

The Association of Meniscal Pathologic Changes With Cartilage Loss in Symptomatic Knee Osteoarthritis

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Objective. To explore the role of meniscal tears and meniscal malposition as risk factors for subsequent cartilage loss in subjects with symptomatic osteoarthritis (OA).

Methods. Study subjects were patients with symptomatic knee OA from the Boston Osteoarthritis of the Knee Study. Baseline assessments included knee magnetic resonance imaging (MRI) with followup MRI at 15 and 30 months. Cartilage and meniscal damage were scored on MRI in the medial and lateral tibiofemoral joints using the semiquantitative whole-organ magnetic resonance imaging score. Tibiofemoral cartilage was scored on MR images of all 5 plates of each tibiofemoral joint, and the meniscal position was measured using eFilm Workstation software. A proportional odds logistic regression model with generalized estimating equations was used to assess the effect of each predictor (meniscal position factor and meniscal damage as dichotomous predictors in each model) on cartilage loss in each of the 5 plates within a compartment. Models were adjusted for age, body mass index (BMI), tibial width, and sex.

Results. We assessed 257 subjects whose mean \pm SD age was 66.6 ± 9.2 years and BMI was 31.5 ± 5.7 kg/m²; 42% of subjects were female, and 77% of knees had a Kellgren/Lawrence radiographic severity grade ≥ 2 . In the medial tibiofemoral joint, each measure of meniscal malposition was associated with an increased

risk of cartilage loss. There was also a strong association between meniscal damage and cartilage loss. Since meniscal coverage and meniscal height diminished with subluxation, less coverage and reduced height also increased the risk of cartilage loss.

Conclusion. This study highlights the importance of an intact and functioning meniscus in patients with symptomatic knee OA, since the findings demonstrate that loss of this function has important consequences for cartilage loss.

Cartilage loss in knee osteoarthritis (OA) is a multifactorial process that is influenced by systemic risk factors such as age, sex, and obesity and by local mechanical factors such as alignment and injury. One of the important local mechanical factors is the integrity and function of the meniscus. The meniscus has many functions in the knee, including load bearing, shock absorption, stability enhancement, and lubrication (1,2). Knee OA after meniscectomy is traditionally considered a result of the joint injury that leads to the meniscectomy in the first instance, and the increased contact stress in the cartilage due to the loss of meniscal tissue (3–8). Meniscectomy is often accompanied by the onset of OA because of the high focal stresses imposed on articular cartilage and subchondral bone subsequent to excision of the meniscus. Studies of meniscectomy affirm the importance of loss of meniscal function as a risk factor for subsequent knee OA (9).

Although meniscectomy appears to be an important risk factor for OA, we know little about the effect of meniscal tears and meniscal extrusion or subluxation on cartilage loss in knees with preexisting OA. Results from a cross-sectional study demonstrated that meniscal subluxation is common in knees with OA and correlated with the severity of joint space narrowing on plain radiographs (10). In another cross-sectional study, joint space narrowing on conventional radiographs was often found to be secondary to meniscal extrusion rather than

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thinning of articular cartilage (11). Studying the effects of unique elements of meniscal disease on cartilage loss in the knee is difficult because the features of meniscal disease are highly intercorrelated. A meniscus subluxes generally after it is torn. An increasing number or length of tears leads to more subluxation. Multiple tears and subluxation may ultimately lead to maceration. Whereas we can assess the position of meniscal substance in the knee on magnetic resonance imaging (MRI), we cannot distinguish these effects from the loss of meniscal integrity that led to it.

To date, little is known about the effect of meniscal integrity and position on the risk of disease progression in patients with OA. The purpose of this study was to explore the role of meniscal tears and meniscal malposition as risk factors for cartilage loss in patients with symptomatic knee OA.

