TITLE PAGES

Patellar tendinosis (jumper's knee): Histopathology, sonography and MR

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ABSTRACT

Purpose: To determine the histopathology of "jumper's knee" demonstrated with sonography and magnetic resonance imaging (MR).

Materials and Methods: Twenty-four sportspeople scheduled for open tenotomy for "jumper's knee" (28 knees) had sonographic patellar tendon examination and 17 of them also had MR (19 knees). Tissue was obtained in all 28 cases for histological examination. Controls for sonography were 11 age-, height- and weight-matched sportspeople (22 knees) without previous knee symptoms. Control material for histopathology was obtained from cadavers (39 knees). Data analysis was by standard statistical methods.

Results: MR and sonography both revealed an abnormal zone at the proximal patellar tendon attachment. Histopathology revealed mucoid degeneration in 100% of subject cases and 8% of control cadaver cases ($p<0.01$).

Conclusions: "Jumper's knee" is characterised by consistent changes on MR, sonography and histopathology and is appropriately described as patellar tendinosis.

Index terms: Knee, injuries, 4528.253 • Knee, MR, 4541.1214 • Tendons, MR, 458.1214 • ultrasound, technology 41.1298 • Tendinitis, 458.253

TEXT

INTRODUCTION

The clinical entity of "jumper's knee" causes significant morbidity in elite and recreational sportspeople, particularly those playing 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 paper we use the term *jumper's knee* to refer to the clinical syndrome and the term *patellar tendinosis* to refer to histologically proven tendon pathology. We eschew use of the misnomer patellar *tendinitis*.

Sonography and MR imaging can be used to detect abnormalities in the patellar tendon itself [4-12]. This helps 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 primary tendon pathology [15]. The aim of this study was to document the pathological changes in cases of jumper's knee demonstrated with sonography and MR. In addition, we compared the dimensions of abnormalities seen on sonography and MR.

MATERIALS AND METHODS

Subjects

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

Clinical assessment

A single investigator administered a questionnaire to subjects regarding their age, height, weight, sports played, level of competition, side(s) affected and type of treatment received. Severity of pain was assessed with commonly used clinical questions regarding pain at rest and with exercise [2] and also using the 7 point Nirschl Pain Phasing System [21] which was combined with a visual analogue scale [21].

Subjects had their knees examined clinically by two treating physicians and one investigator physician. Sonographic controls had their knees examined clinically by one investigator physician. Tenderness was recorded as present or absent and when present, its location noted.

Imaging

As part of their preoperative workup, all subjects had sonographic studies of both knees performed using high resolution linear array 7.5

or 10 MHz ultrasound transducers (Acoustic Imaging Dornier). This procedure was performed by one of four radiologists. The radiologists took care to examine the tendon with the probe exactly perpendicular to the tendon to avoid a false positive image due to artefactual hypoechogenicity [22].

Eleven sportspeople who did not recall ever having symptoms of anterior knee pain served as a specific sonographic control group. These sportspeople were selected from a large database of asymptomatic athletes [23, 24] to match the subjects for age, height and weight. The 13 subjects undergoing surgery who had unilateral symptoms also had their asymptomatic tendon sonographically examined. These subjects, however, were not included in the "sonographic control group".

Subjects had MR studies acquired using high field (1.5 Tesla) equipment (General Electric, Milwaukee, United States) at one of two centres. Only symptomatic knees were examined. Multiple sagittal and axial sequences were obtained using a 3" surface coil placed directly over the patellar tendon. The following sequences were performed: T1 weighted spin echo, sagittal 2D 400/20 (TR/TE), 3.0mm/0.0mm, fov =12cm, 256*256, two excitations, time=3m.31s; Fast spin echo (FSE) T2 weighted sagittal with fat saturation, 3000/110 ef (TR/TE ef), ETL=8, fov =12cm, 3.0mm/0.0mm, 512*256 matrix, two excitations, time=3m.24s; Gradient echo (GRE) T2*, sagittal 2D, 800/30/70 (TR/TE/deg), fov =12cm, 3.0mm/0.0mm, 256*256 matrix, one and a half excitations, time=5m.10s; Short tau inversion recovery (STIR) sagittal 2D, 4000/17/140 (TR/TE/TI), ETL=6, fov =12cm, 3.0/0.0, 256*192 matrix, two excitations, time=4m.32s T1 axial, 2D, 600/20 (TR/TE), 3.0mm/0.0mm, fov =12cm, 256*256, one and a half excitations, time=3m.59s.

