

Karim M. Khan, BMedSci, MBBS • Fiona Bonar, MBBCh, BAO • Patricia M. Desmond, MSc, MBBS
Jill L. Cook, BAppSci • David A. Young, MBBS • Paul J. Visentini, BAppSci • Michael W. Fehrmann, ME
Zoltan S. Kiss, MBBS • Patricia A. O'Brien, BA, MBA • Peter R. Harcourt, MBBS • Richard J. Dowling, MBBS
Richard M. O'Sullivan, BSc, MBBS • Kenneth J. Crichton, MBBS • Brian M. Tress, MBBS, MD
John D. Wark, MBBS, PhD • Victorian Institute of Sport Tendon Study Group

Patellar Tendinosis (Jumper's Knee): Findings at Histopathologic Examination, US, and MR Imaging¹

PURPOSE: To determine the histopathologic findings of patellar tendinosis ("jumper's knee") demonstrated with ultrasonography (US) and magnetic resonance (MR) imaging.

MATERIALS AND METHODS:

Twenty-four athletes (28 knees) with jumper's knee (23 men, one woman; mean age, 30.9 years) scheduled to undergo open tenotomy underwent US patellar tendon examination. Seventeen patients (19 knees) also underwent MR imaging. Tissue was obtained for histopathologic examination in all 28 cases. Eleven age-, height-, and weight-matched athletes (22 knees) without previous knee symptoms served as control subjects for the US examination. Control material for histopathologic examination was obtained in 20 cadavers (39 knees). Data were analyzed with standard statistical methods.

RESULTS: MR imaging and US both revealed an abnormal zone at the proximal patellar tendon attachment. Histopathologic examination revealed mucoid degeneration in all tendons in patients and in 8% (three of 39) of tendons in cadavers ($P < .01$).

CONCLUSION: Jumper's knee is characterized by consistent changes at MR imaging, US, and histopathologic examination and is appropriately described as patellar tendinosis.

THE clinical entity of "jumper's knee" causes substantial morbidity in elite and recreational athletes, especially those who play basketball, volleyball, soccer, tennis, and track (1). The syndrome refers to anterior knee pain associated with tenderness of the patellar tendon near its patellar attachment (1-3). In this article, we use "jumper's knee" to refer to the clinical syndrome and "patellar tendinosis" to refer to histopathologically proved tendon disease. We eschew use of the misnomer "patellar tendinitis."

Ultrasonography (US) and magnetic resonance (MR) imaging can be used to detect abnormalities in the patellar tendon itself (4-12) and help differentiate the condition from patellofemoral syndrome in clinical practice (13). Imaging has also been used to guide clinicians as to the severity of jumper's knee.

Various pathologic changes have been reported in jumper's knee. These include pseudocyst change (14,15), fibrinoid necrosis (16,17), mucoid degeneration (17,18), randomized collagen with neovascularization and tenocyte infiltration (17,19), microtears of the tendinous tissues (17,19), chronic inflammatory cell

infiltration (4), focal degeneration near the bone-tendon insertion (17,19,20), and angiofibroblastic tendinosis (8). Some of these changes may be due to the effect of hydrocortisone injection (17) rather than to the primary tendon abnormality (15). The aim of this study was to document the pathologic changes in cases of jumper's knee demonstrated with US and MR imaging. In addition, we compared the dimensions of abnormalities seen at US and MR imaging.

MATERIALS AND METHODS

Collaborating surgeons notified a central registry when they scheduled patients with jumper's knee for open tenotomy. The decision to perform surgery was made on the basis of failed conservative management. In all cases, surgeons used US to confirm the diagnosis and the location of the abnormality. All patients who were identified in this way agreed to participate in this study; thus, the study group consisted of consecutive individuals undergoing patellar tenotomy for jumper's knee. At the time of the study, MR imaging findings were not used for clinical decision making in jumper's knee. Patients underwent MR imaging for research purposes if it could be scheduled between

Index terms: Knee, MR, 4528.121412, 4528.121411 • Knee, US, 4528.1298 • Tendinitis, 4528.253 • Tendons, 4528.259 • Tendons, injuries, 4528.259 • Tendons, MR, 4528.121412, 4528.121411 • Tendons, US, 4528.1298

Abbreviations: GRE = gradient echo, SE = spin echo, STIR = short inversion time inversion recovery.

Radiology 1996; 200:821-827

¹ From the Departments of Medicine (K.M.K., J.D.W.) and Radiology (P.M.D., R.J.D., B.M.T.), Royal Melbourne Hospital, Parkville, Victoria, 3050 Australia; the Victorian Institute of Sport, South Melbourne, Australia (F.B., J.L.C., D.A.Y., P.J.V., M.W.F., Z.S.K., P.R.H., R.M.O., K.J.C.); the Department of Forensic Medicine, Monash University, South Melbourne, Australia (P.A.O.); and Hanly-Moir Pathology, North Ryde, Australia (F.B.). Received December 27, 1995; revision requested February 2, 1996; revision received April 8; accepted April 15. Supported in part by the Australian Sports Research Programme; the Sports Medicine Australia-Syntex Research Foundation; Australian National Health and Medical Research Council Medical Postgraduate Research Scholarship 958160; the Alphonso Sports Medicine Clinic; the Department of Radiology, Royal Melbourne Hospital; Basketball Australia; and the Victorian Institute of Sport. Address reprint requests to B.M.T.

recruitment and the date of surgery. Asymptomatic volunteers were recruited as control subjects for US. Cadavers were used to provide control tissue for histopathologic analysis.

Clinical Assessment

An investigator (K.M.K.) administered a questionnaire to patients and control subjects about their age, height, weight, sports played, level of competition, side(s) affected (for patients), and type of treatment received (for patients). Severity of pain was assessed with common clinical questions about pain at rest and with exercise (2) and by using the seven-point Nirschl Pain Phasing System, which was combined with a visual analog scale (21).

