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Pattern of joint damage in persons with knee osteoarthritis and concomitant ACL tears

Verena Stein,

Division of Research, New England Baptist Hospital, Boston, MA, USA

Ling Li,

Division of Research, New England Baptist Hospital, Boston, MA, USA

Grace Lo,

Division of Rheumatology, Tufts University School of Medicine, Boston, MA, USA

Ali Guermazi,

Boston University School of Medicine at Boston Medical Center, Boston, MA, USA

Yuqing Zhang,

Boston University School of Medicine at Boston Medical Center, Boston, MA, USA

C. Kent Kwoh,

University of Pittsburgh and Pittsburgh VA Healthcare System, Pittsburgh, PA, USA

Charles B. Eaton, and

Warren Alpert Medical School of Brown University, Providence, RI, USA

David J. Hunter Division of Research, New England Baptist Hospital, Boston, MA, USA

Northern Clinical School, University of Sydney, Sydney, NSW, Australia

Abstract

Anterior cruciate ligament (ACL) tears are known to be a risk factor for incident knee osteoarthritis (OA). At the present time, it is unknown whether an incidental ACL tear in those with established knee OA alters the pattern of synovial joint damage. Therefore, our aim was to assess whether ACL tears in persons with knee OA are associated with specific patterns of cartilage loss, meniscal degeneration, and bone marrow lesion (BML) location. We included 160 participants from the progression subcohort of the Osteoarthritis Initiative (OAI) Study, an ongoing 4-year, multicenter study, focusing on knee OA. Regional cartilage morphometry measures including cartilage volume (mm³), denuded area, normalized cartilage volume, bone surface area, as well as location of meniscal pathology and BMLs in index knees on the same side were compared between those with and without ACL tears. Of the 160 subjects (51% women, age $62.1 (\pm 9.9)$, BMI $30.3 (\pm 4.7) \text{ kg/m}^2$), 14.4% had an ACL tear. After adjusting for age, BMI and gender participants with ACL tears had significantly greater cartilage volume in the posterior lateral femur (P = 0.04) and the central medial tibia (0.001) compared to those without ACL tears. Normalized cartilage volume was not different between those with and without ACL tears in the medial tibia

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Correspondence to: David J. Hunter.

David.Hunter@sydney.edu.au.

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(P=0,006), the central medial tibia (P=0.008), the posterior lateral femur (P=0.004), and the posterior medial femur (P=0.04). Furthermore, participants with ACL tears showed significantly more meniscal derangement in the lateral posterior horn (P=0.019) and significantly more BMLs in the lateral femur (P=0.0025). We found clear evidence of predominant lateral tibiofemoral involvement, with OA-associated findings on MRI, including increased denuded area and bone surface area, BMLs, and meniscal derangement in knees of individuals with ACL tears compared to those without.

Keywords

Knee osteoarthritis; Anterior cruciate ligament tear; Cartilage loss; Bone marrow lesions; Meniscal pathology

Introduction

Osteoarthritis (OA) is the leading cause of disability in older adults [1]. Although radiographic features such as joint space narrowing (JSN) and the presence of osteophytes define the presence of OA [2], new technologies such as magnetic resonance imaging (MRI) may improve the assessment of early disease development and progression. MRI has shown a higher sensitivity and specificity for the diagnosis of acute traumatic lesions within the knee and its early-stage post-traumatic degenerative changes [3–5] and the assessment of joint morphology [6, 7].

Knee injuries are an important risk factor for the development of knee OA [8–11]. Rupture of the anterior cruciate ligament (ACL) occurs more often at a younger age [12, 13] with an incidence for different sports between 18/1,000 players in soccer [14] and 1/1,000 game hours in handball [15]. Persons with a rupture of the ACL are at an elevated risk for knee OA [16–20], shown severalfold in young, athletic persons [14, 17, 20–22], due to subsequent anteroposterior instability of the knee.

There are some data suggesting that persons with ACL tears are at increased risk for cartilage loss [23, 24]; however, there are little data on the association of ACL tears with meniscal degeneration or bone marrow lesions (BMLs) in persons with OA. The presence of both of these structural features could facilitate further structural deterioration if ACL tears increased their frequency and severity.

In knees with acute ACL tears, it has been shown that chondral lesions seem to appear more frequently in the lateral tibiofemoral compartment [25]. Furthermore, the contact pattern of ACL-injured knees compared to healthy controls was shown to be more posterior in acutely injured knees, and this seemed to be associated with severity of symptoms [26, 27].

Therefore, the aim of this study was to explore the pattern of articular damage and the relationship to bone interface in persons with OA and ACL tears by testing the following hypothesis.

ACL tears in persons with knee OA will be associated with increased cartilage loss, meniscal degeneration, specific patterns of BMLs location, and other bony changes. This pattern of damage would be consistent with the predominant initial location of the osteochondral injury in those with ACL tears in the lateral tibiofemoral compartment.

Materials and methods

Study sample

The Osteoarthritis Initiative (OAI) is an ongoing 4-year, multicenter, longitudinal, prospective observational cohort study designed to identify biomarkers of the development and/or progression of knee OA. On an annual basis, 4,796 participants undergo detailed evaluation, including knee MRIs, fixed-flexion knee radiographs, detailed questionnaires on pain, demographic information, and physical examination (e.g., BMI). The local institutional review boards approved and reviewed the study protocol, amendments, and informed consent documentation. The data for this research were drawn from the OAI and are available for public access at http://www.oai.ucsf.edu/. The specific datasets used are clinical dataset 0.1.1 and Image Release 0.B.1.

