.

Key words: Infrapatellar fat pad, Knee, Knee osteoarthritis

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INTRODUCTION

Knee osteoarthritis (KOA) is an irreversible degenerative disease in which the principal complaint is pain during loading, including walking[1)]. Pain and functional deterioration caused by knee osteoarthritis negatively impacts activity of daily living (ADL)[2)]. The morbidity rate of KOA, including a secondary decline in ADL increases with age, causing a heavy burden on patients and society[3)].

The infrapatellar fat pad (IPFP) space, or the space in front of the knee surrounded by the patella, patellar tendon, tibia, femur, and the transverse ligament, should change shape and volume during knee movement. The smallest volume of the IPFP space was measured at full extension and 120°, whereas the maximum volume was observed at 50°[4)]. Fontanella et al.[5)] showed that IPFP volume and depth, as well as length of the femoral and tibial arches (superior and inferior contours of the IPFP in the sagittal image) in end-stage KOAipfp using 3D models derived from MRI were smaller than those of controls.

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Therefore, the IPFP in KOA is required to change its shape to accommodate with the reduced space. These studies only evaluated IPFP in one position and, therefore, shape changes during knee movement were not measured. Understanding how the IPFP changes shape during knee motion in KOA requires measurements of the shape and volume of the IPFP in two or more positions. To this end, a true 3D analysis with different knee positions was required and we reported that the IPFP moved anteriorly during 30° to 0° knee extension, with an increase in volume for the antero-inferior portion of the IPFP in young, healthy individuals[6)]. Therefore, it was desirable to compare the behavior of the IPFP between KOA and healthy knees. The purposes of this study were to determine changes of shape and volume of the IPFP between 0 and 30° knee flexion in patients with KOA and in young, healthy individuals, and to characterize differences in patellar mobility and patellar tendon mobility and length between the two groups.

In order to establish specific study hypotheses, we need to reiterate the characteristics of the IPFP in KOA. Stiffness[7)] and volume[3)] of the IPFP may increase in KOA because of inflammation that causes fibrosis. However, shape changes of the IPFP in KOA are unknown. Artificial adhesion of the patellar tendon and IPFP in cadaveric knees translated the patella more distally by about 5.5 mm at 0°[8)]. The incidence of patella infera and contracture of the IPFP were greater in the "poor" group compared with the "good" group after anterior cruciate ligament reconstruction, in which the poor group involved greater knee flexion contracture[9)]. Steadman et al.[10)] reported that infrapatellar fat pad contracture existed with patients after knee trauma of surgery. They reported arthroscopy release could improve intractable anterior knee pain and knee range with patients after that. Arthroscopic release for symptomatic scarring of the anterior interval of the knee. Infrapatellar fat pad contracture might be in knee osteoarthritis patients too, not only patients with a history of knee trauma or previous surgery. Therefore, improving mobility of the IPFP may increase the mobility and shape changes and/or reduce patella infera. In order to help establish a treatment strategy to reduce the effects of IPFP stiffness and optimize knee movement in KOA, a comparison between KOA and young, healthy knees which demonstrate optimal knee motion would be required since age-comparable control may have pre-existing stiffness or volume changes due to aging alone.

The hypothesis of this study was that the movement of the IPFP during 30°–0° knee extension in KOA would be smaller than that in young, healthy knees. The second hypothesis was that there was limited associations between the movement of the IPFP and shape change of IPFP, patellar mobility, the angle of the patellar tendon, the shape changes of patellar tendon, or femorotibial movement in KOA than those in healthy knees. Understanding the movement of the IPFP in KOA should help to improve our treatment strategy to modify the IPFP mobility during knee extension. Understanding the function of IPFP mobility in KOA may help improve the treatment strategy for patients with KOA.