MR studies were initially reported by four MR radiologists who were blinded to the sonography results. These MR reports were rendered irrelevant by the subsequent controlled blinded multi-observer analysis performed by two MR radiologist authors (PMD, BMT). These authors evaluated the MRs of all the subjects that had been studied on a single magnet in our institution (14 tendons). To analyse the four sagittal sequences they conferred and decided on a single sagittal T1 slice which showed the abnormal region to be largest and recorded the slice position (e.g. 2.54 R). For each of the other sagittal sequences (GRE T2*, FSE T2, STIR) they independently selected a slice that corresponded to the slice position they had agreed upon for the T1 sequence. From the sagittal sequences the measurements made were: (a) anteroposterior size of the tendon, (b) anteroposterior dimension of abnormal signal, and (c) craniocaudad (superoinferior) dimension of abnormal signal. To analyse the axial sequence (T1) the radiologists conferred to decide the slice which showed the abnormal region to be largest. They then independently measured (d) the anteroposterior and (e) the mediolateral dimensions of abnormal signal.

Thus, in each patient studied both reporting radiologists made 12 measurements on the sagittal sequences (3 measurements x 4 sequences) and 2 measurements on the axial sequence. Results showed acceptable levels of interobserver agreement with Pearson r values ranging from 0.89 to 0.99 for the 14 sets of measurements.

In order to compare the size of sonographic abnormalities with those seen on MR, the site where the lesion appeared maximal on sonography was identified and measured in the sagittal plane. The craniocaudad (superoinferior) and the anteroposterior dimension were measured independently, prior to MR, by the sonographic radiologists.

Surgery

All subjects had open tenotomy performed. In each case the patellar tendon was examined macroscopically, and the specimen of macroscopically abnormal tissue excised *in-toto*. The proximal portion of the excised tendon (patellar insertion site) was identified by a suture before the specimen was placed in formalin.

Histopathology

All specimens were examined by a single musculoskeletal histopathologist author (FB). The patellar insertion site was marked for recognition microscopically and specimens were serially sectioned longitudinally. Longitudinal sections enabled changes to be recognised throughout the length of the tendon to correspond with sonographic and MR findings.

Specimens were stained with haematoxylin and eosin and examined with polarisation microscopy. Polarisation microscopy is a simple and useful method of assessing continuity and alignment of collagen fibres. Special stains included Alcian Blue (pH 2.5) to detect any increase in ground substance, prussian blue to detect haemosiderin (suggests prior bleeding into the tendon) and immunoperoxidase for smooth muscle actin (SMA) using an Avidin Biotin technique at dilution 1/50 (DAKO) to detect myofibroblastic proliferation and vascular proliferation. There was no attempt to quantify abnormality on histopathological sections.

Cadaver patellar tendon specimens were obtained from sequential cases that met the ethics criteria for the study. These were for cadavers with no known relatives and cadavers resulting from trauma. We examined the specific region of cadaver tendon (patellar insertion) that corresponded with the site of surgery in the subjects.

Statistical analyses were performed using StatViewSE+. The age, height and weight of subjects and the two control groups were compared using ANOVA. Inter-observer correlations for MR measurements were performed using Pearson r correlation coefficients. The differences in the size of the tendon lesion measured by sonography and each MR sequence was determined using repeated measures ANOVA followed by post-hoc Fisher PLSD tests. The incidence of tendon pathology in the subjects and controls was compared using a 2 x 2 Chi-square. Statistical significance was determined to be at the 0.05 level.

The ethics committees of The Royal Melbourne Hospital and the Victorian Institute of Forensic Pathology approved the study. Subjects and sonographic controls gave informed consent.