PATIENTS AND METHODS

Study population. Patients were recruited to participate in a natural history study of symptomatic knee OA, the Boston Osteoarthritis of the Knee Study. The recruitment for this study has been described in detail elsewhere (12). Briefly, patients were recruited from 2 prospective studies on quality of life among veterans (1 involving a male cohort and the other, a female cohort). Patients were also recruited from clinics at Boston Medical Center in Massachusetts, and from advertisements in local newspapers. Potential participants were asked 2 questions: "Do you have pain, aching, or stiffness in one or both knees on most days?", and "Has a doctor ever told you that you have knee arthritis?". For patients who answered yes to both questions, we conducted a followup interview in which we asked about other types of arthritis that could cause knee symptoms. If no other forms of arthritis were identified, then the individual was eligible for recruitment.

For each patient, a series of knee radiographs (posteroanterior, lateral, and skyline) was obtained to determine whether radiographic OA was present. If patients had a definite osteophyte on any view in the symptomatic knee, they were eligible for the study. Because they had frequent knee symptoms and radiographic OA, all patients met the American College of Rheumatology criteria for symptomatic knee OA (13).

The study included a baseline examination and followup examinations at 15 and 30 months. At baseline, patients who did not have contraindications to MRI underwent MRI of the more symptomatic knee. MRI of the same knee was also performed at the 15- and 30-month followup visits. At the baseline assessment, patients were also weighed, with their shoes removed, on a balance-beam scale, and height was assessed. The institutional review boards of Boston University Medical Center and the Veterans Administration (VA) Boston Health Care System approved the examinations.

MRI measurements. All studies were performed with a Signa 1.5T MRI system (General Electric, Milwaukee, WI) using a phased-array knee coil. A positioning device was used to ensure uniformity among patients, with the patient reclining in the supine position, the knee fully extended and immobi-

lized in the knee coil, and the foot perpendicular to the table. Coronal, sagittal, and axial images were obtained. Fat-suppressed fast spin echo (FSE) proton density and T2-weighted images (repetition time 2,200 msec, echo time 20/80 msec) with a slice thickness of 3 mm, a 1-mm interslice gap, 1 excitation, a field of view of 11–12 cm, and a matrix of 256 × 128 pixels were obtained.

Meniscal damage and cartilage morphologic features were assessed using a semiquantitative, multifeature scoring method, the whole-organ magnetic resonance imaging score (WORMS). This method of whole-knee evaluation is applicable for use in conjunction with conventional MRI techniques (14).

Tibiofemoral cartilage on MRI was scored on all 5 plates (central and posterior femur, and anterior, central, and posterior tibia) in both the medial and lateral tibiofemoral joints. The anterior femur was not included in this analysis because this is part of the patellofemoral joint. Scoring was carried out by grading the fat-suppressed T2-weighted FSE images on a 7-point scale, as follows: 0 = normal thickness and signal; 1 = normal thickness but increased signal on T2-weighted images; 2 = partial-thickness focal defect <1 cm in greatest width; 3 = multiple areas of partial-thickness (grade 2) defects intermixed with areas of normal thickness, or a grade 2 defect wider than 1 cm but <75% of the region; 4 = diffuse ($\geq 75\%$ of the region) partial-thickness loss; 5 = multiple areas of full-thickness loss wider than 1 cm but <75% of the region; 6 = diffuse ($\geq 75\%$ of the region) full-thickness loss.

In the WORMS system, grade 1 does not represent a morphologic abnormality, but rather indicates a change in signal in cartilage having otherwise-normal morphologic features. Grades 2 and 3 represent similar types of abnormality of the cartilage, that is, focal defects without overall thinning. Scores of 1 and 2 were exceedingly unusual. Therefore, to create a consistent and logical scale for evaluation of cartilage morphologic change, we collapsed the WORMS values to a new scale of 0–4, where the original WORMS values of 0 and 1 were collapsed to 0, the original scores of 2 and 3 were collapsed to 1, and the original scores of 4, 5, and 6 were considered to be 2, 3, and 4, respectively.