PARTICIPANTS AND METHODS

This was a cross-sectional study comparing KOA and healthy control groups (Fig. 1) and the level of evidence was III. After obtaining approval from the local ethics committee (ethics committee of Sadamatsu hospital 13-1-11), we recruited patients with KOA and young healthy people to serve as controls. Selection criteria for the KOA group were: 1) age 40 to 79 years at the time of recruitment, 2) Kellgren–Laurence (K/L) grade of 1 to 4, and 3) no history of surgery or fracture of either lower limb. Selection criteria for a healthy group were: 1) age 20 to 25 years, 2) healthy knees, 3) normal range of motion, and 4) no history of surgery or fracture of either lower limb. Common exclusion criteria for both groups were: 1) problems with communication, 2) difficulty in understanding the research, 3) pregnancy, 4) medical risks, or 5) rheumatoid arthritis. All of participants agreed to participate in this study. Twelve KOA patients (3 males, 9 females) averaged 61.73 ± 8.58 years of age and the eight healthy individuals (4 males, 4 females) averaged 22.0 ± 2.0 years.

Movement of the IPFP was quantified in cross-section for both groups using 3D models of the patella, patellar tendon, femur, tibia and IPFP from MRI taken at 0 and 30° knee flexion. Outcomes were (1) movement of the IPFP, (2) volume

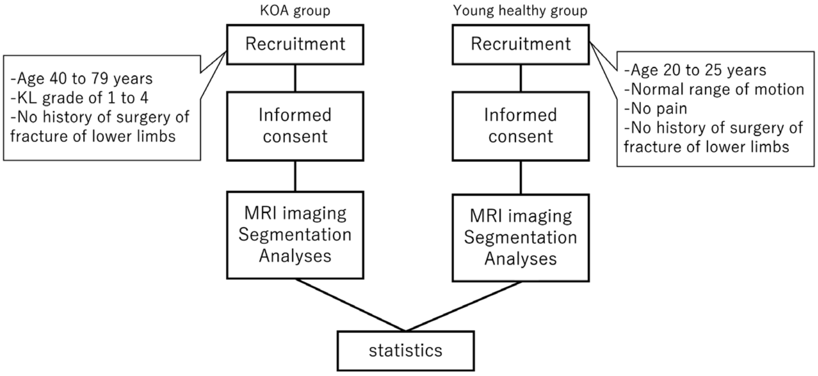

Fig. 1. Study design.
KOA: knee osteoarthritis; KL grade; Kellgren–Laurence grade; MRI: magnetic resonance imaging.

change of the IPFP, (3) mobility of the patella, (4) changes in the surface length of the patellar tendon, (5) changes in the patellar tendon angle relative to the tibia, and (6) the femorotibial movement. Body mass index (BMI), K/L grade, Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) and Q-angle were measured at baseline.

Briefly, the methods of creating MRI-based models are outlined blow and detailed methodology was described in our previous publication[6]. MRI of the knee was taken using a 0.3T APERTO (Hitachi Medical Corporation, Hitachi, Tokyo Japan) at 0° and 30° knee flexion in the supine position (The imaging sequence was 3-dimensional T1 of sagittal images with a slice pitch of 1 mm spanning 250 mm across the knee (TR:3700 TE:90)). 3D coordinate systems (The X, Y and X axis) were embedded in the femur, tibia, and patella using commercial 3D-Aligner software (GLAB Corp., Higashihiroshima, Japan). Knee positions corresponding to the six degrees-of-freedom of the tibia were calculated with regard to the femoral 3D coordinate system with reference to the study of Andriacchi[11].

Movement of the bones and IPFP are calculated as outlined below[6]. For movement of the IPFP (Fig. 2), we calculated the antero-posterior position of the anterior surface point of the IPFP using a coordinate system and the value got by the IPFP position at 30° subtracted from the IPFP position at 0°. For volume change of the IPFP (Fig. 3), the IPFP model was divided into eight portions by three planes (the sagittal, horizontal and coronal plane) and the divided IPFP models in each hyperoctant at 30° were subtracted from the divided IPFP models at 0° to determine the volume changes in each hyperoctant. Mobility of the patella was calculated corresponding to the 6 degrees-of-freedom and orientations of the patella at 0°, those at 30° using the tibial coordinate system. For surface length of the patella tendon was measured the curved surface of it using 3D models. The distance from the patellar inferior pole to the tibial tuberosity (Fig. 4) was obtained using 3D model and this reflects patellar height. The patellar tendon angle was calculated from the sagittal plane using Image J and the patellar tendon angle of 30° were subtracted from those of 0°. For mobility of the femur, positions and orientations of the femorotibial joint were calculated corresponding to the 6 degrees-of-freedom and orientations of the femur at 0°, those at 30° using the tibial coordinate system.