Patients had their knees examined clinically by two treating physicians (sometimes D.A.Y., P.R.H.) and one investigator physician (K.M.K.). Control subjects had their knees examined clinically by one investigator physician (K.M.K.). Tenderness was recorded as present or absent and, when present, its location was noted.

Patient, Control Subject, and Cadaver Characteristics

Open patellar tenotomy was performed in 24 patients (28 knees). There were 23 men and one woman (age range, 18–43 years). Eleven athletes who did not recall ever having symptoms of anterior knee pain served as a specific US control group (22 tendons). These subjects were selected from a large database of asymptomatic athletes (J Cook et al, unpublished data, 1996) (22) to match the patients for age, height, and weight. Thirteen of the 24 patients scheduled to undergo surgery who had unilateral symptoms also underwent US examination of their asymptomatic tendon. These subjects, however, were not included in the US control group. Specimens were obtained in 20 cadavers (39 tendons; 18 men, two women; aged 24–80 years). The mean age, height, and weight of the patients, control subjects, and cadavers are reported in the Table. There was no statistically significant difference in the mean age, height, and weight of the patients and control subjects. The cadavers, however, were older, shorter, and lighter than the patients (Table).

Clinical Features

In the patients, 14 right and 14 left tendons were affected by substantial symptoms. The most aggravating sport was basketball (eight patients), followed by running (six patients), Australian-rules football (two patients) and soccer (one patient), squash (two patients), cricket (three patients), karate (one patient), and equestrian (one patient). Eleven of the 24 (46%) athletes reported bilateral symptoms. Of these, five had previously undergone contralateral tendon surgery and four were undergoing bilateral surgery at the time of this study. All patients had pain with exertion. All

Characteristics of Patients, Control Subjects, and Cadavers

Characteristic	Patients (n = 24)	Control Subjects (n = 11)	Cadavers (n = 20)
No. of patellar tendons studied	28	22	39
Mean age (y)	30.9 (1.7)	31.5 (1.1)	60.8 (4.4)*
Mean height (cm)	180.0 (1.8)	185.5 (2.1)	169 (2.5)*
Mean weight (kg)	81.2 (2.3)	83.4 (3.7)	68.8 (4.6)*
Mean duration of symptoms (mo)	35.4 (8.2)	0 (0)	...

Note.—Numbers in parentheses are standard errors.

* $P < .05$ for patients versus cadavers.

but four patients had pain with light activities of daily living (Nirschl phase VI). Seven patients had a constant ache at rest and pain that disturbed sleep (Nirschl phase VII). The mean (\pm standard error) Nirschl pain phase score was 5.9 ± 0.2 .

The amount of time that jumper's knee kept the patients from competition varied from none, where the patient had reduced training but still competed weekly, to more than 12 months (mean \pm standard error, 4.6 months \pm 1.2). Cortisone acetate had been injected in eight patients. Three of those patients reported that cortisone acetate made no difference; the remainder reported relief of symptoms for 1 week to 6 months.

The two treating physicians and one investigating physician all detected tenderness at the junction of the inferior pole of the patella and the tendon that corresponded to the site of the lesion at imaging in all patients. The physicians agreed that jumper's knee was the clinical diagnosis. None of the athletes in the US control group had marked tenderness at this site.

Imaging

As part of their preoperative work-up, all patients underwent US of both knees by using linear-array transducers (Dornier—Acoustic Imaging, Phoenix, Ariz) at 7.5 or 10.0 MHz. US was performed by one of four radiologists (including Z.S.K., R.J.D.). The radiologists took care to examine the tendon with the probe exactly perpendicular to the tendon to avoid a false-positive finding due to hypoechoic artifacts (23).

Patients underwent MR imaging with a high-field-strength (1.5-T) unit (GE Medical Systems, Milwaukee, Wis) at one of two centers. Only symptomatic knees were examined. Multiple sagittal and axial sequences were performed by using a 3" surface coil placed directly over the patellar tendon. The following sequences were performed: two-dimensional, T1-weighted, sagittal, spin-echo (SE) imaging (400/20 [repetition time msec/echo time msec], 3.0-mm-thick sections with no gap, 12-cm field of view, 256 \times 256 matrix, two signals acquired, imaging time of 3 minutes 31 seconds); T2-weighted, sagittal, fast SE imaging with fat saturation (3,000/110 [effective echo time], echo train length of eight, 12-cm field of view, 3.0-mm-thick sections with no gap, 512 \times 256 matrix, two signals acquired, imaging time of 3 minutes 24 seconds); two-dimensional,

T2'-weighted, sagittal, gradient-echo (GRE) imaging (800/30, 70° flip angle, 12-cm field of view, 3.0-mm-thick sections with no gap, 256 \times 256 matrix, one and a half signals acquired, imaging time of 5 minutes 10 seconds); two-dimensional, sagittal, short inversion time inversion-recovery (STIR) imaging (4,000/17 with an inversion time of 140 msec [4,000/17/140], echo train length of six, 12-cm field of view, 3.0-mm-thick sections with no gap, 256 \times 192 matrix, two signals acquired, imaging time of 4 minutes 32 seconds); and two-dimensional, axial, T1-weighted imaging (600/20, 3.0-mm-thick sections with no gap, 12-cm field of view, 256 \times 256 matrix, one and a half signals acquired, imaging time of 3 minutes 59 seconds).

MR imaging findings were initially reported by four MR radiologists (including P.M.D., R.M.O.) who were blinded to the findings of US. These MR reports were rendered irrelevant by the subsequent controlled, blinded multiobserver analysis performed by two MR radiologists (P.M.D., B.M.T.). These investigators evaluated the MR images obtained in all subjects who had undergone imaging with a single magnet at Royal Melbourne Hospital, Parkville, Australia (12 patients [14 tendons]).