RESULTS

Subject characteristics

There were 24 (23M, 1F) subjects (28 knees) in whom open patellar tenotomy was performed. The age, height and weight of the subjects and controls are reported in Table I. There was no difference in the mean age, height and weight of the subject group and the sonographic control group. The cadaver control group (18M, 2F), however, was older, shorter and lighter than the subject group (Table I).

Clinical features

Subjects had 14 right and 14 left tendons affected. Subjects reported that the most aggravating sport was basketball (8 subjects), running (6), football codes (3), squash (2), cricket (3), karate and equestrian. Eleven (46%) athletes reported symptoms bilaterally. Of these, five reported previous contralateral tendon surgery and four were undergoing bilateral surgery at the time of this study.

All subjects had pain with exertion. All except four had pain with light activities of daily living (Nirschl Phase VI). Seven had constant ache at rest and pain that disturbed sleep (Nirschl Phase VII). The mean (SE) Nirschl pain phase score was 5.9 (0.2).

The amount of time that jumper's knee kept the subjects from competition varied from nil, where the patient had reduced training but still competed weekly, to greater than 12 months (mean [SE] = 4.6 [(1.2] months). Cortisone injection had been used in 8 cases. Three of those patients felt cortisone made no difference, the remainder reported relief of symptoms ranging from 1 week to 6 months.

The two treating and one investigating physicians all detected tenderness at the junction of the inferior pole of the patella and the tendon corresponding with the site of the lesion on imaging in all subjects. The physicians agreed that jumper's knee was the clinical diagnosis. None of the sportspeople in the specific sonographic control group had significant tenderness at this site.

Imaging results

Maximal dimensions of tendon lesion The maximal craniocaudad (superoinferior) dimension of tendon lesions measured by sonography (longitudinal/sagittal image) was compared with the corresponding image on the each of the 4 sagittal MR sequences. The mean craniocaudad (superoinferior) dimensions of the tendon lesions measured from sonography and MR are graphed in Figure 1. On the GRE T2* sequence the lesion had greater superoinferior (craniocaudad) dimension than it did on the sonographic and other MR images ($p<0.05$) (Figure 1). There were no differences in the mean anteroposterior dimensions of lesions as measured by sonography and the various MR sequences. Also, there were no differences in the mean

anteroposterior dimensions of the tendon as measured by sonography and the various MR sequences.

Sonographic appearance: In 25 of 28 symptomatic knees there was one hypoechoic area in the proximal portion of the patellar tendon on sonography, in the other knees there were two lesions. Sonographic appearances in each case consisted of a focal hypoechoic area (Figure 2) combined with various amounts of swelling of the surrounding tendon. Hyperechoic regions within the tendon, considered to be calcification, were seen in 8 of the 27 tendons. Dystrophic ossification was present on histopathology in all of these cases.

In the specific sonographic control group of sportspeople who had never reported symptoms, there were 4 tendons out of 22 (18%) with a focal hypoechoic region. Of the 13 jumper's knee subjects whose contralateral patellar tendon had never been symptomatic, there were 2 subjects (15%) with a hypoechoic region.

MR appearance: Each athlete had an area of abnormal signal intensity within the tendon at the junction with the patella (Figures 3, 4). Lesions had increased signal intensity relative to the tendon on T1 weighted images (Figures 3a,4) and were markedly hyperintense on the GRE T2* weighted image (Figure 3b), the FSE T2 weighted image (Figure 3c) and the short-tau inversion recovery (STIR) sequence (Figure 3d). Maximal anteroposterior (AP) and craniocaudad (SI) lesion sizes were compared on each of the sagittal sequences. In all cases except one the maximal dimension of the lesion was bigger on the sagittal GRE T2* sequence than on other MR sequences.

The STIR and FSE T2 sequences revealed abnormal signal in the patella (Figure 3c, d) in all but one of the cases in this study. The T1 weighted

signal (Figure 3a) only revealed this abnormality in 7 out of 19 (37%) cases.

Histopathology results

Cadaver cases: Tendons were retrieved from 18 male and 2 female cadavers (39 tendons). They measured approximately 35 x 25 x 10 mm and comprised glistening, stringy white tendinous tissue.