Chi-square tests were used to assess differences in male and females between the groups, because it is just to compare the difference in the number of males versus females. The significance level was set at alpha=0.05. Comparisons of 0° and 30° flexion positions were performed using paired t-tests with Bonferroni correction based on Shapiro–Wilk test. Three pairwise comparisons involving intra-group comparisons for both groups, as well as an inter-group comparison of changed values between 30° to 0° knee extension. Therefore, the final alpha was 0.0167 after correction. Associations between IPFP

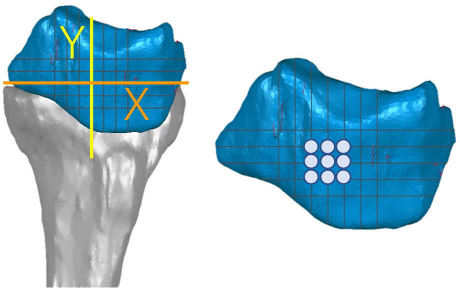

Fig. 2. Definition of the nine points at the anterior surface of the infra-patellar fat pad (IPFP)

Using the tibial coordinate system, 9 crossing points were defined onto the anterior surface of the IPFP. The antero-posterior position of the IPFP (or IPFP position) was obtained from the coordinates of the 9 crossing points.

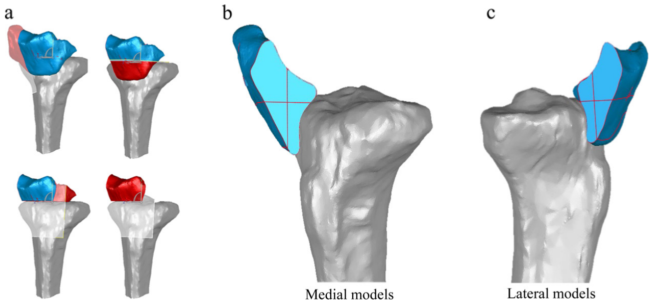

Fig. 3. Definition of 8 hyperoctants for calculation of infrapatellar fat pad (IPFP) volume using the tibial coordinate system,

The infrapatellar fat pad (IPFP) model was divided into eight hyperoctants by three planes, XY plane (or sagittal plane), ZX plane (or horizontal plane) and YZ plane (coronal plane) parallel to through the most anterior surface of the tibial tubercle.

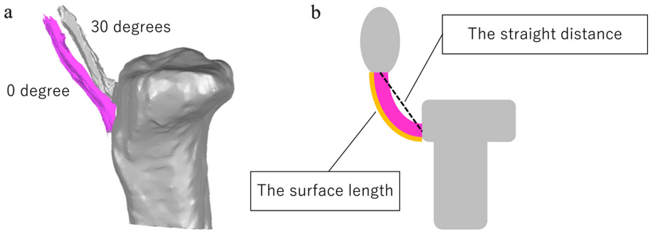

Fig. 4. Definition of distance between the inferior pole of the patella and tibial tuberosity, surface length of the patella tendon.

a. Pink represents the patellar tendon at 0°, gray at 30°.

b. Black dashed line represents the straight distance between the attachments, and yellow the surface of the patellar tendon.

displacement and volume and other factors were compared using Pearson's correlation coefficient or Spearman's correlation coefficient. The significance level was set at alpha=0.05. SPSS Ver.14 (IBM, Chicago, IL, USA) was used for statistical tests.