To analyze images obtained with the four sagittal sequences, they chose one sagittal T1-weighted image that showed the abnormal region to be largest and recorded the section position (eg, 2.54 right). For each of the other sagittal sequences (T2'-weighted GRE, T2-weighted fast SE, STIR), they independently selected an image that corresponded to the section position they had agreed on for the T1-weighted sequence. The following measurements were obtained on the sagittal images: (a) anteroposterior size of the tendon, (b) anteroposterior dimension of the area of abnormal signal intensity, and (c) craniocaudal (superoinferior) dimension of the area of abnormal signal intensity. To analyze the T1-weighted axial sequence, the radiologists conferred to determine the section that showed the abnormal region to be largest. They then independently measured the anteroposterior and mediolateral dimensions of the area of abnormal signal intensity.

Thus, in each patient, both reporting radiologists performed 12 measurements with the sagittal sequences (three measurements \times four sequences) and two mea-

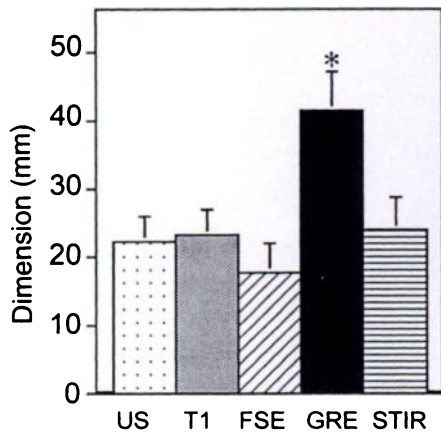


Figure 1. Mean length (\pm standard error) of the largest lesion (hypoechoic region at US and area of abnormal signal intensity at MR imaging) in the craniocaudal (superoinferior) direction. * indicates $P < .05$ in a comparison with all other modalities. FSE = fast SE MR imaging, T1 = T1-weighted MR imaging.

measurements with the axial sequence. Results showed acceptable levels of interobserver agreement, with Pearson r values of .89–.99 for the 14 sets of measurements.

To compare the size of the US abnormalities with those seen on MR images, the site at which the lesion appeared maximal on US scans was identified and measured in the sagittal plane. The craniocaudal (superoinferior) and anteroposterior dimensions were measured independently, before MR imaging, by the US radiologists (including Z.S.K., R.J.D.).

Surgery

All patients underwent open tenotomy. In each case, the patellar tendon was examined macroscopically, and the specimen of macroscopically abnormal tissue was excised in toto. The proximal portion of the excised tendon (patellar insertion site) was identified with a suture before the specimen was placed in formalin.

Histopathologic Examination

All specimens were examined by a musculoskeletal histopathologist (F.B.). The patellar insertion site was marked for recognition microscopically, and specimens were serially sliced longitudinally. Longitudinal slices enabled changes to be recognized throughout the length of the tendon to correspond to US and MR findings.

Specimens were stained with hematoxylin and eosin and examined with polarization microscopy. Polarization microscopy is a simple and useful method of assessing the continuity and alignment of collagen fibers. Special stains used were alcian blue (pH, 2.5), to detect any increase in ground substance; Prussian blue, to detect hemosiderin (suggests previous bleeding into the tendon); and immunoperoxidase, to detect smooth muscle actin by using an avidin biotin technique at a dilution of 1/50 (Dako, Carpinteria, Calif) to detect myofibroblastic

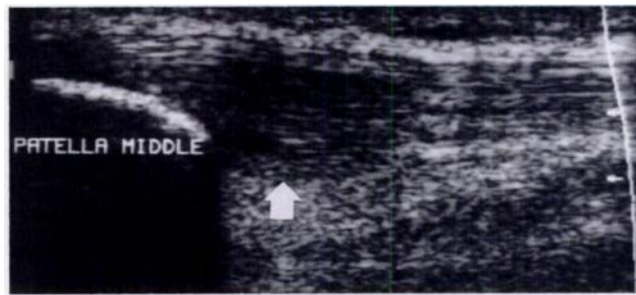


Figure 2. Longitudinal (sagittal) US scan of the patellar tendon in an 18-year-old man with jumper's knee shows a focal hypoechoic region (arrow) at the deep portion of the tendon adjacent to the lower pole of the patella.

and vascular proliferation. There was no attempt to quantify the abnormality seen at histopathologic examination.

Patellar tendon specimens were obtained from sequential cadavers that met the Victorian Institute of Forensic Pathology (Melbourne, Australia) ethics committee criteria for the study (ie, there were no known relatives and death resulted from trauma). We examined the specific region of cadaver tendon (patellar insertion) that corresponded to the site of surgery in the patients.

Statistical analysis was performed by using the STATVIEWSE+ program (Abacus Concepts, Berkeley, Calif). The age, height, and weight of the patients, control subjects, and cadavers were compared by using the analysis of variance test. Interobserver correlations for MR measurements were performed by using Pearson r values. The differences in the size of the tendon lesion measured at US and with each MR sequence was determined by using repeated-measures analysis of variance followed by posthoc Fisher multiple comparison tests. The prevalences of tendon abnormalities in the patients and control subjects were compared by using a $2 \times 2 \chi^2$ test. Statistical significance was determined to be at the .05 level.

The ethics committees of the Royal Melbourne Hospital and the Victorian Institute of Forensic Pathology approved the study. All patients and control subjects gave informed consent.

RESULTS

Imaging Results

Maximal lesion dimensions.—The maximal craniocaudal (superoinferior) dimension of tendon lesions measured on US scans (longitudinal [sagittal] image) was compared with that measured on the corresponding MR images obtained with each of the four sagittal sequences. The mean craniocaudal (superoinferior) dimensions of the tendon lesions measured at US and MR imaging are given in Figure 1. The lesions had greater superoinferior (craniocaudal) dimensions with the T2'-weighted GRE sequence than with US and the other MR sequences ($P < .05$) (Fig 1). There

were no statistically significant differences between the mean anteroposterior dimensions of lesions measured at US and those measured with the various MR sequences. Also, there were no statistically significant differences between the mean anteroposterior dimensions of the tendon measured with US and those measured with the various MR sequences.