The OAI consists of a progression subcohort, from which our sample is drawn, and an incidence subcohort. A total of 1,389 participants with radiographic signs (radio-graphic evidence of OA (ROA) defined as definite tibiofemoral osteophytes (OARSI atlas grade 1) on X-ray) and symptoms of knee OA, such as pain, stiffness and aching on most days of a month during the past year at baseline, were recruited for the progression subcohort. Subjects with severe narrowing (OARSI grade 3 narrowing or bone on bone) in both knees were excluded. The X-ray readings of osteophytes and joint space narrowing were taken at each OAI Clinical Center.

For the purposes of this study, a group of 160 participants was selected from the progression subcohort of the OAI, due to previously available data including high-quality MRI readings for ACL tears and data on chondral morphometry. For this convenience, sample of 160 participants, imaging data from baseline with the abovementioned data from a previous investigation and imaging data from one-year follow-up, was available. The detailed selection criteria for these participants and the index knee have been described previously [28].

Assessment of joint injury

History of joint surgery was evaluated at the enrollment visit by asking the participants whether they ever had any kind of knee surgery including arthroscopy, ligament repair surgery or meniscectomy, or they ever had a hip replacement surgery. In addition, participants were asked whether they ever injured their knee(s) badly enough to limit their ability to walk for at least 2 days. Results from this survey for our study population can be found in Table 2.

Radiographic assessment

A SynaFlexerTM frame (Synarc, Inc., San Francisco, CA) was used to obtain bilateral posteroanterior (PA) views of the knee to position the subject's feet reproducibly. The acquisition protocol required that the participant's body weight was distributed equally between their two legs and knees, and their thighs were pressed directly against the wall of the frame. The anterior wall of the frame was in contact with the Bucky or reclining tabletop of the radio-graphic unit. The goal of this positioning was to achieve a fixed angle of knee flexion of about 20°. Additionally, a V-shaped angulation support on the base of the frame was used to fix the foot below the index knee in 10° external rotation. The X-ray beam is angled 10° caudal [29].

For a prior study [28], the baseline radiographs of these 160 subjects had been read independently by one bone and joint radiologist and a rheumatologist (DH). The readers were blinded to sequence and performed paired readings of the knee X-rays using the

Kellgren and Lawrence (KL) grade on a 0–4 scale [30]. If disagreements occurred, they used adjudicated readings that were arrived at by a consensus of both readers, for KL grade and JSN.

Selection of knee for analysis

Both the presence of symptoms (frequent knee pain) and ROA were required to be present in the same knee, to meet the inclusion criteria. There were 100 participants with unilateral OA that met these criteria. For those participants with bilateral symptomatic knee OA, we chose the knee with moderate disease and with a higher chance of disease progression. In individuals with KL grade 2 or 3 in both knees, we employed the following features to rank the risk of progression for both knees in order to select the knee at greatest risk for medial tibiofemoral progression. If knees still ranked equal after applying the first feature, we moved to the next feature listed below:

greater anatomic axis varus angulation,

2.0 mm medial minimum joint space width (JSW),

greater grade of medial JSN (grade 1-3),

the presence of any medial tibial or femoral osteophyte grade 2 with greater grade than lateral osteophytes,

the presence of any medial tibial or femoral osteophyte, and finally

the right knee

If neither of the knees was graded KL grade 2 or 3, the knee with the higher KL grade was chosen. If the participant was graded KL 0, 1, or 4 on both knees, then the same process described earlier was followed.

MRI sequence parameters

Images were acquired on identical 3 T MRI scanners (Siemens Magnetom Trio, Erlangen, Germany) using a quadrature transmit-receive knee coil (USA Instruments, Aurora, OH). For the purposes of reading BMLs, we used the sagittal intermediate-weighted (IW) turbo spin echo (TSE) fat-suppressed (FS) images. The Dual Echo in the Steady State (DESS) sequences was used to assist with the localization of some lesions. The menisci were scored with the same sequences and in addition on the 2D coronal IW TSE sequences. For the purpose of the cartilage segmentation, an unsupervised segmentation was done using the DESS sequences. Sequence parameters can be found in detail in Table 1 and also on the OAI homepage http://www.oai.ucsf.edu/.

MRI readings

The bilateral MRIs from the enrollment visit for 160 participants included in this study were obtained from the OAI Coordinating Center.

Bone marrow lesions (BMLs)—BMLs and meniscal derangement were evaluated in two different reading sessions by a rheumatologist (GHL) with expertise in musculoskeletal MRI readings and centrally involved in the development of the Boston Leeds Osteo-arthritis Knee Score (BLOKS) [31]. This reader was blinded to subject data and scored each knee MRI for BMLs and MRI meniscal derangement using the BLOKS grading system. The intra-rater reliability was kappa = 0.88 and was assessed by reading a sample of 10 knee MRIs twice by the same reader, at least 2 days separated from each other.

On the sagittal IW TSE FS images (Table 1), a BML was described when seeing an illdefined hyperintense signal in the subchondral bone, proximal to the epiphyseal line (Fig. 1). Additionally, the DESS sequences (Table 1) were consulted to assist with the localization of lesions. The BMLs were evaluated for size on the IW TSE FS images from 0 to 3 at each of the following locations using BLOKS [31]: medial and lateral patella, medial and lateral trochlea, medial and lateral weight-bearing femur, medial and lateral tibia, and subspinous tibia with 0 = none, 1 = <10% of the whole bone volume, 2 = 10-25% of the whole bone volume, and 3 = >25% of the whole bone volume.