Films were read paired and unblinded to sequence by 2 readers (AG and MG, both of whom are musculoskeletal radiologists) using MRI sequence data from the sagittal and coronal planes. The intraobserver agreement (kappa value) for reading of cartilage morphologic changes ranged from 0.72 to 0.97, and the interobserver agreement was 0.62. We defined a lesion as occurring in either the medial or the lateral compartment if it was present in the femur or the tibia of that compartment. Although we conducted analyses using the collapsed WORMS cartilage scale of 0–4, analyses using the original scale of 0–6 yielded the same results.

The anterior horn, body segment, and posterior horn of each of the medial and lateral menisci were graded from 0 to 4 based on both the sagittal and the coronal images, with 0 = intact menisci, 1 = minor radial tear or parrot-beak tear, 2 = nondisplaced tear or prior surgical repair, 3 = displaced tear or partial resection, and 4 = complete maceration/destruction or complete resection. A tear was defined when there was a signal change within the meniscus that extended to the surface of the meniscus. This global scoring of meniscal integrity incorporates all elements of meniscal disease and also requires incorporation of factors of meniscal position. We therefore considered this to be a global meniscal score, recog-

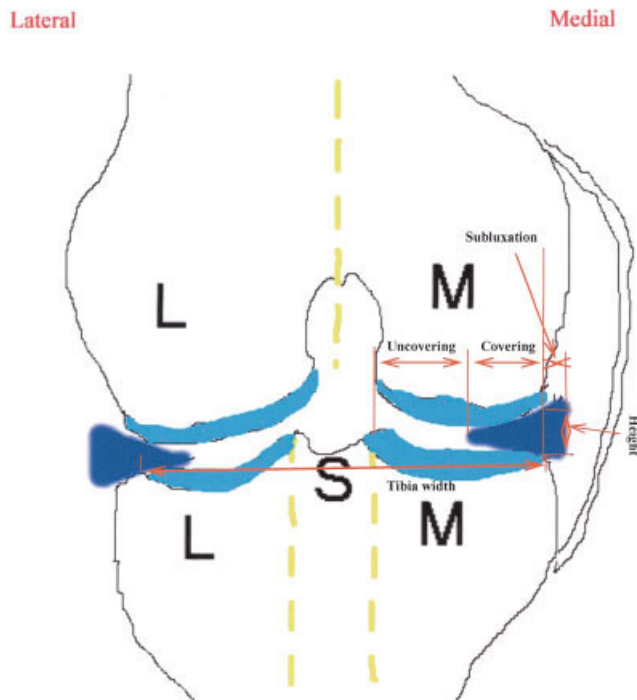


Figure 1. Meniscal position measurements obtained on coronal magnetic resonance imaging of the affected knee.

nizing that abnormal meniscal position represents only one aspect of the total score. For the purposes of this analysis, we refer to this as the meniscal damage score. The interobserver agreement (interclass correlation coefficient [ICC]) for reading of meniscal damage ranged from 0.95 to 0.97. These films were read paired and blinded to sequence. To minimize bias, the reading of meniscal damage was performed independent of the cartilage morphology reading.

Using the coronal MRIs and eFilm Workstation software, we determined the following meniscal position measures, to the nearest millimeter, in both the medial and the lateral compartments: subluxation, meniscal height, and meniscal covering and uncovering of the tibial plateau (see Figure 1). On the coronal sequence, the image in which the medial tibial spine volume was maximal was selected for all readings. On this image, the reference point for measuring the extent of subluxation, tibial width, and amount of coverage was the edge of the tibial plateau without osteophytes (Figure 2). The proportion of coverage was calculated as meniscal covering divided by the sum of meniscal covering and meniscal uncovering. In the sagittal plane, anterior subluxation of the medial and lateral menisci was assessed (Figure 3). A meniscus that was completely macerated or destroyed (as defined above) did not generate a measure of subluxation. Thus, when the WOMBS value assigned to the meniscus was equal to a score of 4 (maceration and often absent meniscus), we did not include these knees in analyses of subluxation. Interobserver reliability (ICC values) for reading the measures of meniscal position ranged from 0.86 to 0.93. For all of these analyses, the predictors (meniscal damage and meniscal position) were read at baseline, and the outcome (cartilage morphologic changes) was read at 0, 15, and 30 months.