RESULTS

The information of the participants is shown in Table 1. And all outcomes are shown in Table 2. We included fourteen medial KOA and one lateral KOA. In both the KOA and healthy groups, the surface coordinates of the IPFP moved anteriorly from 30° to 0° knee extension (Fig. 5). Mean displacements of the IPFP for the KOA and healthy groups were 1.7 [0.4, 3.1] mm (95% CI) (p=0.023) and 5.8 [3.1, 8.4] mm (p=0.005), respectively (Table 2). The healthy group showed a significant anterior displacement of the IPFP from 30° to 0° knee extension, but the KOA group did not. Mean anterior movement in the KOA group (1.7 mm) was significantly less than in the healthy group (5.8 mm), in which the difference between the two groups was 4.0 [1.2, 6.9] mm (p=0.008).

The KOA group showed an increase in IPFP volume in the postero-infero-lateral hyperoctant and a decrease in the postero-superior quadrant during 30° to 0° knee extension (Fig. 6). There was an increase in IPFP volume in the postero-infero-lateral hyperoctant from 2,384.7 [1,920.4, 2,848.9] to 2,695.3 [2,261.5, 3,129.1] mm^3 (p=0.007) from 30° to 0° knee extension. There were also decreases in IPFP volume in the postero-supero-lateral hyperoctant from 4,300.9 [3,354.7, 5,247.1] to 2,781.4 ([2,039.7, 3,523.1] mm^3 (p=0.003), and in the postero-supero-medial hyperoctant from 1,796.2 [1,046.2, 2,546.2] to 851.4 mm^3 ([469.5, 1,233.3], p=0.006).

The healthy group showed an increase in IPFP volume in the antero-inferior hyperoctants and a decrease in the postero-supero-lateral hyperoctant during 30° to 0° knee extension. There were increases in IPFP volume in the antero-infero-lateral hyperoctant from 1,650.6 [1,015.9, 2,285.2] to 2,797.8 [2,017.1, 3,578.4] mm^3 (p=0.015), in the antero-infero-medial hyperoctant from 602.7 [454.9, 750.5] to 2,161.5 [1,338.5, 2,984.5] mm^3 (p=0.004), and in the anterior-supero-medial hyperoctant from 6,182.4 [4,484.9, 7,879.9] to 8,175.9 [6,416.9, 9,934.9] mm^3 (p<0.001) from 30° to 0° knee extension. There was a decrease in IPFP volume in the postero-supero-lateral hyperoctant from 6,926.2 [5,219.6, 8,632.8] to 3,283.8 [1,910.5, 4,657.1] mm^3 (p<0.001).

Significant patellar anterior translation, superior translation, and anterior rotation occurred in both groups. The patella translated medially in the healthy group from 30° to 0° knee extension, but not significantly in the KOA group. Patellar medio-lateral position (where positive numbers indicate medial positions) in the healthy group moved from −6.6 [−9.6, −3.6] mm to −3.2 [−6.1, 0.1] mm (p=0.003), exhibiting a 3.5° medial translation during knee extension. That in the KOA group moved from −4.1 [-6.4, −1.8] mm to −5.1 [−8.8, −1.4] mm (p=0.401), exhibiting a 1.0-mm lateral translation during knee extension. There was no association between the patellar mobility and the amount of anterior IPFP movement in the KOA or healthy groups.

Patellar tendon angles were also measured based on the tibial coordinate system. Patellar tendon angles at 30° and 0° knee extension for the KOA group were 30.8 [25.7, 35.8]° and 30.1 [26.1, 34.1]° (p=0.834), respectively. Those for the healthy group were 29.7 [26.6, 32.8] ° and 35.5 [31.4, 39.5]° (p=0.014), respectively. Therefore, the healthy group showed a significant increase in the patellar tendon angle from 30° to 0° knee extension. There was no relationship between the patellar tendon angle and amount of anterior IPFP movement in the KOA (r= 0.418, p=0.121) or healthy group (r=−0.129, p=0.872).