Thirty four (87%) of these tendons appeared entirely normal microscopically. They were composed of dense fibrotendinous tissue (Figure 5a) with a dense, homogenous polarisation pattern (Figure 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 (8%) tendons showed mild changes similar to those noted in all the subject cases (see below). In addition, one tendon showed an increase in ground substance in the paratendinous fibroadipose tissue although the tendon collagen itself was unremarkable and one tendon showed scarring and traumatic neuroma formation consistent with old trauma.

Subject cases: Twenty eight tendons were examined. The specimens measured on average 20 x 15 x 8 mm and lacked the distinctive "stringy" appearance seen in normal tendons.

The histologic 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 homogenous polarisation pattern (Fig 5c&d). In most instances a definite vector marking transition from normal to abnormal could be

seen - the more marked pathological findings being proximally situated (i.e. close to the patellar insertion site).

The abnormality was manifest by a gradual and increasing separation of collagen fibres on polarisation light microscopy with a gradual increase in mucoid ground substance, confirmed by Alcian blue stain. Maximal mucoid change was present proximally at the insertion site where tenocytes, when present, were plump and chondroid in appearance (exaggerated fibrocartilagenous metaplasia) (Figures 6a&b). These changes were accompanied by increasingly conspicuous cells within the tendinous tissue, most of which had a fibroblastic and myofibroblastic appearance.

The cellular proliferation was maximal centrally within the tendinous tissue and was accompanied by a prominence of capillary proliferation (Fig 6c). Polarisation microscopy showed a tendency to discontinuity of collagen fibres in this area (Figure 5d). In two cases, foci of ossification ('tug 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 prior to the area of maximal mucoid degeneration/fibrocartilaginous metaplasia present at the area of insertion into the patella (Figure 7).

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

Chi-square test revealed that the proportion of subjects with changes of mucoid degeneration in their patellar tendon (28/28=100%) was greater than the proportion of controls with similar changes (3/39=8%) ($p<0.001$).

DISCUSSION

Imaging aspects

In our study the GRE T2* sequence consistently revealed a significantly larger area of high signal intensity than did the T1 and FSE T2 sequences (Figures 1, 2a-c). Increased tendon high signal intensity on GRE T2* can arise from the 'magic angle' phenomenon[25]. However, this does not appear to explain our finding for several reasons. Firstly, in our MR coil arrangement the proximal patellar tendon courses at less than 30° to the constant magnetic induction field (Figure 2). Secondly, the region of increased signal intensity only affects the posterior part of the tendon even when both anterior and posterior portions of the tendon run parallel. Thirdly, the abnormal signal 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 [26]. Fourthly, the abnormal signal extends from the patellar insertion to the mid tendon despite changes in the angle of the tendon relative to the magnetic field. Fifthly, our subjects had sonographic demonstration of abnormal signal on sonography. We do, however, note the presence of the 'magic angle' phenomenon at the tibial insertion of the patellar where the tendon courses at 55° to the magnetic field in some subjects. These distal regions of abnormal signal were not included in our analysis.

Areas of increased GRE T2* signal have been reported at the proximal and distal end of the patellar tendon in asymptomatic individuals independent of 'magic angle' findings [27]. However, the region of abnormal GRE T2* signal in our symptomatic patients extended far beyond the patellar attachment site described by Reiff and colleagues.

We postulate that the larger area of increased signal on GRE T2* weighted sequence may reflect increased sensitivity to minor damage in tendon. If this were the case, then the GRE T2* weighted sequence could prove to be an effective method for monitoring elite high-risk athletes, e.g. NBA basketballers, during a period of intense training. In this scenario, asymptomatic 'tendon strain' detected on the GRE T2* sequence might be a precursor to frank tendon damage. McLoughlin has also noted this phenomenon of increased GRE T2* signal relative to FSE T2 signal and speculated as to its histopathological significance[12].

The STIR and FSE T2 weighted sequences revealed abnormal signal in the patella (Figure 2c, d) in most of the athletes 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.