US appearance.—Twenty-five of the 28 symptomatic knees had one hypoechoic area in the proximal portion of the patellar tendon at US. Three knees had two hypoechoic lesions. US appearances in all 28 cases consisted of a focal hypoechoic area (Fig 2) combined with various amounts of swelling of the surrounding tendon. Hyperechoic regions within the tendon, considered to be calcification, were seen in eight of the 28 tendons. Dystrophic ossification was present at histopathologic examination in all eight cases.

In the control subjects, who were athletes who had never reported symptoms, four of the 22 tendons (18%) had a focal hypoechoic region. Of the 13 patients with jumper's knee whose contralateral patellar tendon had never been symptomatic, two (15%) had a hypoechoic region.

MR imaging appearance.—All patients had an area of abnormal signal intensity within the tendon at the junction with the patella (Figs 3, 4). Lesions had increased signal intensity relative to the tendon on T1-weighted images (Figs 3a, 4) and had markedly increased signal intensity on T2'-weighted GRE images (Fig 3b), T2-weighted fast SE images (Fig 3c), and STIR images (Fig 3d). Maximal anteroposterior and craniocaudal (superoinferior) lesion sizes were compared with those in each of the sagittal sequences. In all but one case, the maximal dimension of the lesion was greater with the T2'-weighted GRE sequence than with the other sagittal MR sequences.

The T2-weighted STIR and fast SE sequences revealed abnormal signal intensity in the patella (Figs 3c, 3d) in all but one case. The T1-weighted sequence (Fig 3a) revealed this abnormality in only seven of 19 cases (37%).

Histopathologic Findings

Cadavers.—Tendons measured approximately 35 × 25 × 10 mm and were composed of glistening, stringy white tendinous tissue. Thirty-four of the 39 tendons (87%) appeared entirely normal microscopically. They were composed of dense fibrotendinous tissue (Fig 5a) with a dense, homogeneous polarization pattern (Fig 5b). The bundles of collagen had smooth borders, inconspicuous tenocytes, an absence of stainable ground substance, inconspicuous vasculature, and no evidence of fibroblastic or myofibroblastic proliferation.

Three of the 39 tendons (8%) showed mild changes similar to those noted in all patients (see below). In addition, one tendon showed an increase in ground substance in the paratendinous fibroadipose tissue (the tendon collagen itself was unremarkable) and one showed scarring and traumatic neuroma formation consistent with previous trauma.

Patients.—Twenty-eight tendons were examined. The specimens measured an average of 20 × 15 × 8 mm and lacked the distinctive stringy appearance seen in normal tendons.

The histopathologic findings in all cases were essentially similar: relative expansion of the tendinous tissue with loss of clear demarcation of collagen bundles accompanied by a loss of the normal dense, homogeneous polarization pattern (Figs 5c, 5d). In most instances, a definite vector that marked the transition from normal to abnormal could be seen—with the more marked pathologic findings proximally situated (ie, close to the patellar insertion site).

The abnormality manifested as a gradual and increasing separation of collagen fibers at polarization light microscopy with a gradual increase in mucoid ground substance, which was confirmed with use of alcian blue stain. Maximal mucoid change was present proximally at the insertion site where tenocytes, when present, were plump and chondroid in appearance (exaggerated fibrocartilaginous metaplasia) (Figs 6a, 6b). These changes were accompanied by increasingly conspicuous cells within the tendinous tissue, most of which had a fibroblastic and myofibroblastic appearance.