Surface lengths of the patellar tendon at 30° and 0° knee extension in the KOA group were 52.3 [47.8, 56.9] mm and 48.2 [45.8, 50.6] mm (p=0.035), respectively. Those in the healthy group were 53.4 [48.5, 58.5] mm to 49.9 [46.3, 53.5] mm (p=0.016), respectively. Significant differences were observed only in the healthy group. There was no correlation between the surface length of the patellar tendon and the degree of anterior IPFP movement in the KOA (r=0.039, p=0.891) or healthy groups (r= 0.332, p=0.466).

Table 1. Demographic data for all participants

	OA group	Healthy group	p-value
Male	3	4	-
Female	12	4	0.166†
Age (years)*	61.7 ± 8.6	22.0 ± 2.0	<0.001‡
BMI (kg/m^2)*	22.5 ± 6.8	21.5 ± 2.5	0.710‡
K/L I	1	-	-
K/L II	11	-	-
K/L III	1	-	-
K/L IV	2	-	-

*: data are presented as the mean ± standard deviation.
†: χ^2 test; ‡: Student t-test.
OA: osteoarthritis; BMI: body mass index; K/L: Kellgren–Lawrence grade.

Distances from the patellar inferior pole to the tibial tuberosity at 30° to 0° knee extension for the KOA group were 50.2 [46.2, 54.2] mm and 47.2 [44.8, 49.6] mm (p=0.083), respectively. Those for the healthy group were 76.3 [68.7, 83.9] mm and 68.1 [63.5, 72.7] mm (p=0.108), respectively. There was no reciprocity between the distance from the patellar inferior pole to the tibial tuberosity and the extent of anterior IPFP movement in either the KOA (r= 0.114, p=0.685) or healthy group (r=0.069, p=0.872).

There were significant differences during knee extension in femoral superior translation, internal rotation, and extension in both groups, while no differences were observed between the groups. The femorotibial joint showed abduction in the healthy

Table 2. Results of each assessment

		Healthy group				Knee OA group				p-value
		0°	30°	Difference	p-value (Intra-group difference)	0°	30°	Difference	p-value (Intra-group difference)	(Inter-group difference)
IPFP movement (mm)		44.5	38.7	5.8	**0.005**	38.9	37.1	1.7	0.023	**0.008**
IPFP volume[*1]	Antero-supero-medial (mm^3)	8,175.3	6,182.4	1,993.0	**<0.001**	4,698.0	4,465.9	232.1	0.499	**0.002**
	Postero-supero-medial (mm^3)	2,021.5	3,345.5	−1,324.0	0.146	851.4	1,796.2	−944.8	**0.006**	0.598
	Antero-supero-lateral (mm^3)	6,877.2	7,904.4	−1,027.3	0.090	5,022.4	4,668.2	354.2	0.267	0.023
	Postero-supero-lateral (mm^3)	3,283.8	6,926.2	−3,642.4	**<0.001**	2,781.4	4,300.9	−1,519.5	**0.003**	**0.004**
	Anterp-infero-medial (mm^3)	2,161.5	602.7	1,558.8	**0.004**	846.1	694.4	151.6	0.385	**0.001**
	Postero-infero-medial (mm^3)	910.1	712.3	197.8	0.187	378.9	353.3	25.6	0.626	0.166
	Antero-infero-lateral (mm^3)	2,797.8	1,650.6	1,147.2	**0.015**	1,541.0	1,104.0	437.0	0.059	0.058
	Postero-infero-lateral (mm^3)	2,335.0	1,967.4	367.5	0.134	2,695.3	2,384.7	310.6	**0.007**	0.786
Patellar tendon	Distance (mm)[*2]	68.1	76.3	−8.2	0.108	47.2	50.2	−3.0	0.083	0.197
	Angle (°)	35.5	29.7	5.8	**0.014**	30.1	30.8	−0.7	0.834	0.164
	Surface length (mm)	49.9	53.4	−3.5	0.159	48.2	52.3	−4.1	0.035	0.422
Patella	Anterior translation (mm)	53.8	48.4	5.3	**<0.001**	48.2	46.1	2.1	**0.002**	**0.003**
	Suferior translation (mm)	29.8	32.5	−2.6	**0.008**	28.9	31.5	−2.6	**<0.001**	0.980
	Medial translation (mm)	−3.2	−6.6	3.5	**0.003**	−5.1	−4.1	−1.0	0.401	**0.004**
	Internal rotation (°)[*3]	2.4	−0.1	2.5	0.190	−2.0	−1.4	−0.6	0.609	0.144
	External tilt (°)[*4]	6.3	11.4	−5.1	0.040	5.6	4.9	0.7	0.708	0.074
	Anterior rotation (°)	15.8	7.9	7.9	**0.017**	12.7	3.5	9.2	**<0.001**	0.687
Femur	Anterior translation (mm)	−1.2	−2.8	1.6	0.133	−3.8	−4.0	0.2	0.805	0.248
	Superior translation (mm)	26.4	24.3	2.0	**0.003**	25.2	22.2	3.0	**0.005**	0.524
	Medial translation (mm)	1.3	0.4	0.9	0.078	0.6	0.2	0.4	0.316	0.347
	Abduction (°)	1.5	−1.1	2.6	**0.012**	1.5	−0.1	1.6	0.030	0.413
	Internal rotation (°)	3.6	−2.8	6.5	**<0.001**	−0.5	−3.8	3.3	**0.004**	0.062
	Extension (°)	12.3	−14.6	26.9	**<0.001**	8.7	−13.2	21.9	**<0.001**	0.161