Only those BMLs with greater than 25% of the surface area adjacent to the subchondral plate were included. We classified large BMLs as those with BLOKS score 2.

Menisci—The same sagittal IW TSE FS (Table 1) images as well as the coronal 2D IW TSE images (Table 1) were used to score the meniscal integrity. Those menisci with disruption of the overall morphology of the meniscus and diffuse hyperintense signal in the body of the meniscus were defined as MRI meniscal derangement. The meniscal derangement was graded in each of the following locations: the anterior horn, body, and posterior horn of the medial and lateral menisci. To assess intra-rater reliability, a sample of 10 knee MRIs were read for MRI meniscal derangement twice by the same reader separated by at least 2 days for a simple kappa of 0.87.

ACL tears—The presence of an ACL tear at baseline was read using sagittal and coronal views and scored on a 0–2 scale, with 0 = normal, 1 = partial tear, and 2 = complete tear. A complete tear was defined as complete disruption of ACL fibers with ligament discontinuity, while a partial tear was defined as residual straight and tight ACL fibers in at least one-pulse sequence, while the anteromedial and posterolateral bundle of the ACL were not evaluated separately in the coronal slices. All ACL tears were read by one board-certified musculoskeletal radiologist (AG) (intra-reader weighted kappa = 0.75), separate from the scoring of other articular features and unaware of the hypothesis being tested. Due to the fact that even partial tears may change the bio-mechanics, and therefore the pattern of joint damage and the small sample size, we combined partial and complete tears.

Cartilage morphometry—For the analysis of the cartilage parameters, we used the DESS sequences. The segmentation was semiautomated and has been described in detail elsewhere [28].

The following measures were evaluated after the segmentation of the images in different areas of the knee joint:

- 1 Cartilage volume.
- 2 Normalized cartilage volume (volume normalized to bone surface interface area).

The bone surface interface area is the area of the cartilage in contact with bone. The normalization was done by dividing the measured cartilage volume by the area of measured cartilage in contact with bone plus the area of full thickness defects (denuded area of bone).

- **3** Denuded area (total cartilage bone interface area denuded of cartilage). The denuded area is the area of bone where a full-thickness cartilage defect is present.
- 4 Bone interface area (total bone surface area, regardless of cartilage denudation)

Depiction of the different areas is shown in Fig. 2.

Statistical analysis

Statistical analyses were performed using SAS software (SAS Institute Inc., Cary, NC, release 8.2). Descriptive statistics were performed using a nonparametric Wilcoxon test for continuous variables and Chi-square tests for categorical variables. The sample was divided into those with ACL tears and those without. We examined whether an ACL tear was associated with increased meniscal degeneration, specific patterns of BMLs location, bone morphology, and cartilage morphometry in the index knee compared with the index knee of those without an ACL tear. We adjusted the analysis of cartilage volume, normalized cartilage volume, and bone cartilage interface surface areas for confounders including age, BMI, and gender. Since denuded area was not normally distributed, we used a quantile regression model—a nonparametric analysis method while adjusting for age, BMI, and gender. The analysis of BMLs and meniscal degeneration were not adjusted for any of the above-mentioned parameters or multiple testing due to a cell frequency of less than five in multiple cells and the nonsignificant result in the simple relationship test at most locations.

Results

The demographic characteristics of the overall study sample and those with and without ACL tears are shown in Table 2. Fifty-one percent of the study population were women, the average age of the subjects was 62.1 years with a standard deviation (SD) of 9.9 years, and 59% of the participants were 65 years of age or older. The mean BMI was 30.3 kg/m² with a SD of 4.7. The left knee was picked as the index knee in 78 participants (49%). Fifteen percent of the participants in this study sample did not meet the criteria of ROA defined by a centralized reading of a KL grade 2. The eligibility criteria used for the OAI progression subcohort were based on the identification of a definite tibiofemoral osteophyte at each OAI Clinical Center, and some disagreement in radiographic assessment with the adjudicated central reading of KL grades was expected.

A partial or complete ACL tear was present in 23 study participants, with 10 ACLs being graded as partially torn and 13 as completely torn, and 22 of these participants reporting a history of substantive knee injury and 2 participants showed an ACL repair that was graded as an intact ACL. For reasons of statistical power, we decided to combine those with complete and partial tears of the ACL.

Results of the cartilage volume, normalized cartilage volume, denuded area, and bone interface area in participants with and without ACL tears are shown in Table 3. Individuals with ACL tears had significantly greater cartilage volume in the following locations: femur plate (P = 0.008), posterior lateral femur (P = 0.004), posterior medial femur (P = 0.006), central lateral femur (P = 0.012), and central lateral tibia (P = 0.024). After adjusting for age, gender, and BMI, only the central medial tibial (P = 0.04) and the posterior lateral femur (P = 0.001) remained significant.

Analysis of the denuded area (total cartilage bone interface area denuded of cartilage) also showed significantly greater denuded area in persons with ACL tears in the lateral tibia plate (0.0004), central lateral tibia (P = 0.003), and central lateral femur (P = 0.03). After adjusting for age, gender, and BMI, none of these regions remained significant.