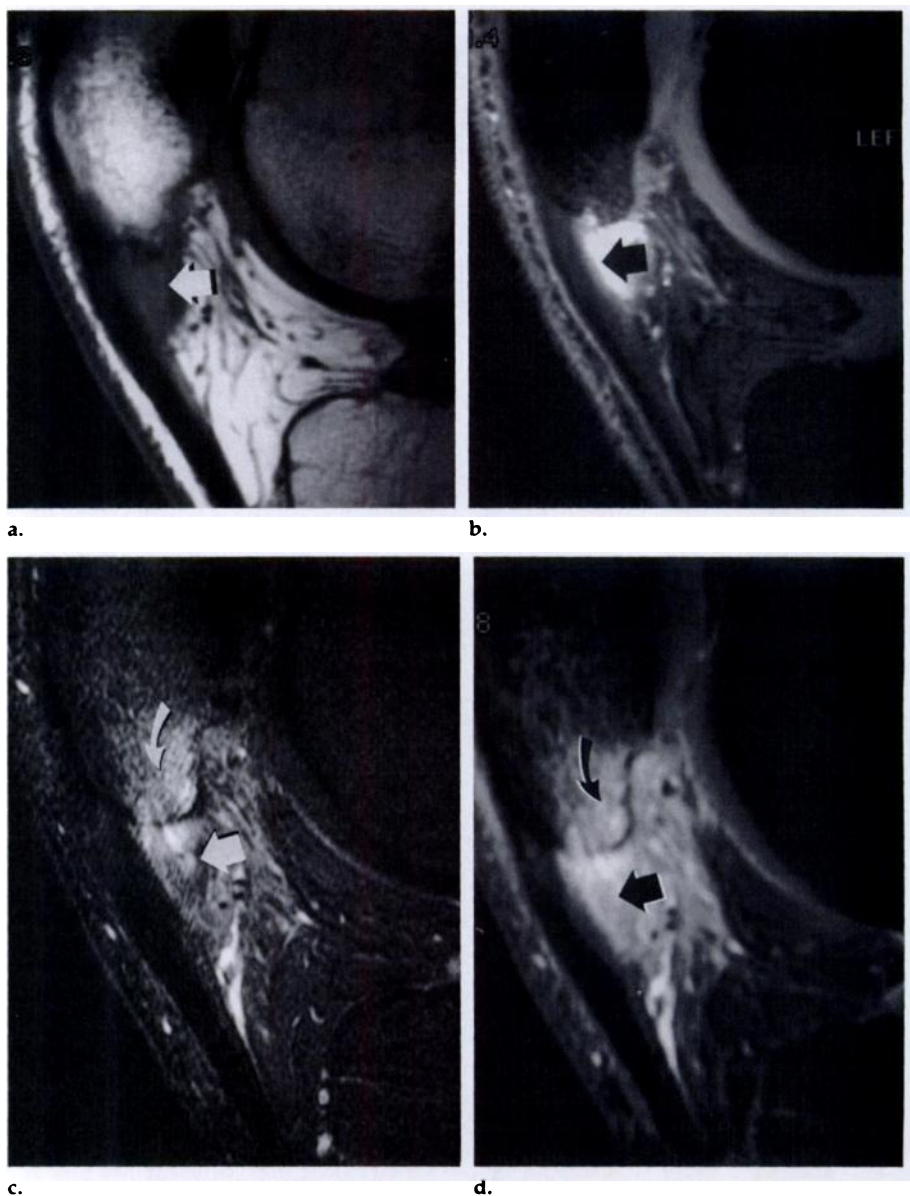


Figure 3. Sagittal MR images obtained in an 18-year-old man with jumper's knee. (a) T1-weighted SE image (400/20) shows an area of increased signal intensity (arrow) relative to that of the tendon. (b) T2*-weighted GRE image (800/30, 70° flip angle) shows markedly hyperintense signal intensity in the tendon at the attachment to the patella. The posterior margin of the patella is irregular and poorly defined (arrow). (c) T2-weighted fast SE image (3,000/110 [effective]) shows a marked increase in signal intensity (straight arrow) in the tendon and increased signal intensity in the lower end of the patella itself (curved arrow). (d) STIR image (4,000/17/140) shows increased signal intensity in the tendon (straight arrow) and patella (curved arrow).

The cellular proliferation was maximal centrally within the tendinous tissue and was accompanied by a prominence of capillary proliferation (Fig 6c). Polarization microscopy showed a tendency to discontinuity of collagen fibers in this area (Fig 5d). In two cases, foci of ossification (traction lesions of bone as distinct from dystrophic ossification) were present in this region.

A distinctive feature noted in all instances was an abrupt discontinuity of both vascular and myofibroblastic proliferation just before the area of maximal mucoid degeneration and

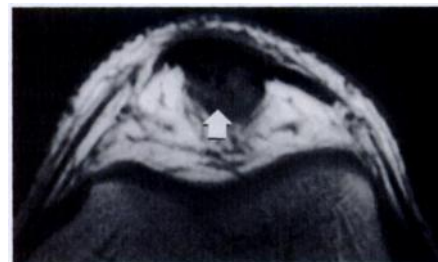


Figure 4. Axial T1-weighted MR image (600/20) obtained in an 18-year-old man with jumper's knee. This image helps localize the lesion in the axial plane, and, in this case, the increase in signal intensity (arrow) arises from the medial aspect of the tendon.