IPFP: infrapatellar fat pad.

[*1]: IPFP was divided into 8 hyperoctants based on the tibial coordinate system and the data show the volume of IFPF in each hyperoctant.

[*2]: Distance between the inferior pole of the patella and tibial tuberosity.

[*3]: Lateral translation is the frontal plane rotation with the inferior pole directed medially.

[*4]: External tilt is the horizontal plane rotation with the lateral boarder moving posteriorly.

Bold numbers indicate a significant change with p-value less than 0.0167.

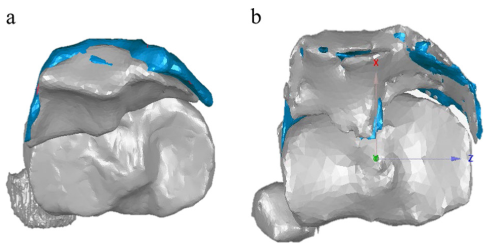

Fig. 5. The anterior movement of the infra-patellar fat pad (IPFP) during the quasi-static knee extension from 30 to 0°.
a: Healthy knee, b: Osteoarthritis knee.

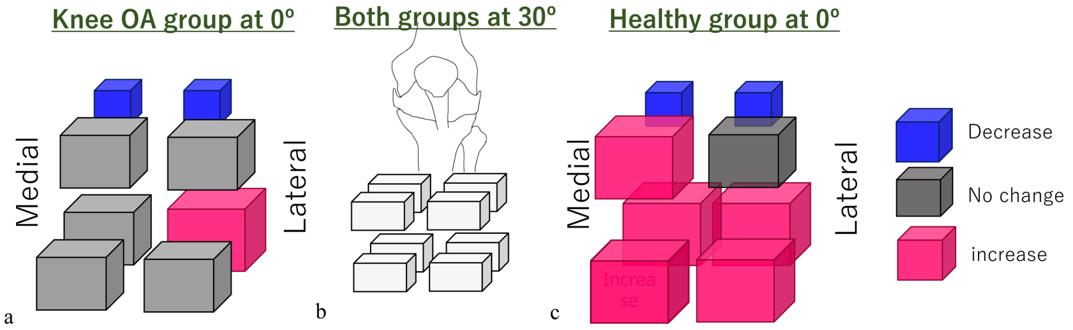

Fig. 6. The infrapatellar fat pad (IPFP) volume at 0° of knee osteoarthritis (OA) and healthy groups at both sides using divided cube models representing relative volume of the eight portions of the IPFP as compared with the equally-sized cubes at 30° at the middle.
a. At 0 degree of Knee OA group.
b. At 30° of both groups.
c. At 0 degree of Healthy group.

