Title

Evolution of Intrameniscal Signal-Intensity Alterations Detected on MRI Over 24 Months in Patients With Traumatic Anterior Cruciate Ligament Tear.

Journal

American Journal of Roentgenology, 208(2)

ISSN

0361-803X

Authors

Guimaraes, Julio Brandao
Facchetti, Luca
Schwaiger, Benedikt J
et al.

Publication Date

2017-02-01

DOI

10.2214/ajr.16.16625

Peer reviewed

Evolution of Intrameniscal Signal-Intensity Alterations Detected on MRI Over 24 Months in Patients With Traumatic Anterior Cruciate Ligament Tear

Julio Brandao Guimaraes[1,2]
Luca Facchetti[1,3]
Benedikt J. Schwaiger[1]
Alexandra S. Gersing[1]
Sharmila Majumdar[1]
Benjamin C. Ma[4]
Xiaojuan Li[1]
Thomas M. Link[1]

OBJECTIVE. The objective of our study was to assess the prevalence and evolution of intrameniscal signal-intensity alteration in subjects with an anterior cruciate ligament (ACL) tear over 24 months and compare clinical outcome and changes of cartilage between subjects with and those without this meniscal abnormality.

MATERIALS AND METHODS. Fifty-seven subjects with an ACL tear were screened for intrameniscal signal-intensity alteration. Morphologic and compositional MRI was performed before ACL reconstruction and 12 and 24 months after ACL reconstruction. Twelve subjects with an intrameniscal signal-intensity alteration and 12 subjects without any meniscal abnormality on MRI were identified. Clinical outcome was measured using the Knee Injury and Osteoarthritis Outcome Score (KOOS), and T1ρ and T2 maps of the cartilage were obtained.

RESULTS. In 10 of 12 subjects (83%) the meniscal signal-intensity abnormality identified on baseline MRI was located at the posterior horn of the medial meniscus. None of these subjects presented with a meniscal tear over 24 months of follow-up. At 12 months after the ACL tear, the intrameniscal signal-intensity alteration detected on baseline MRI had completely resolved in seven of 12 subjects (58%), showed a signal-intensity decrease in four (33%), and remained stable in one subject (8%). Of the 10 subjects who underwent MRI at 24 months, the meniscal signal-intensity alteration had completely resolved in eight (80%), and the signal intensity had decreased in the other two subjects. Changes in the KOOS and cartilage T1ρ and T2 values from baseline and 24 months did not differ significantly between subjects with and those without intrameniscal signal-intensity alteration ($p > 0.05$).

CONCLUSION. High intrameniscal signal-intensity alterations are a common finding in subjects with an ACL tear and have a benign course over 24 months after surgical repair of the ACL tear.

Keywords: anterior cruciate ligament (ACL) tear, meniscal contusion, MRI

DOI:10.2214/AJR.16.16625

Received April 14, 2016; accepted after revision August 16, 2016.

Supported by NIH/NIAMS P50 AR060752.

[1]Department of Radiology and Biomedical Imaging, University of California, San Francisco, 185 Berry St, Ste 350, San Francisco, CA 94158. Address correspondence to J. B. Guimaraes (Julio.BrandaoGuimaraes@ucsf.edu).

[2]Department of Radiology, Federal University of Sao Paulo, Sao Paulo, Brazil.

[3]Department of Radiology, University of Brescia, Brescia, Italy.

[4]Department of Orthopedic Surgery, University of California, San Francisco, San Francisco, CA.

AJR 2017; 208:1–7

0361–803X/17/2082–1

An anterior cruciate ligament (ACL) tear is a common and serious knee injury; 250,000 new ACL ruptures are estimated to occur in the United States each year, most of which occur in young people [1]. More than 70% of ACL injuries occur as a result of a noncontact mechanism, and they frequently occur as a result of landing from a jump and lateral cutting maneuvers used in different athletic activities such as basketball, soccer, skiing, and football. The majority of ACL injuries occur in men because a greater number of men participate in sports; however, women have a higher risk of being injured [2]. Besides common comorbidities such as abnormalities of the collateral ligaments, abnormalities of the posterolateral ligament complex, or cartilage defects, different degrees of meniscal injuries commonly occur in conjunction with ACL tears [3]. The reported incidence of meniscal injury varies considerably, ranging from 16% to 82% in patients with acute ACL tears and up to 96% in those with chronic ACL tears [3, 4]. The lateral meniscus is injured more often in patients with acute ACL tears, and the medial meniscus is more likely involved in those with chronic ACL tears [4, 5].

MRI is a sensitive noninvasive method for assessing joint morphology [6, 7] and is highly accurate in depicting ACL tears and associated abnormalities, especially of the soft-tissue structures of the joint [8, 9]. With arthroscopy being considered the standard of reference, MRI shows a high sensitivity (93% for the medial meniscus and 79% for the lateral meniscus) and specificity (88% for the medial meniscus and 96% for the lateral meniscus) for the detection of meniscal tears

[10]. The detection of meniscal abnormalities is essential for both determining prognosis and planning therapy because meniscal injury and meniscal surgery or meniscectomy performed at the same time as ACL reconstruction have previously been shown to be associated with an increased risk for knee osteoarthritis [11, 12].

However, most previous studies correlating ACL tears and meniscal abnormalities have focused on meniscal tears. Meniscal contusions have been described as another possible entity of meniscal lesions by Cothran et al. [13]; they defined meniscal contusions as an area of increased internal signal intensity in the meniscus that was less discrete and less well defined than the signal-intensity abnormality associated with a meniscal tear.