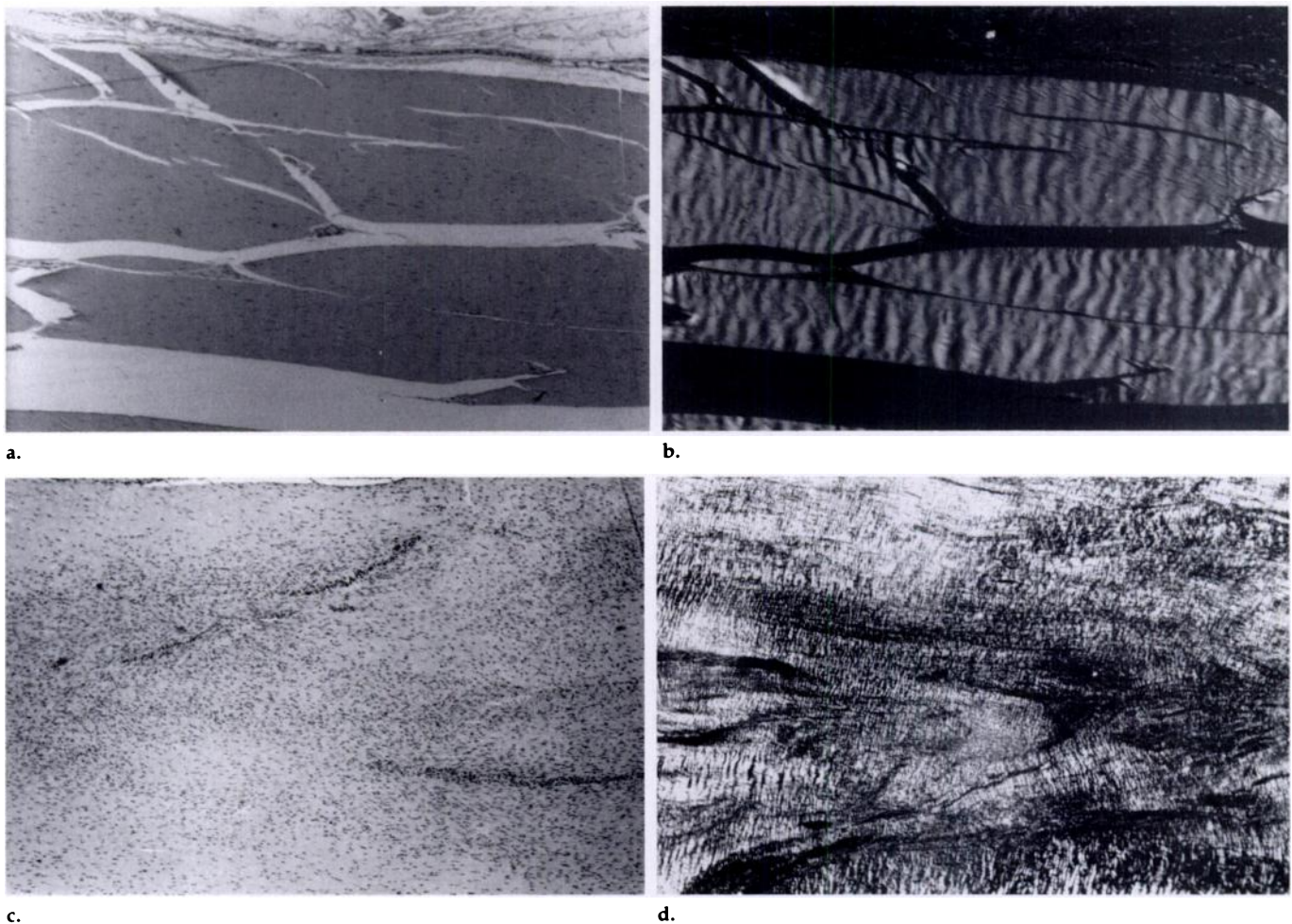


Figure 5. (a, b) Specimens from a male cadaver (37 years old at death). (a) Specimen from a normal tendon (hematoxylin-eosin stain; original magnification, $\times 40$). The tendon appears smooth, and tenocytes and blood vessels are inconspicuous. (b) Polarization microscopic specimen (hematoxylin-eosin stain; original magnification, $\times 40$) shows tightly cohesive continuous collagen bundles with a dense, homogeneous polarization pattern. Normal crimping is evident. (c, d) Corresponding tissue specimens from an abnormal tendon in an 18-year-old man with jumper's knee. (c) Specimen (hematoxylin-eosin stain; original magnification, $\times 40$) shows loss of the normal smooth tendon appearance, an increased number of fibroblasts and myofibroblasts, and prominent blood vessels. (d) Polarization microscopic specimen (hematoxylin-eosin stain; original magnification, $\times 20$) demonstrates a loss of the normal, dense, homogeneous polarization pattern, separation of collagen fibers, and focal discontinuity of fibers.

fibrocartilaginous metaplasia present at the area of insertion into the patella (Fig 7).

In most cases, a demonstrable increase in mucoid ground substance could be seen in surrounding fibrofatty tissue. Inflammatory cells were not seen in any specimen. The findings seen in all cases were constant, irrespective of whether cortisone injection had been administered to the patient.

Results of the χ^2 test revealed that the proportion of patellar tendons with changes of mucoid degeneration (28 of 28 = 100%) was greater than that of cadaver tendons with similar changes (three of 39 = 8%) ($P < .001$).

DISCUSSION

Imaging Aspects

In our study, the sagittal T2'-weighted GRE sequence revealed a

statistically significantly larger area of high signal intensity than did the T1-weighted and T2-weighted fast SE sequences (Figs 1, 3a-3c). Increased signal intensity in tendons on T2'-weighted GRE images can arise owing to the "magic angle" phenomenon (24). However, this does not appear to explain our finding for several reasons. First, in our MR coil arrangement, the proximal patellar tendon courses at less than 30° to the constant magnetic induction field (Fig 2). Second, the region of increased signal intensity affects only the posterior part of the tendon, even when both anterior and posterior portions of the tendon run parallel to each other. Third, the area of abnormal signal intensity extends into the paratendinous fat deep to the tendon. Fat does not have the properties of collagen that are necessary to permit the magic angle phenomenon to occur

(25). Fourth, the area of abnormal signal intensity extends from the patellar insertion to the midtendon despite changes in the angle of the tendon relative to the magnetic field. Fifth, our patients had abnormal signals at US. We do, however, note the presence of the magic angle phenomenon at the tibial insertion of the patella where the tendon courses at 55° to the magnetic field in some patients. These distal regions of abnormal signal intensity were not included in our analysis.

Areas of increased signal intensity at T2'-weighted GRE imaging have been reported at the proximal and distal ends of the patellar tendon in asymptomatic individuals independently of magic angle findings (26). However, the region of abnormal signal intensity seen at T2'-weighted GRE imaging in our patients extended far beyond the patellar attach-