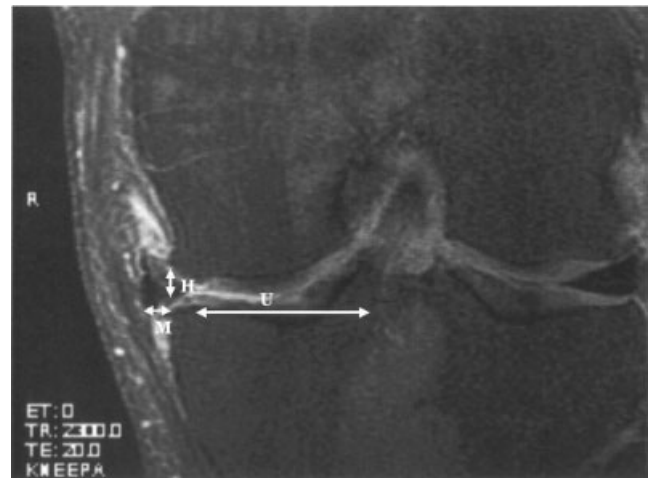


Figure 2. Representative example of meniscal position measurement in the coronal plane, demonstrating medial meniscal subluxation (M) in the coronal plane (measured from the most subluxed edge of the meniscus to the edge of the medial tibial plateau), uncovering (U) of the tibial plateau (from the meniscus to the medial tibial spine), and reduced height (H) of the meniscus at the edge of the tibial plateau, compared with maximal height of the meniscus.

Statistical analysis. Cartilage loss (the primary outcome variable) was defined as an increase of at least 1 in the cartilage morphologic score (modified WOMBS) over time at any plate, using the compressed scale of 0–4. This primary ordinal outcome (based on the change in score by plate, with a possible range for change of 0–4) was defined in each plate as the change in cartilage score from baseline to followup. Of all the plates assessed, 10.5% in the medial compartment had the maximal cartilage score at baseline, and 4.3% in the lateral compartment had the maximal cartilage score at baseline; the plates with a maximal cartilage score at baseline were excluded

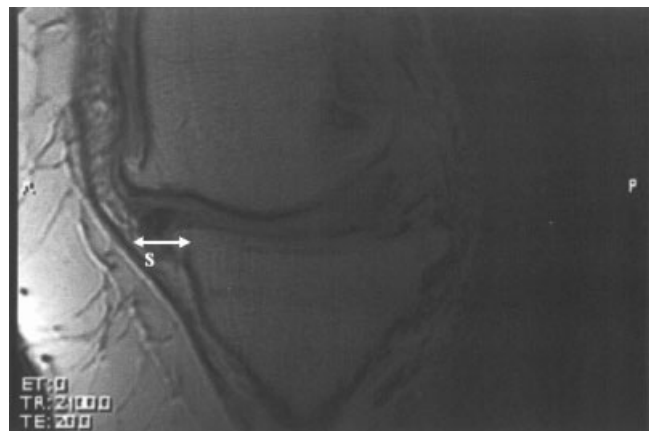


Figure 3. Representative example of meniscal position measurement in the sagittal plane, demonstrating anterior medial meniscal subluxation (S) in the sagittal plane (measured from the anterior tibial plateau, excluding osteophytes, to the most subluxed edge of the meniscus anteriorly).

from the analysis, but other plates in the same subject were used in the analysis.