Nevertheless, the exact cause as well as the incidence and clinical significance of this high intrameniscal signal-intensity alteration remains unclear. It is also not known how a high intrameniscal signal-intensity alteration impacts cartilage health and whether this abnormality induces early degenerative changes in the biochemical composition of cartilage, which can be measured with T1ρ and T2 mapping using advanced quantitative MRI techniques. Previous studies have shown that these techniques are highly sensitive in detecting very early cartilage abnormalities in posttraumatic knees [14, 15]. Therefore, we hypothesized that high intrameniscal signal-intensity abnormalities are a transient, reversible imaging finding without substantial clinical relevance in postsurgical ACL repair subjects.

The purpose of this longitudinal study therefore was to assess the prevalence and natural evolution of high intrameniscal signal-intensity alteration in subjects with an ACL tear, investigate compositional changes of cartilage using MRI-based T1ρ and T2 measurements after ACL tear over 24 months related to high intrameniscal signal-intensity alteration, and compare the clinical outcomes between subjects with and those without high intrameniscal signal-intensity alteration.

Materials and Methods

Subjects

For this study, 57 subjects with complete traumatic acute ACL tear (32 women and 25 men; mean age ± SD, 32.6 ± 8.3 years; mean body mass index [BMI; weight in kilograms divided by the square of height in meters], 24.5 ± 3.5) from an ongoing prospective study funded by the Arthritis Foundation were screened on MRI for meniscal contusions. All patients gave written informed consent, and the study was approved by our institutional review board. We included subjects with a clinically diagnosed acute complete ACL rupture, which had been confirmed on preoperative MRI, who underwent ACL reconstruction and standard pre- and postoperative rehabilitation. Complete preoperative MRI, clinical examinations, and arthroscopic surgery performed within 12 weeks after the ACL injury were required for inclusion in the study. Exclusion criteria were a history of osteoarthritis, inflammatory arthritis, injury, or surgery of the knee and repeated knee injuries during the follow-up period. The subjects also underwent knee MRI and clinical examinations at 12 and 24 months after surgery.

In this population, 24 subjects were identified without any meniscal (medial and lateral) tear or higher-grade meniscal abnormality at arthroscopic surgery. The preoperative MRI examinations of these 24 subjects were retrospectively reviewed. Twelve subjects showed a high intrameniscal signal-intensity alteration on baseline MRI, according to the imaging criteria described below (case cohort). The remaining 12 subjects without any meniscal abnormalities on baseline MRI were used as control subjects for the comparison of clinical and compositional cartilage analyses. MRI examinations of the contralateral knees of these 24 subjects before surgery and 12 and 24 months after surgery were analyzed.

We also retrospectively reviewed the preoperative MR images of the 33 subjects who presented with a meniscal tear or higher-grade meniscal abnormality at arthroscopic surgery, and none of these patients showed an isolated high intrameniscal signal-intensity alteration in the medial or lateral menisci.

Arthroscopy Surgery

All 57 subjects underwent arthroscopic surgery and standard-of-care anatomic reconstruction of the ACL after ACL injury.

Imaging Protocol and Analysis

Bilateral knee MRI examinations were acquired using a 3-T MRI scanner with an 8-channel knee coil. The imaging protocol included a high-resolution 3D fast spin-echo (FSE) (Cube, GE Healthcare) sequence and T1ρ and T2 mapping.

The detailed MRI acquisition parameters (morphologic and compositional) are summarized in Table 1. Preoperative and 12-month follow-up MRI examinations were available for all subjects, whereas 24-month follow-up MRI examinations of 10 subjects in each group were analyzed because two subjects with meniscal contusion and two subjects from the control group were lost to follow-up.

Morphologic Imaging

All examinations were independently evaluated using all available MRI sequences for the presence of a high intrameniscal signal-intensity alteration by two radiologists (one board-certified musculoskeletal radiologist with 6 years of experience [reader 1] and a radiology resident with 3 years of experience [reader 2]). In cases of disagreement, consensus readings were performed with a third board-certified musculoskeletal radiologist with 23 years of experience (reader 3).

A high intrameniscal signal-intensity alteration was defined as an intrasubstance high-signal-intensity abnormality on fluid-sensitive sequences seen in more than two images or planes that did not reach the articular surface or the free margin of the meniscus, as previously described by Cothran et al. [13] (Fig. 1). Follow-up MR images (12 and 24 months) of both groups were assessed for the presence of signal-intensity alterations indicating a meniscal contusion and for the presence of meniscal tears or

TABLE 1: Morphologic and Compositional MRI Sequence Parameters

MRI Sequence	TR (ms)	TE (ms)	BW (kHz)	ETL	No. of Signals Acquired	FOV (cm)	Matrix	Slice (mm)	Gap (mm)	Additional Options or Parameters
Sagittal fat-saturated 3D FSE	1500	23	50	32	2	16	384 × 384	1		Fat saturation
Sagittal PD	5100	30	62.5	13	1–2	16	512 × 480	3.5	0	TRF
Coronal PD	5100	30	62.50	13	1	14	512 × 416	3.5	0	TRF
3D T1ρ	9.3	3.7				14	256 × 192	4	4	TSL = 0, 10, 40, 80 ms; FSL = 500 Hz
3D T2	9.3	3.7				14	256 × 192	4	4	Preparation TE = 2.9, 13.6, 24.3, 45.6 ms

Note—BW = bandwidth, ETL = echo-train length, FSE = fast spin-echo, PD = proton density–weighted, TRF = tailored radiofrequency pulse, TSL = time of spin lock, FSL = frequency of spin lock.