There were no significant differences in any region, however, when the cartilage volume was normalized to the bone surface area. Therefore, we also examined the bone interface area between participants with and without ACL tears and found a significantly larger bone interface area in participants when compared to those without ACL tears. The regions with significantly greater bone cartilage interface areas were the femur (P= 0.01), lateral tibia (P = 0.04), central lateral femur (P= 0.01), cent

femur (P = 0.01), and posterior medial femur (P = 0.01). After adjusting for age, gender, and BMI, the medial tibia (P = 0.006), central medial tibia (P = 0.008), posterior lateral femur (P = 0.004), and the posterior medial femur (P = 0.04) remained significant.

Analysis of the meniscal pathology showed significantly (P = 0.019) more meniscal derangement in the lateral posterior horn when comparing participants with to those without ACL tears (Table 4).

As shown in Table 5, participants with ACL tears had larger BMLs in the lateral femur (P = 0.0025) compared to those without.

Discussion

In this study of 160 participants with knee OA, we demonstrated an altered pattern of cartilage loss, increased bone area, meniscal degeneration, and location of BMLs between participants with and without ACL tears. This study demonstrated a clear predisposition to lateral tibiofemoral articular damage in persons with concomitant OA and ACL tear.

With acute tears of the ACL being one of the most common severe sports injuries [32], the fact that patients with ACL injuries may also suffer from associated chondral and subchondral damages, as well as meniscal injuries, has been extensively investigated [33–37].

Several recent studies [27, 38–41] suggested a relation between ACL injuries and cartilage damage predominant in the lateral compartment. In a radiographic case–control study, Swaerd et al. [38] compared the location of structural changes of 176 post-traumatic patients with 155 controls who suffered from knee pain without prior injury. JSN and osteophytes were graded accordingly to the OARSI atlas, and the results showed a lateral shift of structural changes in the traumatic group.

Nishimori et al. [41] reported a high proportion of cartilage injuries in the lateral femur and lateral tibial plateau in addition to the posterior horn of the lateral meniscus in 39 patients with acute ACL tears. Even though this study design did not provide a noninjured control group, the arthroscopically described pattern of cartilage and meniscal injury after acute tears of the ACL is similar to that found in our study.

Tibiofemoral contact pattern between 31 participants with and without ACL tears was investigated in an MRI study by Scarvell et al. [27]. The lateral compartment loading pattern in ACL-injured knees was more posterior on the tibial plateau than in the healthy knees, and the different loading pattern was accompanied by more severe knee symptoms. These findings relate to the structural changes in our study, which may be determined by changed kinematics after unrepaired ACL injury.

Meniscal injuries are common findings in ACL-injured patients. Both lateral and medial meniscal tears in ACL-injured knees are described [42–44]. Two studies reported a relationship between lateral meniscal damage and acute injury, and between medial injuries in chronic ACL tears. Frequency differences in meniscal tears may be explained by various reasons, including different injury mechanisms, alignment, and timing of investigation with regards to the time point of the injury. Several investigators have demonstrated an association between ACL tears and the presence of meniscal tears, especially medially [24, 39, 45]. While any injury to the knee could result in concurrent meniscal damage and ACL tear, some have demonstrated that ACL tears themselves may cause secondary meniscal damage [39, 44, 45], which, in turn, could contribute to cartilage loss, BML formation, and

potentially increasing malalignment. The extent of this risk needs to be clarified in order to ascertain whether this should be the target of future therapeutic interventions.

Bone bruises in association with acute ACL tears are very common [46–50] and have been investigated in several MRI studies [51–53], with the common finding of increased bruises in the lateral compartment. Additional histological research [51] showed chondrocyte degeneration, proteoglycan loss, and osteochondral necrosis in the bruise overlying cartilage. These findings correspond with our results on subchondral BMLs, which were significantly higher in the lateral femur for participants with ACL tears.

In addition to the changes in the acutely injured knee, the presence of osteoarthritic findings in the knee joint after ACL injury have been investigated in several long-term studies [14, 18, 54–56]. Jones et al. [57] summarized how acute and chronic ACL injuries associated with chondral and meniscal loss may lead to OA of the knee.

In a recent study by Amin et al. [58], cartilage loss in 49 participants with ACL tears over 15 or 30 months was compared to that in 216 without an ACL tear over the same period. Unadjusted odds ratios showed a significantly higher risk for cartilage loss in the medial compartment for those with ACL tears. However, after adjusting for age, BMI, gender, and meniscal damage, no increase in risk for cartilage loss could be detected. Our study results suggest that in participants with a predominant ACL tear, the pattern of cartilage loss changes toward the pattern found in acute ACL-injured knees. Therefore, ACL tears may act as an influencing factor regarding to the location and pattern of osteoarthritic changes. In our study, we found some differences in cartilage volume (with increase volume in certain regions), but after normalizing for area (analogous to cartilage thickness), these differences were attenuated and no longer reached statistical significance. These findings, in addition to the differences we found in bone surface area, seem to indicate that much of the morphologic adaptation to ACL injury occurs in bone rather than cartilage morphometry.

Our findings of significantly higher meniscal maceration in the lateral posterior horn in participants with ACL tears suggest a higher risk for lateral disease progression in patients with symptomatic knee OA and a prevalent ACL tear.

The location of BMLs may also determine progression pattern of knee OA in patients with ACL tears. In our study, the lateral predominance of BML in participants with ACL tears underlines the lateral propensity in post-traumatic knee OA.