We used a proportional odds logistic regression model with generalized estimating equations (GEE) to assess the effect of each predictor (quartiles of the meniscal position factor, and meniscal damage from the baseline assessment, as predictors in each model) on cartilage loss in each of the 5 plates within a compartment (change in cartilage morphologic score from baseline to followup). Models were adjusted for age, sex, body mass index (BMI), and tibial width. Cartilage plates with a maximal score at baseline were not included in the analysis. Statistical analyses were performed using SAS software (release 8.2; SAS Institute, Cary, NC). We also tested the correlation between different measures of meniscal disease and malpositioning, with Spearman's rank correlation coefficients.

RESULTS

The characteristics of the study population are presented in Table 1. We assessed 257 subjects whose mean \pm SD age was 66.6 ± 9.2 years and BMI was 31.5 ± 5.7 kg/m²; 42% were female, and 77% of knees had a Kellgren/Lawrence (K/L) radiographic severity grade ≥ 2 (those with K/L grades of < 2 had patellofemoral OA) (15). The predominance of male subjects reflects the VA population from which many of them were drawn.

At baseline, the majority (86%) of knees had cartilage morphologic abnormalities in the medial tibiofemoral compartment, and 63% of knees had morphologic abnormalities in the lateral compartment (Table 2). Although the average increase in cartilage morphologic score in the medial compartment was 0.24 on the 0–4 scale, 53% of knees showed no change in score, 34% of knees had an increase of 1 unit in at least 1 plate, and 13% of knees had an increase of > 1 unit (on the 0–4 scale) in at least 1 plate. In the lateral compartment, scores were unchanged in 78% of knees, 17% of knees had an increase of 1 unit in at least 1 plate, and 5% of knees had an increase of > 1 unit (on the 0–4 scale) in at least 1 plate.

As shown in Table 3, in the medial tibiofemoral joint, each measure of meniscal malposition was associated with an increased risk of cartilage loss. There was also a strong association of meniscal damage with cartilage loss. Since meniscal coverage and meniscal height diminished with subluxation, less coverage and reduced height of the meniscus also increased the risk of cartilage loss.

The results in the lateral tibiofemoral compartment (Table 4) were similar in terms of the direction of the effect, although the magnitude of the risk conferred by lateral meniscal damage was smaller than that con-

Table 1. Characteristics of the 257 study participants at baseline*

Age, mean \pm SD (range) years	66.6 \pm 9.2 (47–93)
Sex, % male	58.4
BMI, mean \pm SD (range) kg/m ²	31.5 \pm 5.7 (21.4–59.7)
K/L grade ≥ 2 , %	77
Previous knee injury in MRI-assessed knee, %	29
Previous knee surgery in MRI-assessed knee, %	27
Previous meniscectomy in MRI-assessed knee, %	5
Meniscal subluxation, mean \pm SD (range)	
Medial meniscus	
Subluxation, mm	4.3 \pm 2.5 (0–16)
Coverage, mm	6.3 \pm 5.5 (0–24)
Uncovering, mm	23.1 \pm 6.2 (4–37)
Proportion of coverage (range 0–1)	0.21 \pm 0.19 (0–0.86)
Meniscal height, mm	2.9 \pm 2.0 (0–7)
Anterior subluxation (sagittal), mm	5.1 \pm 3.0 (0–14)
Lateral meniscus	
Subluxation, mm	1.3 \pm 1.7 (0–9)
Coverage, mm	9.4 \pm 3.9 (0–25)
Uncovering, mm	20.4 \pm 4.6 (0–36)
Proportion of coverage (range 0–1)	0.31 \pm 0.14 (0–1)
Meniscal height, mm	5.4 \pm 2.5 (0–11)
Anterior subluxation (sagittal), mm	1.8 \pm 3.0 (0–16)
Meniscal damage	
Medial score (possible range 0–12)	3.8 \pm 3.8 (0–12)
Lateral score (possible range 0–12)	1.8 \pm 3.2 (0–12)
Mechanical alignment, mean \pm SD (range) degrees†	3.6 \pm 5.7 (–11 to 20)

* BMI = body mass index; K/L = Kellgren/Lawrence; MRI = magnetic resonance imaging.