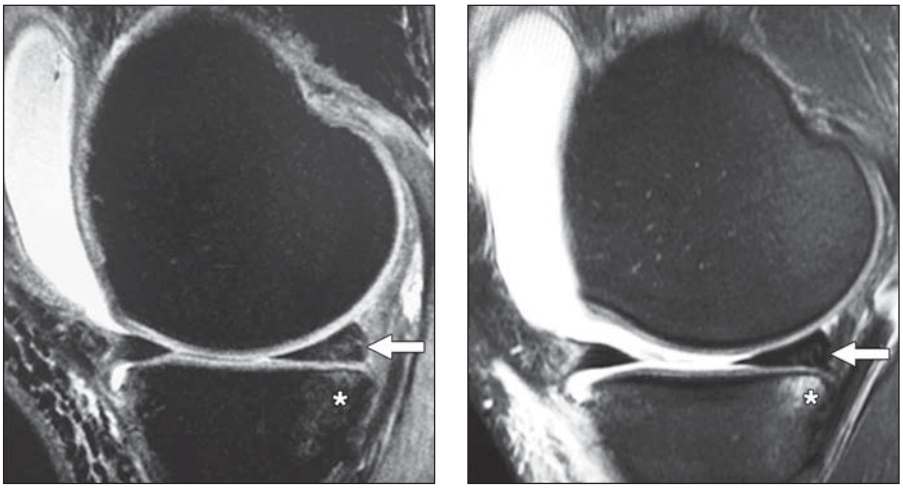

Fig. 1—24-year-old woman with anterior cruciate ligament (ACL) tear.
A and **B,** Sagittal 3D fast spin-echo (**A**) and sagittal 2D proton density–weighted fat-saturated (**B**) images obtained before ACL surgery show intrasubstance abnormal high signal intensity. Abnormal high signal intensity on fluid-sensitive sequences seen in more than two images or planes that does not reach articular surface or free margin of meniscus at posterior horn of medial meniscus (*arrow*) is characterized as high intrameniscal signal-intensity alteration. Also, we identified bone marrow edema pattern (*asterisk*) at posteromedial tibia.

any change of meniscal morphology or signal intensity. Radiologists were blinded to preoperative findings, and MRI examinations of each follow-up time point were read at least 1 month after the baseline MRI examinations were read or 1 month after the previous reading had been performed to minimize any recall bias from previous examinations.

After the meniscal abnormalities at different time points were registered, the intensities of the intrameniscal signal at the different time points were compared in subjects showing a meniscal contusion on both baseline and follow-up (12 and 24 months) MRI examinations. For this comparison, a visual side-by-side comparison of the high-resolution 3D FSE (Cube) images obtained at the different time points was performed to determine whether the signal intensity had increased, decreased, or remained stable over time (Fig. 2). If the signal intensity of the intrasubstance signal abnormality was lower on follow-up MRI than on preoperative MRI, the signal-intensity change was graded as a decrease. If the signal intensity of the intrasubstance abnormality was similar to that shown on preoperative MRI, the signal-intensity change was graded as stable. If the signal intensity of the intrasubstance abnormality was higher than that on baseline MRI, the signal-intensity change was graded as an increase. The menisci of the contralateral knee were also assessed.

MRI Relaxation Time Quantification

Cartilage was segmented semiautomatically on sagittal Cube images using a Matlab version 8.6 (MathWorks)-based algorithm developed at our institution, as previously described [16]. The following cartilage compartments were analyzed at baseline and 24 months after surgery: medial femur, lateral femur, medial tibia, and lateral tibia. The subcompartments consisting of the posterior medial tibia, posterior central medial femur, and posterior medial femur were included in these analyses.

Piecewise rigid registration was applied along both T1ρ and T2 echoes to account for nonrigid movement of the femur, tibia, and patella with respect to one another. T1ρ and T2 maps were reconstructed by fitting the echo images pixel by pixel to the following equations:

$$S(TSL) \propto S_0 \exp(-TSL / T1\rho)$$

for *T1*ρ and

$$S(TE) \propto S_0 \exp(-TE / T2)$$

for *T2* where *TSL* is the time of spin lock, *TE* is the echo time, and S is the signal intensity. Cube images were registered to T1ρ and T2 maps, and cartilage contours generated from the Cube images