group from −1.1 [−1.9, −0.3] mm at 30° to 1.5 [0.2, 2.8] mm at 0° (0.012) exhibiting 2.6° abducted translation during knee extension, but not significantly in the KOA group. The amount of femoral internal rotation in KOA group was 3.3 ° from −3.8[-6.0, −1.6] ° at 30° to −0.5[−1.3, 0.5] ° at 0°($p=0.004$). The amount of femorotibial extension in KOA group was 21.9° from −13.2[−16.3, −10.1] ° at 30° to 8.7[5.9, 11.5] ° at 0°($p<0.001$). The amount of femoral superior translation in the KOA group was 3.0 mm, from 22.2 [20.5, 23.9] mm at 30° to 25.2 [24.2, 26.2] mm at 0° ($p=0.005$). No differences were observed in femoral anterior or medial translation. There was no association between femoral movement and anterior IPFP movement in either KOA or healthy patients.

DISCUSSION

The first hypothesis of this study was that movement of the IPFP during 30° to 0° knee extension would be reduced in KOA, compared to young, healthy knees. Overall, the anterior movement of the IPFP was smaller during 30° to 0° knee extension in the KOA group. IPFP volume of the postero-infero-lateral hyperoctant increased in the KOA group, while the volume of the postero-supero-lateral and postero-supero-medial hyperoctants decreased, suggesting that there was less movement of the posterior part of the IPFP. On the other hand, IPFP volume in the healthy group increased in the antero-infero-lateral, antero-infero-medial, and anterior-supero-medial hyperoctants, while the volume of the postero-supero-lateral hyperoctant decreased, suggesting that the IPFP moved anteriorly.

The second hypothesis was that association between the shape change of the IPFP and patellar mobility, the angle of the patellar tendon, the shape changes of patellar tendon, and tibial mobility would be weaker in KOA than in healthy knees. The patellar tendon angle in the healthy group changed significantly, but not in the KOA group. The healthy group demonstrated greater IPFP movement and greater change in the patellar tendon angle than did the KOA group. However, no significant association was observed between IPFP movement and change in the patellar tendon angle.

We selected young, healthy individuals as the control group because asymptomatic early KOA (healthy aged individuals) and radiographic early KOA are largely overlapped, and we are unaware of changes in the IPFP movement can be the cause or result of KOA. If reduced movement of the IPFP comes first, a comparison between the age-matched groups would not answer the research question. Aging induces fibrotic changes and reduced movement of the body structures such as muscles, ligaments, skin, etc.[12–14], which led us to assume the fibrous connective tissue around the IPFP suffer reduced movement from aging. On the other hand, comparisons between young healthy individuals and patients with KOA allow us to determine how the behavior of the IPFP would change from the optimal knee.

The IPFP moved anteriorly in both groups during 30° to 0° knee extension, and the amount of IPFP motion was significantly reduced in the KOA group. The KOA group demonstrated inferior movement only in posterior IPFP sections, while the healthy group showed anterior movement of the IPFP. Fascia containing the IPFP attach to the inferior patellar pole, the femoral intercondylar notch, the proximal patellar tendon, the inter-meniscal ligament, and both menisci[15]. The shape of the anterior knee compartment, surrounded by the patella, femur, tibia, and patellar tendon, reportedly changes during knee movement, according to a cadaveric study[4]. Therefore, the IPFP, filling the anterior knee compartment, needs to change shape to accommodate changes in the shape of the anterior compartment during knee flexion and extension. Restricted changes in IPFP shape may cause abnormal kinematics or restricted mobility of the patellofemoral and femorotibial joints. Contracture of the IPFP or "infrapatellar contracture syndrome (IPCS)" is considered one of the causes of knee contracture[16].