Limitations

The frequency of ACL tears in this study sample was smaller than previously described in the literature with 13 (8.1%) complete ACL tears and 10 (6.25%) partial ACL tears and a tear rate of 14.4% in the whole study population. Three previous studies investigating ACL tears with other findings in knee OA presented rates from 22% [23], over 28% [59] to 35% [60], for full-thickness tears. This decreased prevalence of ACL tears may be related to the selection factors used to define the 160 participants chosen from OAI study to part of this substudy. Also 85% of the participants in our study had KL grades 2, which may influence this discrepancy. Another concern may be misclassification bias in the defining an ACL tear. The fidelity of MRI in ACL diagnosis has an accuracy between 90 and 100% [61–64] compared to the gold standard knee arthroscopy but is yet to be demonstrated in patients with knee OA. Such misclassifications. Regarding the statistical power of our analysis, the sample size of 160 participants with 23 suffering from a complete or a partial tear is a rather small sample size; hence, the results need to be confirmed in a larger cohort. Previous studies [27, 41, 58] investigating joint changes in participants with ACL tears did have a

similar sample size to our study. As a result of the cross-sectional design of the study, we are unable to make any cause and effect conclusions based on our findings and more longitudinal research is needed.

Conclusion

In summary, we found a significant lateral tibiofemoral tendency of OA-associated findings including BMLs, increased bone surface area, and meniscal derangement in participants when compared to those without ACL tears. These findings suggest prevalent ACL tears in symptomatic OA patients may be an indicator for a specific pattern and location of disease progression. Further longitudinal research is necessary to corroborate our findings of the pattern of articular damage in persons with OA and ACL tears.

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Fig. 1.

Sagittal view of the medial tibiofemoral compartment with large ill-defined subchondral bone marrow lesions in the tibia and femur (*white arrows*). There is extensive cartilage loss in the weight-bearing tibia and femur and partial maceration of the posterior horn of medial meniscus





MRI slices and schematics of depicting the cartilage areas. *Top panel*: lateral sagittal slice of the human knee. *Middle panel*: medial sagittal slice of the human knee. *Bottom panel*: coronal slice of the human knee

Table 1

MRI sequence parameters: also see OAI homepage http://www.oai.ucsf.edu

MRI parameters			
	2D TSE	2D TSE	DESS
Weighting	Int	Int	T2
Plane	Coronal	Sagittal	Sagittal
Fat sat	No	Yes	WE
Matrix (phase)	307	313	307
Matrix (freq)	384	448	384
No. of slices	41	37	160
FOV (mm)	140	160	140
Slice thickness (mm)	3	3	0.7
Skip (mm)	0	0	0
Flip angle (°)	180	180	25
TE/TI (ms)	29	30	4.7
TR (ms)	3,850	3,200	16.3
BW (Hz/pixel)	352	248	185
Chemical shift (pixel)	1.3	0	0
NAV (NEX)	1	1	1
Echo train length	7	5	1
Phase encode axis	R/L	S/I	A/P
Phase partial fourier $(8/8 = 1)$	1	1	0.875
Readout partial fourier $(8/8 = 1)$	1	1	1
Slice partial fourier $(8/8 = 1)$	1	1	0.875
Options	Elliptical k-space filter and large FOV filter	Elliptical k-space filter and large FOV filter	Elliptical k-space filter, elliptical sampling, and lager FOV filter
Distance factor (%)	0	0	0
Phase oversampling	20	4	0
Slice oversampling	0	0	10
Phase resolution	80	70	80
Averaging technique	Short term	Short term	Short term
Gradient rise time	Fast	Fast	Fast
RF amplitude	Normal	Normal	Fat
X-resolution (mm)	0.365	0.357	0.365
Y-resolution (mm)	0.456	0.511	0.456
Calc time (min)	3.4	4.7	11.2
Scan time (min)	3.4	4.7	10.2

Table 2

Descriptive characteristics of study sample (n = 160)

	All (<i>n</i> = 160)	No ACL tear (<i>n</i> = 137)	ACL tear $(n = 23)$
Gender (female <i>n</i> (%))	81 (51)	76 (56)	5 (22)
Age (mean, SD) years	62.1 (9.9)	62.9 (9.5)	57.5 (10.8)
Age group (65 years $n(\%)$)	95 (59)	77 (56)	18 (78)
BMI (mean, SD) kg/m ²	30.3 (4.7)	30.3 (4.7)	30.5 (4.8)
Index knee (left $n(\%)$)	78 (49)	69 (50)	9 (39)
Kellgren and Lawrence grade of index knee, no. (%)			
0	6 (4)	6 (4)	0 (0)
1	18 (11)	17 (12)	1 (4)
2	58 (36)	51 (37)	7 (30)
3	64 (40)	54 (39)	10 (44)
4	14 (9)	9 (7)	5 (22)
History of knee and hip surgery including arthroscopy (yes for study knee n (%))	45 (28.9)	31 (22.6)	14 (60.9)
History of knee injury (yes for study knee n (%)) ever injured badly enough to limit ability to walk for at least 2 days	75 (47.2)	53 (38.7)	22 (95.7)