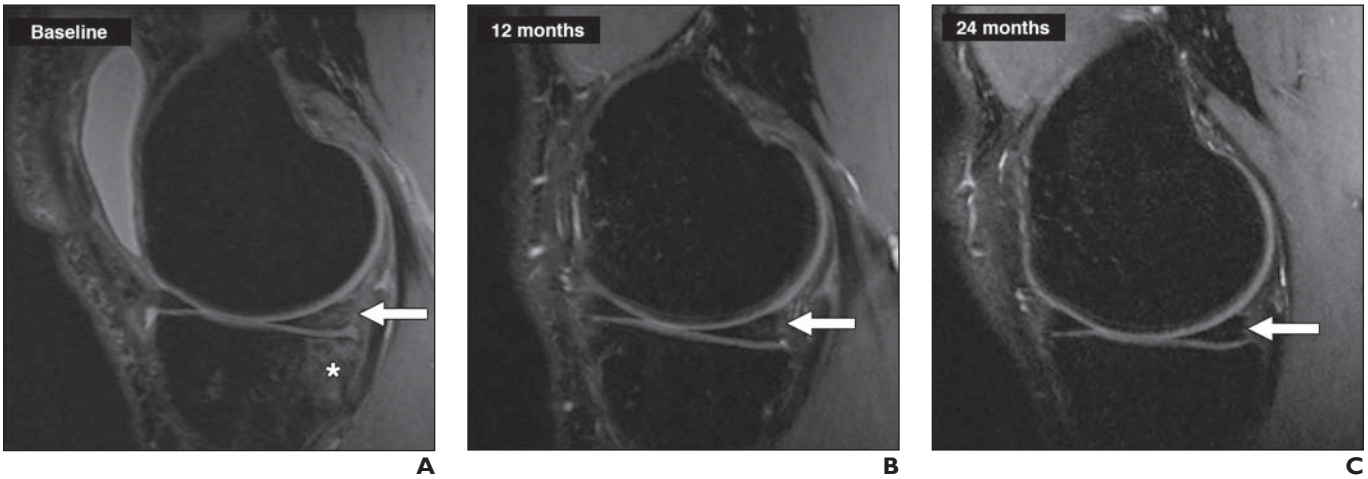

Fig. 2—Longitudinal series of sagittal 3D fast spin-echo images of 35-year-old man with anterior cruciate ligament (ACL) tear that illustrates evolution of high intrameniscal signal-intensity alteration. Complete resolution of high intrameniscal signal-intensity alteration is seen over 24 months.
A, Preoperative MR image shows high intrameniscal signal intensity (*arrow*) at posterior horn of medial meniscus. Also, we identified bone marrow edema pattern (*asterisk*) at posteromedial tibia, which completely resolved after 12 months.
B, MR image obtained at 12-month follow-up shows decreased intrameniscal signal intensity (*arrow*). Bone marrow edema pattern identified on baseline MRI has completely resolved.
C, MR image obtained at 24-month follow-up shows that signal-intensity alteration has completely resolved and morphology of menisci (*arrow*) is preserved.

were overlaid onto the T1ρ and T2 maps. The T1ρ and T2 maps at follow-up time points were also registered to baseline T1ρ and T2 maps. Changes in absolute values at baseline and 24 months and mean T1ρ and T2 measurements on a compartmental level over 24 months (Δ mean T1ρ and Δ mean T2) were computed by subtracting compartmental mean baseline T1ρ and T2 value measurements from mean values at 24-month follow-up.

Clinical Outcome

At baseline and 24 months after surgery, all five subscales of the Knee Injury and Osteoarthritis Outcome Score (KOOS) [17, 18] were assessed: pain, symptoms other than pain, activities of daily living (ADL), sport and recreation function, and knee-related quality of life (QOL). KOOS subscales range from 0 to 100, and the maximum KOOS value achievable by a healthy patient is 100. For each subscale, the differences of the absolute values at follow-up and baseline examinations were expressed as changes in KOOS (ΔKOOS).

Statistical Analysis

Statistical analysis was performed using statistics software (SPSS, version 22.0, IBM); a two-sided 0.05 level of significance was used. The Student *t* test (for numeric and approximately normally distributed data) and Pearson chi-square test (for categoric variables) were used to evaluate differences between subjects with and those without intrameniscal contusion. Differences in absolute KOOS, differences in T1ρ and T2 values at baseline and 24 months, and ΔKOOS, ΔT1ρ, and ΔT2 measurements subscale scores of subjects with and those without meniscal contusion were compared using the independent-samples *t* test.

Reproducibility

The interreader reproducibility for the evaluation of the presence of high intrameniscal signal-intensity alteration was assessed between the two radiologists in all 57 cases using the intraclass correlation coefficient (ICC). The ICC for interreader reproducibility was 0.93 (95% CI, 0.91–0.95).

For the intrareader reproducibility analysis, both readers repeated the readings of 25 randomly selected patients after at least 2 weeks. The ICC for intrareader reproducibility was 0.94 (95% CI, 0.92–0.96).

In the preoperative MRI analyses, reader 1 identified 12 subjects (12/57 subjects) with a high intrameniscal signal-intensity alteration and reader 2 identified 10 subjects (10/57 subjects). The MRI examinations of the two subjects with discrepant reads were analyzed by reader 3, and a high intrameniscal signal-intensity alteration was determined to be present on both examinations.

Results

Prevalence of Meniscal Intrasubstance Signal-Intensity Abnormalities and Baseline Imaging Characteristics

In 21% (n = 12) of the subjects with an ACL tear (n = 57), a high intrameniscal signal-intensity alteration was detected on preoperative MRI. There were no significant differences between subjects with and those without a high intrameniscal signal-intensity alteration in age (31.4 ± 2.6 vs 36.8 ± 2.4 years, respectively; $p > 0.14$) or BMI (23.6 ± 1.9 vs 24.9 ± 3.5; $p > 0.26$). None of subjects presented with any meniscal abnormalities in the contralateral knee on MRI examinations performed at baseline or at 12 or 24 months after surgery; these analyses confirmed that the intrasubstance signal-intensity abnormalities were strictly unilateral.

None of the subjects with a high intrameniscal signal-intensity alteration showed any meniscal abnormalities in the same region during arthroscopic surgery, which was performed to reconstruct the ACL. In 83% (10/12) of the subjects, the high intrameniscal signal-intensity alteration was located at the posterior horn of the medial meniscus. In the remaining 17% (2/12), it was located at the posterior horn of the lateral meniscus.

In 67% (8/12) of the subjects with a high intrameniscal signal-intensity alteration, we identified a bone marrow edema pattern at the posteromedial tibia on the baseline MRI study. This finding was present in 80% (8/10) of the subjects with a high intrameniscal signal-intensity alteration at the medial meniscus (Fig. 2). The bone marrow edema pattern at the posteromedial tibia completely resolved in 88% (7/8) of the subjects, and the bone marrow edema pattern significantly decreased in size in 13% (1/8) after 12 months and had completely resolved in all studied subjects (100%) after 24 months.

Evolution of Meniscal Intrasubstance Signal-Intensity Abnormalities Over 24 Months

None of the subjects with a high intrameniscal signal-intensity alteration on baseline MRI presented with a meniscal tear or other higher-grade meniscus abnormality on MRI at the two follow-up time points (12 and 24 months). Twelve months after surgery, the high intrameniscal signal-intensity alteration had completely resolved in 58% (7/12) of the 12 subjects. The high intrameniscal signal-intensity alteration had decreased in 33% (4/12) when compared side-by-side with the baseline MRI examination, whereas the high intrameniscal signal-intensity alteration had remained stable in 8% (1/12).