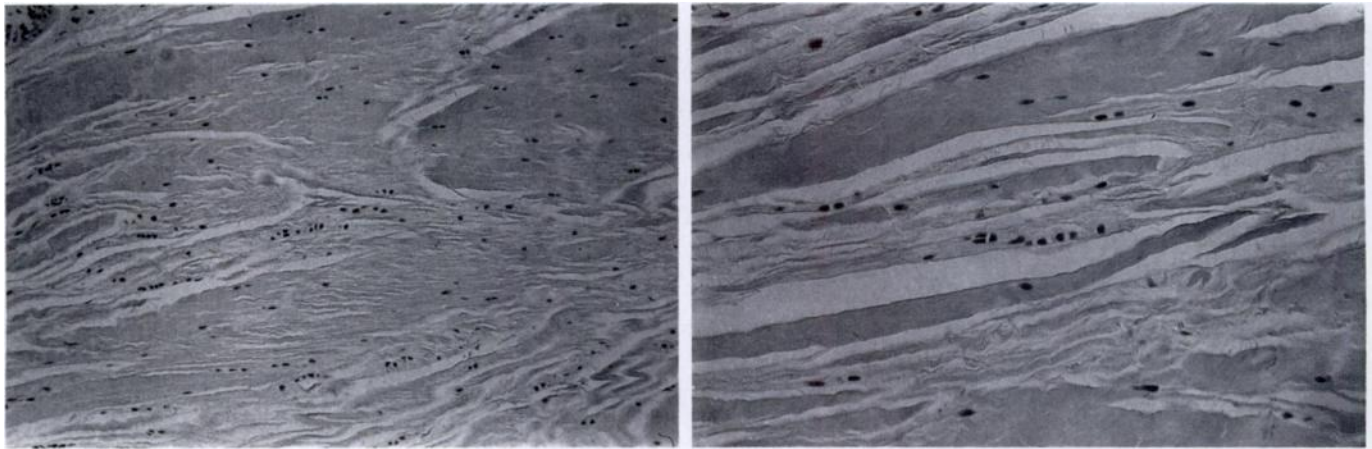


Figure 6. Histopathologic changes in an 18-year-old man with jumper's knee. (a) Specimen obtained close to the patellar insertion site shows separation of collagen (hematoxylin-eosin stain; original magnification, $\times 100$). (The intervening spaces can be shown to have an increased mucoïd ground substance by means of alcian blue stain.) Tenocytes are plumper and more chondroid in appearance. (b) Same specimen as in a at a higher power (hematoxylin-eosin stain; original magnification, $\times 200$) shows plumper tenocytes with a chondroid appearance. (c) Specimen from the pathologic region of the tendon shows a marked increase in cellularity (hematoxylin-eosin stain; original magnification, $\times 200$).

ment site described by Reiff and colleagues (26).

We postulate that the larger area of increased signal intensity on T2'-weighted GRE images may reflect increased sensitivity to minor damage in the tendon. If this were the case, then the T2'-weighted GRE sequence could prove to be an effective method for monitoring elite high-risk athletes (eg, in professional basketball players during a period of intense training). In this scenario, asymptomatic tendon strain detected with T2'-weighted GRE imaging, might be a precursor to frank tendon damage. McLoughlin et al (12) have also noted this phenomenon of increased signal intensity on T2'-weighted GRE images relative to that seen on T2-weighted fast SE images and have speculated as to its histopathologic importance.

The T2-weighted STIR and fast SE sequences revealed abnormal signal intensity in the patella (Figs 3c, 3d) in most of the patients in this study. It is not known whether the enthesopathy of patellar tendinosis causes the patella itself to contribute to the pain of jumper's knee. If it did contribute to the pain, this would have implications for management.

Histopathologic Aspects

We reported a definite craniocaudal (superoinferior) vector in jumper's knee with more marked findings at

the proximal (patellar) end of the tendon. Because our surgical specimens were labeled to permit orientation, we were able to confidently correlate abnormal histopathologic findings with US and MR imaging appearances. Measures of the severity of the condition (ie, abnormality shown with light microscopy) (Fig 5) and the amount of abnormal ground substance between the tenocytes (Fig 6) point to greater mucoïd degeneration at the patellar insertion that decreases distally toward the tibia.

Inflammatory cells were totally absent at the regions of the greatest mucoïd degeneration (Fig 6) and at the margins of the specimens, where the mucoïd degeneration was least. Even in two cases in patients who underwent surgery after only 4 months of symptoms, inflammatory cells were absent. This suggests that the inflammatory component in this condition, if present at any stage, does not persist.

Whether patellar tendinosis results from poor vascularity of the tendon insertion (27,28) or from tearing of collagen and secondary mucoïd degeneration is not known. Histopatho-

logic changes of ongoing reparative fibrosis, however, are evident in each case in this study (fibroblastic, myofibroblastic, and vascular proliferation), and this may reflect repeated minor trauma.

Surgical treatment for jumper's knee is recommended only "if a prolonged and well-supervised conservative treatment program fails" (29). A variety of procedures, differing in the relative amount of bone surgery involved, have been reported (1,29). There are no reports of the rationale for these procedures, but presumably excision of degenerative, painful tissue is designed to promote healing (30).

Another treatment of jumper's knee in clinical practice is cortisone injection (31), although some authors discourage its use (29). In this study of patients in whom conservative management failed, we could not differentiate tendons that had been injected from those that had not with either radiologic or histopathologic tests. In these cases, cortisone did not permanently affect the tendon or did not reach the site eventually excised. Well-designed clinical and basic stud-



Figure 7. Specimen from the area close to the patellar insertion site in an 18-year-old man with jumper's knee reveals discontinuity of vascular proliferation and decreased myofibroblastic proliferation (straight arrows) just before the area of maximal mucoid degeneration (curved arrow) (immunoperoxidase stain for smooth muscle actin; original magnification, $\times 40$).

ies of the effect of cortisone injection in tendon disorders are needed.

In summary, our data reveal that athletes with severe cases of jumper's knee, confirmed by means of US and MR imaging, clearly have tendinosis. This tendinosis is not a normal feature of either aged or young cadaveric patellar tendons. Because jumper's knee has been shown to be patellar tendinosis, we recommend that treatment protocols address this disease rather than a supposed inflammatory tendinitis. ■

Acknowledgments: The authors thank the staff of the Departments of Medicine and Radiology at the Royal Melbourne Hospital, Parkville, Australia, especially Pam Lukaszewski, Mark Stein, Kim Bennell, Richard Larkins, Stephen McEwan, Jaya Sathasivam, Debbie Howard, and David Witte. The authors also thank Meditron, Ringwood, Australia, for use of their equipment. The investigators in the Victorian Institute of Sport Tendon Study are as follows: Ian Anderson, John Bartlett, Simon Bell, Fiona Bonar, David Bracy, Christopher Bradshaw, Francis Burke, Bruce Caldwell, Jill Cook, Ken Crichton, Rodney Dalziel, Patricia Desmond, Richard Dowling, Peter Ebeling, Michael Fehrmann, Peter Fuller, Andrew Garnham, Margaret Grant, Peter Harcourt, William Hare, Ian Henderson, Duncan Kellaway, Karim Khan, Zoltan S. Kiss, Peter Larkins, Patricia O'Brien, Richard O'Sullivan, Clive Morris, Craig Purdham, Ronald Quirk, John Read, Ronald Shnier, Brian Tress, Paul Visentini, John Wark, Peter Wilson, and David Young. The following institutions are represented: Departments of Medicine and Radiology, Royal Melbourne Hospital, University of Melbourne, Melbourne, Australia; Australian Institute of Sport, Canberra, Australia; and Victorian Institute of Sport, South Melbourne, Australia.