Table 3

parameters and bone surface area between patients with and without ACL tears

volume a	at baseline (mm ³)			Denuded area at	baseline (mm²)			Normalize	ed cartilage volume at l	oaseline		Bone cartilage ir	nterface surface Areas	(mm ²)	
, Mean 1)	W/ACL Mean (Std)	Unad <i>p</i>	Adj <i>p</i>	W/o ACL Median (IQR)	W/ACL Median (IQR)	Unadj <i>p</i>	$\operatorname{Adj} p$	W/o ACL Mean (Std)	W/ACL Mean (Std)	Unadj <i>p</i>	Adj <i>p</i>	W/o ACL Mean (Std)	W/ACL Mean (Std)	Unadj <i>p</i>	$\operatorname{Adj} p$
3001.6)	14043.3 (3927.8)	0.008	0.26	58.1 (1.8, 376.4)	74.5 (36.3, 353.7)	0.25	0.20	2.3 (0.3)	2.5 (0.5)	0.18	0.95	4869.2 (830.6)	5423.8 (840.1)	0.01	0.35
719.0)	2555.6 (738.0)	0.06	0.53	$1.1\ (0.0,\ 1.1)$	2.9 (1.1, 30.0)	0.0004	0.69	2.3 (0.4)	2.3 (0.4)	0.60	0.15	967.3 (187.8)	1068.0 (216.2)	0.04	66.0
637.4)	2145.3 (23.1)	0.11	0.15	3.4 (0.0, 8.5)	3.4 (0.0, 131.5)	0.56	1.00	1.8 (0.3)	1.9 (0.5)	0.21	0.54	1039.1 (208.7)	1035.2 (242.2)	0.51	0.006
214.3)	3180.0 (1 = 0.0)	0.06	0.71	$1.9\ (0.0,\ 61.1)$	0.06 (0.0, 103.3)	0.68	0.92	2.2 (0.8)	2.5 (0.8)	0.20	0.89	1050.0 (304.7)	1175.5 (263.3)	0.07	0.89
440.1)	4969.1 (1002.0)	0.30	0.28	$5.4\ (0.0,58.0)$	12.4 (0.1, 43.6)	0.55	0.32	2.3 (0.5)	2.5 (0.7)	0.35	0.73	1841.8 (387.2)	1918.5 (304.4)	0.47	0.18
524.6)	2066.9 (\$0.3)	0.01	0.16	$0.6\ (0.0,\ 0.6)$	$0.6\ (0.3, 5.0)$	0.03	1.00	2.2 (0.4)	2.3 (0.5)	0.16	0.55	765.0 (138.2)	862.5 (142.4)	0.01	0.10
571.9)	2240.3 (第2.6)	0.02	0.46	$1.1 \ (0.0, 1.1)$	1.1 (1.1, 19.8)	0.003	0.93	2.3 (0.5)	2.4 (0.4)	0.41	0.21	779.6 (152.6)	905.7 (210.8)	0.01	0.06
(8.069	1755.3 (898.6)	0.12	0.76	7.1 (0.0, 66.5)	7.1 (0.0, 101.1)	0.69	1.00	1.8 (0.7)	2.0 (0.8)	0.21	0.99	730.7 (212.0)	767.9 (193.3)	0.29	0.21
503.9)	1370.1 ($\overline{\underline{32}}$ 7.8)	0.41	0.04	$3.3\ (0.0,\ 8.3)$	3.3 (0.0, 131.3)	0.51	1.00	1.7 (0.4)	1.8 (0.6)	0.29	0.33	713.1 (193.3)	682.0 (223.6)	0.95	0.008
441.7)	2139.9 (899.9)	0.004	0.001	$0.0\ (0.0,\ 0.0)$	$0.0\ (0.0,\ 0.0)$	0.17	1.00	2.5 (0.4)	2.7 (0.5)	0.22	0.62	622.5 (142.4)	777.0 (233.8)	0.01	0.004
464.0)	2047.5 (\$7.9)	0.006	0.10	$0.3\ (0.0,\ 0.3)$	0.3 (0.0, 2.7)	0.27	1.00	2.3 (0.4)	2.4 (0.5)	0.74	0.68	716.7 (143.3)	830.2 (198.4)	0.01	0.04
062.2)	3802.8 (1555.1) ii	0.04	0.56	7.4 (0.0, 66.5)	7.4 (0.0, 110.0)	0.56	0.42	4.1 (1.0)	4.4 (1.1)	0.38	0.85				
160.3)	3125.3 (1 1	0.191	0.30	10.4 (0.0, 87.9)	$10.4\ (0.0,235.5)$	0.75	0.42	3.5 (1.1)	3.8 (1.4)	0.23	0.70				
ed cartila;	ge volume ad the for	age, BMI, ⁶	und gende	ır											

ed cartulage volume ad Bated for age, BMI, and gender < 0.05 level M . 10 . 10 Page 17