In the subgroup of 10 subjects who underwent both 12- and 24-month follow-up examinations, the high intrameniscal signal-intensity alteration had completely resolved in 50% (5/10) after 12 months and did not reoccur after 24 months. In 30% (3/10) of the subjects with complete follow-up, the signal-intensity alteration decreased after 12 months and was completely gone after 24 months. In 20% (n = 2) of the subjects, the intrasubstance signal intensity was still slightly abnormal but had decreased when compared side-by-side with the previous examinations. Therefore, at the 24-month follow-up, the high intrameniscal signal-intensity alteration had completely resolved in 80% (8/10) of subjects who had undergone follow-up MRI at both time points and had clinical data available. In the remaining two subjects in whom the high intrameniscal signal-intensity alteration had not completely resolved, the signal intensity had at least decreased when compared with the findings on baseline MRI.

Clinical Outcome

Absolute KOOS values at baseline and 24 months after surgery and ΔKOOS for pain, symptoms, ADL, sport and recreation function, and knee-related QOL did not differ significantly ($p > 0.05$) between subjects with and those without a high intrameniscal signal-intensity alteration. The absolute KOOS scores and ΔKOOS scores of subjects with and those without meniscal contusion are summarized in Table 2.

Compositional Analyses of Cartilage Using T1ρ and T2 Values at Baseline and 24 Months

Interestingly, T1ρ and T2 values at the medial tibia tended to be higher on baseline MRI in the subjects with high intrameniscal signal-intensity alteration than in the control subjects; however, on 24-month follow-up MRI, the T1ρ and T2 values at the medial tibia had increased more in the control subjects than in the case subjects. Unfortunately, none of these differences were statistically significant ($p > 0.05$).

Overall absolute cartilage T1ρ and T2 values at baseline and 24 months did not significantly ($p > 0.05$) differ between subjects with and those without high intrameniscal signal-intensity alteration. Moreover, ΔT1ρ and ΔT2 over 24 months did not show significant differences ($p > 0.05$) between the two groups. The absolute T1ρ and T2 values

TABLE 2: Summary of Subscale Scores From Knee Injury and Osteoarthritis Outcome Score (KOOS) at Baseline and 2 Years After Anterior Cruciate Ligament (ACL) Surgery for Patients With Meniscal Contusion and Control Subjects

KOOS Subscale	Absolute KOOS at Baseline			Absolute KOOS 2 Years After ACL Surgery			ΔKOOS (Baseline – 2 Years After ACL Surgery)		
	Patients With Meniscal Contusion	Control Subjects	*p*	Patients With Meniscal Contusion	Control Subjects	*p*	Patients With Meniscal Contusion	Control Subjects	*p*
Pain	75.0 ± 17.1	73.6 ± 17.3	0.84	83.0 ± 11.4	87.0 ± 7.5	0.46	5.2 ± 22.3	14.3 ± 11.7	0.38
Symptoms other than pain	65.5 ± 24.9	67.85 ± 21.75	0.81	73.8 ± 10.4	79.7 ± 12.3	0.33	8.9 ± 23.3	17.2 ± 19.4	0.49
Activities of daily living	81.2 ± 16.6	83.0 ± 16.1	0.79	94.2 ± 9.4	97.7 ± 2.5	0.39	10.4 ± 14.0	16.6 ± 11.4	0.39
Sport and recreation function	55.0 ± 31.3	45.8 ± 26.7	0.45	72.8 ± 12.5	81.6 ± 12.1	0.19	18.7 ± 36.1	34.1 ± 38.13	0.45
Knee-related QOL	56.8 ± 29.7	43.2 ± 26.9	0.26	61.8 ± 8.5	66.6 ± 14.0	0.41	15.5 ± 31.9	26.0 ± 35.4	0.21

Note—Data are expressed as mean ± SD. The clinical outcomes based on KOOS were not significantly inferior for patients with meniscal contusion than for control subjects. QOL = quality of life.

TABLE 3: Summary of T1ρ Values at Baseline and 2 Years After Anterior Cruciate Ligament (ACL) Surgery and Change in T1ρ Values (ΔT1ρ) for Subjects With Meniscal Contusion and Control Subjects

Cartilage	T1ρ Values at Baseline			T1ρ Values 2 Years After ACL Surgery			ΔT1ρ (Baseline – 2 Years After ACL Surgery)		
	Patients With Meniscal Contusion	Control Subjects	*p*	Patients With Meniscal Contusion	Control Subjects	*p*	Patients With Meniscal Contusion	Control Subjects	*p*
Compartments									
Lateral femur	39.7 ± 1.6	41.4 ± 2.7	0.63	41.3 ± 2.2	41.5 ± 1.9	0.81	1.7 ± 2.5	1.6 ± 2.7	0.94
Lateral tibia	35.3 ± 3.0	35.8 ± 2.5	0.66	35.0 ± 3.6	34.6 ± 2.1	0.76	–0.5 ± 3.5	–1.7 ± 1.3	0.16
Medial tibia	34.7 ± 3.5	34.0 ± 3.3	0.64	35.0 ± 2.1	36.7 ± 3.1	0.18	0.05 ± 2.4	0.24 ± 1.2	0.81
Medial femur	38.9 ± 3.1	38.9 ± 2.5	0.99	42.3 ± 1.3	42.4 ± 2.3	0.96	2.6 ± 2.3	2.9 ± 2.7	0.79
Subcompartments									
Posterior femur	36.2 ± 2.8	36.9 ± 2.8	0.62	39.6 ± 2.5	39.0 ± 3.0	0.62	2.9 ± 2.3	2.0 ± 4.1	0.57
Posterior central medial femur	40.8 ± 4.1	40.5 ± 3.0	0.82	43.2 ± 3.4	43.9 ± 2.2	0.61	1.8 ± 3.5	2.0 ± 3.1	0.88
Posterior medial tibia	36.5 ± 3.8	34.9 ± 3.6	0.31	36.6 ± 1.6	38.2 ± 2.9	0.16	–0.5 ± 2.7	0.9 ± 1.3	0.13

Note—Data are expressed as mean ± SD. The cartilage quality based on T1ρ values and ΔT1ρ was not significantly inferior in subjects with meniscal contusion compared with control subjects.