† Positive value denotes varus alignment, while negative value denotes valgus alignment.

ferred by medial meniscal damage. The results in Tables 3 and 4 were similar after adjusting for baseline cartilage score.

All measures of meniscal malposition were correlated with one another, and all were also correlated with the global measure of meniscal disease, the damage

Table 2. Baseline cartilage scores and loss of cartilage in knees over followup, in the medial and lateral tibiofemoral (TF) compartments*

	Medial TF compartment	Lateral TF compartment
Baseline cartilage score in 5 plates in compartment	1.29 \pm 0.97	0.59 \pm 0.77
Subjects with mean baseline cartilage score > 0 , %	86	63
Increase in cartilage score from baseline to followup in 5 plates	0.24 \pm 0.38†	0.09 \pm 0.20†

* Except where indicated otherwise, values are the mean \pm SD modified (scale 0–4) whole-organ magnetic resonance imaging score for cartilage morphologic abnormalities.

† $P < 0.0001$ versus baseline, by paired t -test.

Table 3. Risk of medial tibiofemoral cartilage loss by quartile of medial meniscal position measure and damage score at baseline*

Predictor variable	Referent group (most normal meniscal position)	Second quartile	Third quartile	Fourth quartile (most abnormal meniscal position)	P for trend
Medial meniscus position factors					
Medial subluxation†	1.0	0.9 (0.4–1.9)	3.2 (1.5–6.9)	2.4 (1.1–5.0)	0.0023
<i>P</i>		0.8471	0.0032	0.0259	
Number of subjects in each quartile	48	78	51	58	
Proportion of coverage‡	1.0	1.6 (0.8–2.9)	1.7 (0.8–3.5)	2.7 (1.5–5.2)	0.0031
<i>P</i>		0.1813	0.1316	0.0018	
Number of subjects in each quartile	85	40	62	62	
Meniscal height§	1.0	1.6 (0.8–3.3)	2.1 (1.0–4.5)	3.0 (1.5–6.2)	0.0009
<i>P</i>		0.2134	0.0641	0.0023	
Number of subjects in each quartile	86	44	67	52	
Anterior subluxation¶	1.0	1.3 (0.6–2.7)	1.7 (0.8–3.6)	3.2 (1.6–6.2)	0.0006
<i>P</i>		0.5569	0.2045	0.0007	
Number of subjects in each quartile	68	55	38	70	
Medial meniscal damage (WORMS)#	1.0	3.4 (1.8–6.2)	3.9 (2.2–7.0)	6.3 (3.1–12.6)	<0.0001
<i>P</i>		<0.0001	<0.0001	<0.0001	
Number of subjects in each quartile	90	60	49	53	

* Except where indicated otherwise, values are the odds ratio (95% confidence interval) adjusted for age, body mass index, sex, and tibial width.
 † Subluxation quartiles: 0–2 mm, 3–4 mm, 5–6 mm, and ≥7 mm; subluxation measures were tested among knees in which meniscal damage scores of 4 were excluded.
 ‡ Proportion of coverage quartiles: 0.29–0.86, 0.12–0.28, and 0.03–0.11.
 § Height quartiles: 5–7 mm, 3–4 mm, 1–2 mm, and 0 mm.
 ¶ Anterior subluxation quartiles: 0–3 mm, 4–5 mm, 6–7 mm, and 8–14 mm.
 # Whole-organ magnetic resonance imaging score (WORMS) range across all segments of the meniscus (each scored 0–4) was 0–12, with a median of 2.

score (Table 5). For example, among all knees (including those with menisci absent and damage scores of 4), correlation coefficients between meniscal damage, height, and proportion of coverage in the medial compartment ranged from 0.69 to 0.87 (all *P* < 0.0001).