Table 4

Meniscal pathology at baseline between participants with and without ACL tears

NoYesYesNo 80 61.5 10 50.0 No 80 61.5 10 50.0 No 80 61.5 10 50.0 No 80 8.5 10 50.0 Medial hody derangement 10 20.0 No 42 32.3 4 20.0 No 42 32.3 4 20.0 No 39 30.0 2 10.0 Ves 91 70.0 18 90.0 No 110 84.6 10 50.0 Ves 20 15.4 1 5.0 No 103 79.2 16 80.0 Ves 27 20.8 4 20.0 Ves 27 20.8 11 55.0 No 105 80.8 11 50.0 No 105 80.8 11 55.0		ACL t	ear			Fisher's exact test P values
hFercent h FercentMedial anterior derangementNo 61.5 10 50.0 No 80 61.5 10 50.0 0.33 Yes 50 38.5 10 50.0 0.33 Medial body derangement 10 50.0 0.30 Yes 88 67.7 16 80.0 0.31 Yes 88 67.7 16 80.0 0.31 Wo 39 30.0 2 10.0 0.31 Yes 91 70.0 18 90.0 0.31 Yes 91 70.0 18 90.0 0.31 Yes 91 70.0 18 90.0 0.31 Yes 20 15.4 1 5.0 0.31 Yes 20 15.4 1 5.0 0.31 Yes 20 10.3 79.2 16 80.0 Yes 27 20.8 4 20.0 1.00 Yes 20 10.3 79.2 10.2 10.0 Yes 20 10.3 79.2 10.2 10.0 Yes 20.8 10.7 55.0 10.0 10.0 Yes 10.5 80.8 10.7 50.0 Yes 10.5 10.5 10.2 10.0		No		Yes		
Medial anterior derangement 0.03 No 80 61.5 10 50.0 0.33 Yes 50 38.5 10 50.0 0.33 Medial body derangement 20.0 20.0 0.31 No 42 32.3 4 20.0 0.31 Yes 88 67.7 16 80.0 0.31 Medial posterior derangement 20.0 2 100 0.31 Ves 91 70.0 18 90.0 0.10 Yes 91 70.0 18 90.0 0.31 Ves 91 70.0 18 90.0 0.31 Ves 20 1.5 1 5.0 0.31 Ves 20 1.5 1 5.0 0.31 Ves 27 20.8 4 20.0 $1.0.0$ Ves 27 20.8 11 5.0 $1.0.0$ Ves 27 20.8 11 5.0 $1.0.0$		u	Percent	u	Percent	
No 80 61.5 10 50.0 0.33 Yes 50 38.5 10 50.0 0.33 Medial body derangement 23.3 4 20.0 0.31 No 42 32.3 4 20.0 0.31 Yes 8 67.7 16 80.0 0.31 Medial posterior derangement 16 80.0 0.10 0.31 Yes 91 70.0 18 90.0 0.31 Yes 91 70.0 18 90.0 0.31 Yes 91 70.0 18 90.0 0.31 Yes 10 84.6 1 5.0 0.31 Yes 20 15.4 1 5.0 1.00 Yes 20.3 10.3 79.2 16 1.00 1.00 Yes 27 20.8 4 20.0 1.00 1.00 1.00 Yes 10.3 <td< td=""><td>Medial ant</td><td>erior de</td><td>rangement</td><td></td><td></td><td></td></td<>	Medial ant	erior de	rangement			
Yes5038.51050.0Medial body derangement \mathbf{N}_0 42 32.3 4 20.0 No 42 32.3 4 20.0 0.31 Medial posterior derangement 16 80.0 0.01 No 39 30.0 2 100 0.10 Yes 91 70.0 18 90.0 0.10 Ves 91 70.0 18 90.0 0.10 Yes 91 70.0 18 90.0 0.31 Ves 10 84.6 19 95.0 0.31 Ves 20 15.4 1 50 0.31 Yes 20 10 80.0 100 100 Yes 27 20.8 4 20.0 10.0 No 105 80.8 11 55.0 0.02 No 105 80.8 11 55.0 0.02 Yes 28 19.2 9.2 0.02 0.02 Yes 102 80.8 11 50 0.02	No	80	61.5	10	50.0	0.33
Medial body derangement 0.31 No 42 32.3 4 20.0 0.31 Yes 88 67.7 16 80.0 0.31 Medial posterior derangement 16 80.0 0.0 0.10 Yes 91 70.0 18 90.0 0.10 Ves 91 70.0 18 90.0 0.10 Ves 91 70.0 18 90.0 0.10 Ves 91 90.0 10.0 95.0 0.01 Ves 20 15.4 1 5.0 0.01 Ves 20 10 95.0 0.01 0.01 Ves 27 20.8 4 20.0 1.00 Ves 27 20.8 11 55.0 0.02 No 105 80.8 11 55.0 0.02 Yes 27 29.2 45.0 0.02 0.02	Yes	50	38.5	10	50.0	
No 42 32.3 4 20.0 0.31 Yes 8 67.7 16 80.0 0.31 Medial posterior derangement 8 67.7 16 80.0 0.31 Ne 39 50.0 2 10.0 9 0.10 0.10 Yes 91 70.0 18 90.0 0.31 0.31 Lateral anterior derangement 95.0 95.0 0.31 Yes 20 15.4 1 5.0 0.31 0.31 Yes 20 15.4 1 5.0 0.31 0.31 Yes 20.3 79.2 16 80.0 1.00 1.00 Yes 27 20.8 4 20.0 1.00 1.00 1.00 Yes 105 80.8 11 55.0 1.00 1.00 1.00 1.00	Medial bod	ly deran	igement			
Yes88 67.7 1680.0Medial posterior derangement 6 6 0 0 No 39 30.0 2 100 0 Yes 91 70.0 18 90.0 0 Lateral anterior derangement 10 84.6 19 95.0 0.31 Ves 10 84.6 19 95.0 0.31 Yes 20 15.4 1 5.0 0.31 Ves 20 102 79.2 16 80.0 No 103 79.2 16 20.0 1.00 Yes 27 20.8 4 20.0 10.0 No 105 80.8 11 55.0 0.02 Yes 27 29.2 9.2 10.0 10.0 Yes 28 19.2 9.60 0.02	No	42	32.3	4	20.0	0.31
Medial posterior Arrangement 0.00 2 0.00 0.10 No 39 30.0 2 10.0 0.10 Yes 91 70.0 18 90.0 0.10 Lateral anterior derangement 95.0 95.0 0.31 Yes 20 15.4 1 5.0 Lateral body derangement 1 5.0 0.31 No 103 79.2 16 80.0 Yes 27 20.8 4 20.0 Vateral body derangement 1 55.0 1.00 Yes 27 20.8 11 55.0 No 105 80.8 11 55.0 Yes 25 19.2 9 45.0	Yes	88	67.7	16	80.0	
	Medial pos	terior d	erangement			
Yes 91 70.0 18 90.0 Lateral anterior derangementNo 110 84.6 19 95.0 0.31 Yes 20 15.4 1 5.0 0.31 Lateral body derangement 10 80.0 1.00 No 103 79.2 16 80.0 1.00 Yes 27 20.8 4 20.0 Lateral posterior derangement 4 No 105 80.8 11 55.0 Yes 25 19.2 9 45.0	No	39	30.0	2	10.0	0.10
Lateral anterior derangement No 110 84.6 19 95.0 0.31 Yes 20 15.4 1 5.0 0.31 Lateral body derangement 1 5.0 0.30 No 103 79.2 16 80.0 1.00 Yes 27 20.8 4 20.0 1.00 Ves 27 20.8 4 20.0 1.00 Lateral posterior derangement 11 55.0 0.02 0.02 Yes 25 19.2 9 45.0 0.02	Yes	91	70.0	18	90.06	
No 110 84.6 19 95.0 0.31 Yes 20 15.4 1 5.0 0.31 Lateral body derangement 5.0 15.4 1 5.0 No 103 79.2 16 80.0 1.00 Yes 27 20.8 4 20.0 1.00 Lateral posterior derangement 4 20.0 1.00 No 105 80.8 1 55.0 0.02 Yes 25 19.2 9 45.0 0.02	Lateral ant	erior de	rangement			
Yes 20 15.4 1 5.0 Lateral body derangement No 103 79.2 16 80.0 1.00 Yes 27 20.8 4 20.0 1.00 Lateral posterior derangement 4 20.0 1.00 Yes 27 80.8 11 55.0 0.02 Yes 25 19.2 9 45.0 0.02	No	110	84.6	19	95.0	0.31
Lateral body derangement 103 79.2 16 80.0 1.00 No 103 79.2 16 80.0 1.00 Lateral posterior derangement 4 20.0 1.00 No 105 80.8 11 55.0 Yes 25 19.2 9 45.0	Yes	20	15.4	-	5.0	
No 103 79.2 16 80.0 1.00 Yes 27 20.8 4 20.0 1.00 Lateral posterior derangement No 105 80.8 11 55.0 Yes 25 19.2 9 45.0 0.02	Lateral bod	ly deran	igement			
Yes 27 20.8 4 20.0 Lateral posterior derangement No 105 80.8 11 55.0 0.02 Yes 25 19.2 9 45.0 0.02	No	103	79.2	16	80.0	1.00
Lateral posterior derangement 6.02 No 105 80.8 11 55.0 0.02 Yes 25 19.2 9 45.0 0.02	Yes	27	20.8	4	20.0	
No 105 80.8 11 55.0 0.02 Yes 25 19.2 9 45.0 0.02	Lateral pos	terior d	erangement			
Yes 25 19.2 9 45.0	No	105	80.8	11	55.0	0.02
	Yes	25	19.2	6	45.0	