TABLE 4: Summary of T2 Values at Baseline and 2 Years After Anterior Cruciate Ligament (ACL) Surgery and Change in T2 (ΔT2) for Subjects With Meniscal Contusion and Control Subjects

Cartilage	T2 Values at Baseline			T2 Values 2 Years After ACL Surgery			ΔT2 (Baseline – 2 Years After ACL Surgery)		
	Patients With Meniscal Contusion	Control Subjects	*p*	Patients With Meniscal Contusion	Control Subjects	*p*	Patients With Meniscal Contusion	Control Subjects	*p*
Compartments									
Lateral femur	30.1 ± 1.4	30.5 ± 2.3	0.57	31.8 ± 1.8	33.1 ± 1.9	0.13	1.4 ± 1.8	1.6 ± 1.4	0.85
Lateral tibia	25.9 ± 2.9	26.1 ± 1.8	0.82	26.2 ± 3.2	26.4 ± 1.4	0.83	0.6 ± 2.8	0.05 ± 1.6	0.57
Medial tibia	26.5 ± 2.7	25.7 ± 2.1	0.41	27.4 ± 2.0	27.4 ± 2.9	0.99	0.6 ± 1.7	1.5 ± 3.0	0.39
Medial femur	30.2 ± 2.6	30.4 ± 1.8	0.81	33.3 ± 1.7	33.8 ± 1.7	0.54	2.3 ± 0.4	2.3 ± 1.7	0.93
Subcompartments									
Posterior femur	27.8 ± 2.8	28.3 ± 2.8	0.66	30.6 ± 2.2	31.6 ± 3.0	0.44	2.2 ± 2.0	1.7 ± 1.5	0.54
Posterior central medial femur	32.1 ± 3.1	32.2 ± 2.4	0.92	34.6 ± 2.9	34.8 ± 2.3	0.83	2.1 ± 2.3	2.4 ± 1.8	0.76
Posterior medial tibia	27.8 ± 2.7	26.7 ± 2.5	0.31	28.6 ± 1.9	29.2 ± 2.7	0.58	0.3 ± 1.9	1.8 ± 2.8	0.21

Note—Data are expressed as mean ± SD. The cartilage quality based on T2 values and ΔT2 was not inferior in subjects with meniscal contusion compared with control subjects.

at baseline and 24 months and ΔT1ρ and T2 values of subjects with and those without traumatic high intrameniscal signal-intensity alteration at the medial femur, medial tibia, lateral femur, lateral tibia, posterior medial tibia, posterior femur, and posterior central medial femur are summarized in Tables 3 and 4.

Discussion

The results of our study showed that hyperintense signal abnormalities within the meniscus indicating posttraumatic meniscal contusions were found in approximately 20% of patients after acute ACL tear. The meniscal contusions were frequently located in the posterior horn of the medial meniscus and were highly associated with a bone marrow edema pattern at the posteromedial tibia. This high intrasubstance signal-intensity abnormality resolved in most of the studied subjects over 2 years, and none of the subjects developed a meniscal tear or other higher-grade meniscus abnormality over that time period. When comparing the clinical outcome and the cartilage composition of subjects with high meniscal intrasubstance signal intensity and the control group, consisting of subjects with an ACL tear and no meniscal abnormality, there were no statistical differences between the two groups. These results suggest that meniscal contusion had no significant impact on the clinical outcome or accelerated progression of knee cartilage degeneration over 2 years.

The term "meniscal contusion" was adopted in 2001 [13]; it was postulated that this abnormal meniscal signal intensity is a type of injury within the meniscus, probably because the meniscus has been exposed to a high-impact compression injury during trauma. We found a high intrameniscal signal-intensity alteration in 21% of the subjects with an ACL tear, which shows that this MRI finding is one that frequently accompanies this type of injury. Mustonen et al. [19] studied the association between meniscal lesions and tibia plateau fractures and reported that meniscal contusions were detected in 28% of the analyzed subjects. We hypothesize that the impact of the trauma was lower in our subjects, which may explain the slightly greater number of cases of high intrameniscal signal-intensity alteration in their study in comparison with ours. In our study, none of the subjects with meniscal contusion detected on MRI showed any meniscal abnormalities in the same region during arthroscopic surgery performed to reconstruct the ACL. This finding suggests that this meniscal abnormality cannot be detected during surgery and may be detected only using MRI. Most high intrameniscal signal-intensity alterations (83%) were found in the posterior horn of the medial meniscus, which is in accordance with the previous results reported in the literature [13]. In most subjects with a high intrameniscal signal-intensity alteration at the medial meniscus (80%, $n = 8$), we identified a bone marrow edema pattern at the posteromedial tibia, subjacent to the posterior horn of the medial meniscus, that was probably related to the trauma mechanism. As discussed by Cothran et al. [13], this injury might be caused by a contrecoup injury [20] in which an impact occurs at the posteromedial aspect of the tibial plateau and, analogously, the posterior horn of the medial meniscus after an ACL tear.