We attempted to distinguish the effect of meniscal

damage from that of meniscal position by identifying knees that were discordant for these measures, but we observed <10 knees in which there was discordance for meniscal damage and meniscal position measures, and there was no consistent pattern of cartilage loss or preservation in these knees. Correlations of meniscal measures were somewhat

Table 4. Risk of lateral tibiofemoral cartilage loss by quartile of lateral meniscal position measure and damage score at baseline*

Predictor variable	Referent group (most normal meniscal position)	Second quartile	Third quartile	Fourth quartile (most abnormal meniscal position)	P for trend
Lateral meniscus position factors					
Subluxation†	1.0	1.7 (0.6–5.0)	3.0 (1.3–7.0)	4.6 (2.0–10.8)	<0.0001
<i>P</i>		0.3552	0.0086	0.0004	
Number of subjects in each quartile	103	44	58	41	
Proportion of coverage‡	1.0	0.6 (0.2–1.7)	1.4 (0.6–3.6)	2.4 (1.1–5.4)	0.0097
<i>P</i>		0.3336	0.4497	0.0368	
Number of subjects in each quartile	62	64	62	61	
Meniscal height§	1.0	1.5 (0.7–3.3)	1.3 (0.5–3.1)	1.6 (0.7–3.8)	0.3817
<i>P</i>		0.3370	0.5457	0.2650	
Number of subjects in each quartile	61	51	54	83	
Anterior subluxation¶	1.0	2.6 (0.9–7.3)	2.9 (1.2–7.0)	2.2 (0.7–7.5)	0.0515
<i>P</i>		0.0803	0.0154	0.2043	
Number of subjects in each quartile	138	29	30	34	
Lateral meniscal damage (WORMS)#	1.0	2.5 (1.1–6.0)	4.5 (2.1–9.9)	4.2 (1.6–11.2)	<0.0001
<i>P</i>		0.0365	0.0002	0.0035	
Number of subjects in each quartile	169	31	25	29	

* Except where indicated otherwise, values are the odds ratio (95% confidence interval) adjusted for age, body mass index, sex, and tibial width.
 † Subluxation quartiles: 0 mm, 1 mm, 2–3 mm, and 4–9 mm.
 ‡ Proportion of coverage quartiles: 0.38–1.0, 0.30–0.37, 0.21–0.29, and 0–0.20.
 § Height quartiles: 7–11 mm, 6 mm, 4–5 mm, and 0–3 mm.
 ¶ Anterior subluxation quartiles: 0 mm, 1–2 mm, 3–5 mm, and 6–16 mm.
 # Whole-organ magnetic resonance imaging score (WORMS) range across all segments of the meniscus (each scored 0–4) was 0–12, with a median of 0.

Table 5. Correlation of the meniscal measures at baseline in the medial and the lateral tibiofemoral joints*

	Height	Subluxation	Anterior subluxation	Meniscal damage
Medial tibiofemoral joint				
Proportion of coverage	0.78	-0.66	-0.37	-0.59
<i>P</i>	<0.0001	<0.0001	<0.0001	<0.0001
Meniscal height		-0.64	-0.38	-0.53
<i>P</i>		<0.0001	<0.0001	<0.0001
Medial subluxation			0.41	0.54
<i>P</i>			<0.0001	<0.0001
Anterior subluxation				0.49
<i>P</i>				<0.0001
Lateral tibiofemoral joint				
Proportion of coverage	0.02	-0.47	-0.41	-0.28
<i>P</i>	0.81	<0.0001	<0.0001	<0.0001
Meniscal height		-0.12	-0.06	-0.11
<i>P</i>		0.06	0.36	0.10
Lateral subluxation			0.41	0.32
<i>P</i>			<0.0001	<0.0001
Anterior subluxation				0.31
<i>P</i>				<0.0001

* Except where indicated otherwise, values are Spearman's rank correlation coefficients.

weaker in the lateral compartment, in which severe meniscal disease was less prevalent ($r = 0.20-0.45$, all $P < 0.0001$). Because of low rates of cartilage loss in the lateral compartment, attempts to evaluate knees with each of the measures separately, meniscal damage or meniscal malposition, were unsuccessful.