The KOA group exhibited poor IPFP shape change, suggesting the presence of IPFP contracture9). Accordingly, limited mobility of the IPFP may be one of the causes of knee contracture in KOA.

The KOA group demonstrated limited movement of the patella and patellar tendon, and a shorter distance between the inferior pole of the patella and the tibial tuberosity. These findings might have resulted from the limited shape change of the IPFP. In the literature, the relationship between patella infera and KOA is unclear. Ahmand et al.8) simulated IPFP contracture by creating adhesion of the IPFP and the patellar tendon in cadaveric knees, and patella infera was observed between 0° and 60°. Paulos et al.16) reported a series of cases showing that patients with anterior knee pain had patella infera. Therefore, patellar infera may be an indicator of IPFP contracture and would be associated with anterior knee pain. On the other hand, KOA demonstrates characteristic knee kinematics. Farrokhi et al.17) used dynamic stereo X-rays to characterize knee kinematics during gait in patients with KOA, and found reduced flexion and internal/external rotation excursion and increased total abduction/adduction excursion in the stance phase. Stationary stepping in patients with severe KOA showed knee adduction and lateral translation motion in the early stance phase18, 19). Specific kinematics in patients with medial KOA during squatting involved tibial external rotation, posterior translation, and adduction20). Meanwhile, Bastiaansen-Jenniskens et al.7) reported a possible association between inflammation and fibrosis, which enhances adhesion. Therefore, IPCS may be induced by inflammation associated with KOA and/or anterior knee pain, and may cause further kinematic changes with reduced patellofemoral and tibiofemoral mobility.

3D models were created by manual segmentation using MRI images taken at a slice pitch of 1 mm. We superimposed 3D models created from two knee positions and analyzed the differences. Analytical methods used in this study proved to be highly reliable in a previous study6). Participants in the healthy group were individuals who had no history of knee pain or surgery on either lower limb, whereas subjects in the KOA group were recruited from among patients diagnosed as KL grades 1 to 4, based on X-ray examinations by orthopedic surgeons at our hospital. Therefore, the external validity of this study was limited to the patients with KOA. This study is the first to quantify and compare dynamics of the IPFP between 30° and 0° knee extension in patients with KOA and in healthy controls. The effect size of this study was 1.45 based on the results of the movement of the IPFP. The post-hoc power analysis was performed and required sample size was fourteen, and therefore, there is a potential beta-errors in our results.

There were five limitations in this study. First, small sample size may have introduced beta errors. Second, joint positions at 30° and 0° knee extension were determined using a goniometer, which might have introduced measurement errors. Exact knee extensions in the KOA and healthy groups using the coordinate systems were 21.9° and 26.9°, respectively. Third, we compared the healthy and KOA groups cross-sectionally, so we are unable to determine whether differences between the two groups were caused by a longitudinal process. Fourth, comparisons of different ages between the two groups involved a limitation to determine whether the osteoarthritic process is the only cause of changes in IPFP volume and mobility. However, we wanted to reveal how much difference in IPFP exists between young, healthy individuals and KOA patients, in order to reveal how much IPFP movement can be restored after an intervention with reference to an ideal condition of the IPFP. Fifth, during MRI scanning, participants were in the supine position with relaxed muscles around the knee. Therefore, results of this study should be applied only to passive knee movement, but not to active knee movement or weight-bearing conditions. Despite the above limitations, there were no apparent sources of bias that would invalidate the conclusions.

During 30° to 0° knee extension, the anterior surface of the IPFP in KOA exhibits less anterior movement than in healthy subjects. The change of the patellar tendon angle from 30° to 0° knee extension showed the same trends in KOA compared with the healthy group. These results suggest IPFP contracture exist in patients with KOA, and that restoring its movement has to be included in therapeutic strategies. These findings should help clinicians to treat the knee to optimize IPFP movement, which may help to improve the symptoms of KOA.

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Conflicts of interest

All authors declare that they have no conflicts of interest.

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