Table 5

Size and location of BMLs in participants with and without ACL tears

Location	BML size	ACL	tear			Fisher's exact test P values
		No		Yes		
		u	Percent	u	Percent	
Lateral trochlea	0	80	61.5	13	65	0.09
	1	16	12.3	2	10	
	2	8	6.2	4	20	
	3	26	20	-	5	
Lateral femur	0	103	79.2	6	45	0.003
	1	21	16.2	٢	35	
	2	ŝ	3.8	0	10	
	3	1	0.8	0	10	
Medial trochlea	0	111	85.4	18	90	0.92
	1	10	7.7	-	5	
	2	4	3.1	-	5	
	3	S	3.8	Ι	ļ	
Medial femur	0	61	46.9	٢	35	0.13
	1	44	33.8	٢	35	
	2	15	11.5	9	30	
	3	10	7.7	I	I	
Lateral patella	0	74	56.9	13	65	0.45
	1	22	16.9	ю	15	
	2	11	8.5	ю	15	
	3	23	17.7	-	5	
Medial patella	0	100	76.9	18	90	0.63
	1	15	11.5	7	10	
	2	3	2.3	I	I	
	3	12	9.2	T	I	
Lateral tibia	0	87	6.99	10	50	0.28
	1	31	23.8	٢	35	

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Location	BML size	ACL	tear			Fisher's exact test P values
		0N0		Yes		
		u	Percent	u	Percent	
	2	9	4.6	7	10	
	3	9	4.6	-	5	
Medial tibia	0	41	31.5	З	15	0.14
	1	47	36.2	٢	35	
	2	27	20.8	4	20	
	3	15	11.5	9	30	
Subspinous tibia	0	66	76.2	15	75	0.69
	1	23	17.7	4	20	
	2	5	3.8	I	I	
	3	ю	2.3	-	5	
Bold values are sign	nificant at $P <$	0.05 le	vel			