DISCUSSION

This study demonstrates the strong association of meniscal position, meniscal damage, and cartilage loss. Each aspect of meniscal abnormality (whether change in position or damage) had a major effect on the risk of cartilage loss. Given the cause and effect relationship between tear and malposition, we found, as expected, that the 2 types of features were highly correlated.

A major function of the meniscus involves load bearing and shock absorption. This function is provided in part through the microstructure of the menisci, which have circumferentially oriented collagen fibers woven together with radial fibers that appear to act like tension rods to maintain shape and structure when axially loaded (16). As load is applied, meniscal fibers elongate as they are pushed toward the periphery. This hoop stress in the meniscus resists displacement by converting axial load into tensile strain. The menisci transmit anywhere from 45% to 60% of the compressive loads in the knee (1). If the meniscus does not cover the articular surface that it is designed to protect, due to change in position, or if a tear leaves it unable to resist axial loading, it will not perform this role. The absence of a functioning meniscus

increases peak and average contact stresses in the medial compartment in a range of 40–700% (17–19).

Previous studies have documented the important influence of meniscectomy on the likelihood of progressing to radiographic OA (3–7). This study clearly demonstrates the importance of meniscal damage for MRI-assessed cartilage loss.

The studies that have explored the relationship between the meniscus and risk of disease progression have been small and did not allow precise estimates of risk (20,21). Biswal et al (20) demonstrated that in 26 of 43 patients who had sustained meniscal tears, a higher average rate of progression of cartilage loss (22%) occurred compared with that seen in subjects who had intact menisci (14.9%) ($P \leq 0.018$). Berthiaume et al (21) investigated the relationship between knee meniscus structural damage and cartilage degradation in 32 subjects, and found similar effects.

This study has some limitations. The semiquantitative measure of meniscal damage does not distinguish the type of tear that may be important for cartilage loss (6). If we are to accurately determine what feature of meniscal damage increases risk of cartilage loss, we will need to develop more refined methods of measuring abnormality in the meniscus. At present, the most profound risk of cartilage loss was seen when assessing the effect of the WORMS meniscal damage variable. This does not allow for clear delineation of the type of tear, and also mixes constructs by including displacement as a variable. If the effects in the meniscus are as

potent as our results suggest, then understanding which elements of meniscal disease increase the risk of cartilage loss and why this occurs is important. Our inquiry suggests that malposition is an important piece of the puzzle, but we did not have enough subjects nor enough with cartilage loss to isolate the effects of malposition from those of meniscal integrity (if they could be separated). Furthermore, our results do not identify a particular measure of malposition that is most predictive of cartilage loss, although they suggest that subluxation in both the coronal and the sagittal planes is important in affecting cartilage loss.

Our findings were in patients with preexisting symptomatic OA. Thus, at the time of study enrollment the patients already had cartilage morphologic abnormalities and potentially other articular morphologic abnormalities that tend to occur collinearly in OA. Our study results suggest that the meniscus plays an important role in further cartilage loss; however, we cannot infer causality on the basis of these findings. Moreover, the reading of cartilage scores was conducted unblinded to sequence to increase the sensitivity to reading progression (with the readers thus being aware of the time sequence of the scans); this may have introduced bias and could lead to having cartilage scores demonstrating regression (improvement).

The MR images in this study were obtained with the patients in a supine position and on non-weight-bearing knees. The measures of meniscal position are thus likely to have yielded conservative values as compared with those that would be obtained during axial loading of the knee.

This study highlights the importance of an intact and functioning meniscus in patients with symptomatic knee OA. It demonstrates that abnormal meniscal function has important consequences for the rate of cartilage loss. At present, efforts are being made to preserve a damaged meniscus rather than remove it, and an industry of meniscal replacement is developing. Our study points to the need for critical, prospective evaluation of these new therapeutic options.

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