References

- Blazina M, Kerlan R, Jobe F, Carter V, Carlson G. Jumper's knee. *Orthop Clin North Am* 1973; 4:665-678.
- Brukner P, Khan K. *Clinical sports medicine*. Sydney, Australia: McGraw-Hill, 1993.
- Crichton KJ, Fricker PA, Purdam CR, Watson AS. Injuries to the pelvis and lower limb. In: Bloomfield J, Fricker PA, Fitch KD, eds. *Textbook of science and medicine in sport*. Melbourne, Australia: Blackwell Scientific, 1992; 381-419.
- Mourad K, King J, Guggiana P. Computed tomography and ultrasound imaging of jumpers knee—patellar tendinitis. *Clin Radiol* 1988; 39:162-165.
- Myllymäki T, Bondestam S, Suramo I, Cederberger A, Peltokallio P. Ultrasonography of jumpers knee. *Acta Radiol* 1990; 31:147-149.
- Davies SC, Baudouin CJ, King JB, Perry JD. Ultrasound, computed tomography and magnetic resonance imaging in patellar tendinitis. *Clin Radiol* 1991; 43:52-56.
- El-Khoury GY, Wira RL, Berbaum KS, Pope TL, Monu JUV. MR imaging of patellar tendinitis. *Radiology* 1992; 184:849-854.
- Yu JS, Popp JE, Kaeding CC, Lucas J. Correlation of MR imaging and pathologic findings in athletes undergoing surgery for chronic patellar tendinitis. *AJR* 1995; 165:115-118.
- Fornage BD, Rifkin MD. Ultrasound examination of tendons. *Radiol Clin North Am* 1988; 26:87-107.
- Fritschy D, de Gautard R. Jumpers knee and ultrasonography. *Am J Sports Med* 1988; 16:637-640.
- Karlsson J, Kalebo P, Goksoer LA, Thomee R, Sward L. Partial rupture of the patellar ligament. *Am J Sports Med* 1992; 20:390-395.
- McLoughlin RF, Raber EL, Vellel AD, Wiley JP, Bray RC. Patellar tendinitis: MR features, with suggested pathogenesis and proposed classification. *Radiology* 1995; 197:843-848.
- McConnell J. The management of chondromalacia patellae: a long-term solution. *Aus J Physiol* 1986; 32:215-223.
- Maffulli N, Regine R, Carrillo F, et al. Ultrasonographic scan in knee pain in athletes. *Br J Sports Med* 1992; 26:93-96.
- Ferretti A, Ippolito E, Mariani P, Puddu G. Jumpers knee. *Am J Sports Med* 1983; 11:58-62.
- Kannus P, Jozsa L. Histopathological changes preceding spontaneous rupture of a tendon. *J Bone Joint Surg [Am]* 1991; 73:1507-1525.
- Roels J, Martens M, Mulier JC, Burssens A. Patellar tendinitis (jumpers knee). *Am J Sports Med* 1978; 6:362-368.
- Józsa L, Reffy A, Kannus P, Demel S, Elek E. Pathological alterations in human tendons. *Arch Orthop Trauma Surg* 1990; 110:15-21.
- Raatikainen T, Karpakka J, Puranen J, Orava S. Operative treatment of partial rupture of the patellar ligament: a study of 138 cases. *Int J Sports Med* 1994; 15:46-49.
- Davidson L, Salo M. Pathogenesis of subcutaneous tendon ruptures. *Acta Chir Scand* 1969; 135:209-212.
- Nirschl RP. Elbow tendinosis/tennis elbow. *Clin Sports Med* 1992; 11:851-870.
- Fornage BD. The hypoechoic normal tendon: a pitfall. *J Ultrasound Med* 1987; 6:19-22.
- Cook J, Harcourt P, Khan K, et al. Ultrasound appearance of the patellar tendon in elite athletes: gender and sport differences (abstr). In: *Proceedings of the Australian Conference of Science and Medicine in Sport, 1995*; Canberra, Australia: Sports Medicine Australia.
- Erickson SJ, Cox IH, Hyde JS, Carrera GF, Strandt JA, Estkowski LD. Effect of tendon orientation on MR imaging signal intensity: a manifestation of the magic angle phenomenon. *Radiology* 1991; 181:389-392.
- Erickson SJ, Prost RW, Timins ME. The magic angle effect: background physics and clinical relevance. *Radiology* 1993; 188:23-25.
- Reiff DB, Heenan SD, Heron CW. MRI appearance of the asymptomatic patellar tendon on gradient echo imaging. *Skeletal Radiol* 1995; 24:123-126.
- Björkstöm S, Goldie IF. A study of the arterial supply of the patella in the normal state, in chondromalacia patellae and in osteoarthritis. *Acta Orthop Scand* 1980; 51:63-70.
- Scapinelli R. Studies on the vasculature of the human knee joint. *Acta Anat* 1968; 70:305-331.
- Karlsson J, Lundin O, Lossing IW, Peterson L. Partial rupture of the patellar ligament: results after operative treatment. *Am J Sports Med* 1991; 19:403-408.
- Colosimo AJ, Bassett FH. Jumpers knee: diagnosis and treatment. *Orthop Rev* 1990; 29:139-149.
- Torstensen ET, Bray RC, Wiley JP. Patellar tendinitis: a review of current concepts and treatment. *Clin J Sport Med* 1994; 4:77-82.