The results from the current study not only suggest that most of these high intrameniscal signal-intensity alterations resolve, but also show that none of the subjects with meniscal contusion had developed a meniscal tear at 12- and 24-month follow-ups; these results are consistent with a benign evolution of a high intrameniscal signal-intensity alteration detected on MRI after an ACL tear. The longitudinal analyses showed that this abnormal signal intensity detected within a meniscal contusion is a type of transient injury and tends to resolve over time.

One possible differential diagnosis for meniscal contusions is the intrasubstance meniscal signal-intensity abnormality, which is associated with intrasubstance degeneration and has been shown to represent an increase in mucoid intercellular ground substance in studies correlating MRI and histologic findings [21]. Intrasubstance degeneration is commonly found in older subjects but is rarely found in younger populations, such as the one in our analysis. Also, intrasubstance signal-intensity abnormalities of the menisci are not related to trauma, which differentiates this finding from a meniscal contusion, which is a common finding in young posttraumatic subjects. A recent study showed that most cases of intrasubstance degeneration are unlikely to regress and are prone to progress to a degenerative meniscal tear [22]. This outcome is different from what we found in the subjects with a traumatic high intrameniscal signal-intensity alteration: In most cases, the intrasubstance signal-intensity alteration either completely resolved or at least decreased, and none of the subjects developed a meniscal tear over 2 years. MRI analyses of the contralateral knee of the subjects with meniscal contusion showed that none presented with a high meniscal intrasubstance signal-intensity alteration or any other abnormality of the contralateral meniscus. These results show that this finding (presumably traumatic high intrameniscal signal-intensity alteration) is most likely related to the previous trauma mechanism and is not related to degenerative changes.

Standard MRI sequences, which include fat-saturated T2-weighted, proton density–weighted FSE, and T1-weighted spoiled gradient-echo sequences, have been reported to be useful in detecting morphologic changes associated with cartilage breakdown noninvasively [6]. These sequences, however, are limited in detecting early degenerative changes of the cartilage matrix [23, 24]. Recent developments in MRI techniques, such as T1ρ and T2 mapping, can be used to quantify the earliest biochemical changes in the cartilage matrix that occur even before cartilage degeneration can be detected morphologically [25]. Several previous studies have evaluated cartilage T1ρ and T2 values to detect compositional cartilage matrix changes after ACL injury and reconstruction [14, 15, 26]. Therefore, these techniques are adequate to analyze initial cartilage damage in the posttraumatic knee. However, by using these techniques (i.e., T1ρ and T2 mapping), we showed that subjects with an ACL tear and a high intrameniscal signal-intensity alteration do not show significantly accelerated progression of cartilage degeneration over 24 months when compared with ACL-injured patients without any meniscal abnormality. These results further support our hypothesis that a high intrameniscal signal-intensity alteration is a transient, reversible imaging finding without substantial relevance. The clinical outcome (mean ΔKOOS score) and the quantitative cartilage analyses (ΔT1ρ and ΔT2 values) did not differ significantly between subjects with a traumatic high intrameniscal signal-intensity alteration and subjects without a meniscal abnormality, again suggesting a benign evolution with regard to not only cartilage composition but also clinical context.

It remains unclear what the abnormal signal intensity in a meniscal contusion is caused by, and the periphery of the meniscus is known to have a vascular zone [27]. Therefore, as previous described [13], we believe that this type of lesion is related to blood products from a traumatic injury to the

vascular zone of the meniscus and results in increased signal intensity on MRI after acute or subacute injury. In most cases, the blood products can be reabsorbed, showing the transient characteristics of this lesion, without any major damage in the meniscal structure or function, as shown by the longitudinal MRI analyses.

There is paucity of studies in the literature and a lack of diagnostic proof about this type of traumatic meniscal alteration. This lack of information is probably because these meniscal abnormalities cannot be identified at knee arthroscopy and there is no specific treatment. Moreover, this meniscal abnormality does not seem to be a cause of major meniscal dysfunction and its clinical evolution suggests a benign course.

This study has several limitations. First, it was based on a relatively small cohort of 57 subjects overall. In these subjects, intrasubstance signal-intensity abnormalities were found in 12 subjects (21%), which probably represents the normal prevalence of this finding. However, together with the control cases, we were able to follow 24 subjects over 12 months and 20 subjects over 24 months in this first longitudinal assessment of this entity. In terms of clinical outcome, the group with meniscal contusions showed mildly reduced improvement with KOOS, but this difference was not statistically significant. The lack of significance may be an issue of a lack of power with the small sample size, so this difference needs to be tested in a larger cohort. Further subgroup analyses such as a comparison of the clinical performance in subjects with versus those without decreasing signal-intensity abnormalities were impossible because of even smaller sample sizes. In the MRI analyses, the intrasubstance signal-intensity abnormality was graded subjectively; in addition, during the visual side-by-side comparisons of the meniscal signal intensities, the baseline study was known to the readers, introducing the possibility of bias. Nevertheless, this study is the first longitudinal study to date to assess the evolution of high intrameniscal signal-intensity alterations in subjects with an ACL tear and is the only study to analyze the clinical outcome and cartilage quality longitudinally over 2 years.

In summary, we have found that a high intrameniscal signal-intensity alteration detected on MRI is a common finding in subjects with an ACL tear and that it is frequently located in the posterior horn of the medial meniscus. Because this meniscal abnormality is likely to be reversible over 24 months and does not have a significant impact on clinical outcome or cartilage quality, it should be considered to be a benign transient finding on postsurgical MRI of subjects who have undergone ACL repair.

References

1. Marrale J, Morrissey MC, Haddad FS. A literature review of autograft and allograft anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 2007; 15:690–704
2. Griffin LY, Agel J, Albohm MJ, et al. Noncontact anterior cruciate ligament injuries: risk factors and prevention strategies. *J Am Acad Orthop Surg* 2000; 8:141–150
3. Bellabarba C, Bush-Joseph CA, Bach Br Jr. Patterns of meniscal injury in the anterior cruciate-deficient knee: a review of the literature. *Am J Orthop* 1997; 26:18–23
4. Warren RF, Levy IM. Meniscal lesions associated with anterior cruciate ligament injury. *Clin Orthop Relat Res* 1983; 172:32–37
5. Cerabona F, Sherman MF, Bonamo JR, Sklar J. Patterns of meniscal injury with acute anterior cruciate ligament tears. *Am J Sports Med* 1988; 16:603–609
6. Eckstein F, Burstein D, Link TM. Quantitative MRI of cartilage and bone: degenerative changes in osteoarthritis. *NMR Biomed* 2006; 19:822–854
7. Peterfy CG, Guermazi A, Zaim S, et al. Whole-organ magnetic resonance imaging score (WORMS) of the knee in osteoarthritis. *Osteoarthritis Cartilage* 2004; 12:177–190
8. Lee JK, Yao L, Phelps CT, Wirth CR, Czajka J, Lozman J. Anterior cruciate ligament tears: MR imaging compared with arthroscopy and clinical tests. *Radiology* 1988; 166:861–864
9. McCauley TR, Moses M, Kier R, Lynch JK, Barton JW, Jokl P. MR diagnosis of tears of anterior cruciate ligament of the knee: importance of ancillary findings. *AJR* 1994; 162:115–119
10. Oei EH, Nikken JJ, Verstijnen AC, Ginai AZ, Myriam Hunink MG. MR imaging of the menisci and cruciate ligaments: a systematic review. *Radiology* 2003; 226:837–848
11. van der Hart CP, van den Bekerom MP, Patt TW. The occurrence of osteoarthritis at a minimum of ten years after reconstruction of the anterior cruciate ligament. *J Orthop Surg Res* 2008; 3:24
12. Neuman P, Englund M, Kostogiannis I, Friden T, Roos H, Dahlberg LE. Prevalence of tibiofemoral osteoarthritis 15 years after nonoperative treatment of anterior cruciate ligament injury: a prospective cohort study. *Am J Sports Med* 2008; 36:1717–1725
13. Cothran RL Jr, Major NM, Helms CA, Higgins LD. MR imaging of meniscal contusion in the knee. *AJR* 2001; 177:1189–1192
14. Bolbos RI, Ma CB, Link TM, Majumdar S, Li X. In vivo T1rho quantitative assessment of knee cartilage after anterior cruciate ligament injury using 3 Tesla magnetic resonance imaging. *Invest Radiol* 2008; 43:782–788
15. Li X, Kuo D, Theologis A, et al. Cartilage in anterior cruciate ligament-reconstructed knees: MR imaging T1{rho} and T2—initial experience with 1-year follow-up. *Radiology* 2011; 258:505–514
16. Carballido-Gamio J, Bauer JS, Stahl R, et al. Inter-subject comparison of MRI knee cartilage thickness. *Med Image Anal* 2008; 12:120–135
17. Marx RG, Stump TJ, Jones EC, Wickiewicz TL, Warren RF. Development and evaluation of an activity rating scale for disorders of the knee. *Am J Sports Med* 2001; 29:213–218
18. Roos EM, Lohmander LS. The knee injury and osteoarthritis outcome score (KOOS): from joint injury to osteoarthritis. *Health Qual Life Outcomes* 2003; 1:64
19. Mustonen AO, Koivikko MP, Lindahl J, Koskinen SK. MRI of acute meniscal injury associated with tibial plateau fractures: prevalence, type, and location. *AJR* 2008; 191:1002–1009
20. Kaplan PA, Gehl RH, Dussault RG, Anderson MW, Diduch DR. Bone contusions of the posterior lip of the medial tibial plateau (contrecoup injury) and associated internal derangements of the knee at MR imaging. *Radiology* 1999; 211:747–753
21. Hajek PC, Gylys-Morin VM, Baker LL, Sartoris DJ, Haghighi P, Resnick D. The high signal intensity meniscus of the knee: magnetic resonance evaluation and in vivo correlation. *Invest Radiol* 1987; 22:883–890
22. Kumm J, Roemer FW, Guermazi A, Turkiewicz A, Englund M. Natural history of intrameniscal signal intensity on knee MR images: six years of data from the Osteoarthritis Initiative. *Radiology* 2016; 278:164–171
23. Li X, Benjamin Ma C, Link TM, et al. In vivo T(1rho) and T(2) mapping of articular cartilage in osteoarthritis of the knee using 3 T MRI. *Osteoarthritis Cartilage* 2007; 15:789–797
24. Dijkgraaf LC, de Bont LG, Boering G, Liem RS. The structure, biochemistry, and metabolism of osteoarthritic cartilage: a review of the literature. *J Oral Maxillofac Surg* 1995; 53:1182–1192
25. Duvvuri U, Reddy R, Patel SD, Kaufman JH, Kneeland JB, Leigh JS. T1rho-relaxation in articular cartilage: effects of enzymatic degradation. *Magn Reson Med* 1997; 38:863–867
26. Potter HG, Jain SK, Ma Y, Black BR, Fung S, Lyman S. Cartilage injury after acute, isolated anterior cruciate ligament tear: immediate and longitudinal effect with clinical/MRI follow-up. *Am J Sports Med* 2012; 40:276–285
27. Arnoczky SP, Warren RF. Microvasculature of the human meniscus. *Am J Sports Med* 1982; 10:90–95