Histopathological aspects

We report a definite craniocaudad (superoinferior) vector in the pathology of jumper's knee with more marked findings at the proximal (patellar) end of the tendon. Because our surgical specimens were labelled to permit orientation, we were able to confidently relate abnormal histopathology with sonographic and MR appearances. Measures of severity of the condition, i.e., abnormality under light microscopy (Figure 5) and amount of abnormal ground substance

between the tenocytes (Figure 6) point to greater mucoid degeneration at the patellar insertion decreasing distally toward the tibia.

Inflammatory cells were totally absent at the regions of greatest mucoid degeneration (Figure 6) and also at the margins of the specimens, where the mucoid degeneration was least. Even in two cases where patients were operated on after only four 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 [28, 29] or whether it results from tearing of collagen and secondary mucoid degeneration, is not known. However, histologic changes of ongoing reparative fibrosis are evident in each case in this study (fibroblastic, myofibroblastic and vascular proliferation) and this possibly reflects repeated minor trauma.

Surgical treatment for jumper's knee is recommended only "if a prolonged and well-supervised conservative treatment program fails" [30]. A variety of procedures, differing in the relative amount of bone surgery involved, has been reported [1, 30]. There are no reports of the rationale for these procedures, but presumably excision of degenerative, painful tissue is designed to promote healing [31].

Another treatment of jumper's knee in clinical practice is cortisone injection [32] although some authors discourage its use [30]. In this study of patients in whom conservative management had failed, tendons that had been injected and those that had not were indistinguishable by both radiological and histological tests. In these cases cortisone did not permanently affect the tendon, or did not reach

the site eventually excised. Well-designed clinical and basic studies of the effect of cortisone injection in tendon disorders are required.

In summary our data reveal that sportspeople with severe cases of the clinical condition known as jumper's knee, confirmed by sonography and MR, are clearly suffering a tendinosis. This tendinosis is not a normal feature of either aged, or younger, cadaver patellar tendons. Since jumper's knee has been shown to be patellar *tendinosis*, we recommend that treatment protocols should address this pathology rather than a supposed "inflammatory" tendinitis.

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TABLES

Table I. Characteristics of subject and control groups. Mean (SE)

	Jumper's knee subjects	Sonographic control group	Cadavers
Number of individuals	24 (23M, 1F)	11 (9M, 2F)	20 (18M, 2F)
No. of patellar tendons studied	28 (27M, 1F)	22 (18M, 4F)	39 (35M, 4F)
Age (yrs)	30.9 (1.7)	31.5 (1.1)	60.8 (4.4) *
Height (cm)	180.0 (1.8)	185.5 (2.1)	169 (2.5) *
Weight (kg)	81.2 (2.3)	83.4 (3.7)	68.8 (4.6) *
Symptoms (mths)	35.4 (8.2)	0 (0)	unknown

*$p<0.05$, Jumper's knee subjects versus cadavers

CAPTIONS

Figure 1. Lengths (mean, SE) of the maximal sonographic hypoechoic region and MR abnormal signal in the craniocaudad direction (T1, FSE T2 & GRE T2* sequences) in the craniocaudad (superoinferior) direction.

Figure 2. Sagittal (longitudinal) sonographic scan of the patellar tendon of an 18 year old man, showing focal hypoechoic region (arrowed) at the deep portion of the tendon adjacent to the lower pole of the patella.

Figure 3. Sagittal MR images of an 18 year old man with jumper's knee.

(a) T1 weighted SE image (400/20) showing increased signal (arrowed) relative to the tendon

(b) GRE T2* image (800/30,with flip angle of 70°) showing marked hyperintense signal in the tendon at the attachment to the patella and an irregular poorly defined posterior margin of the patella (arrowed).

(c) T2-weighted FSE image (3000/110ef) with marked increased signal (straight arrow) in the tendon and increased signal in the lower end of the patella itself (curved arrow)

(d) Short tau inversion recovery (STIR) image (4000/17/140) with increased signal in the tendon (straight arrow) and the patella (curved arrow).

Figure 4. Axial T1 weighted image (600/20) of an 18 year old male patient with jumper's knee. This sequence localises the lesion in the axial plane and in this case the increased signal (arrow) arises from the medial aspect of the tendon.

Figure 5. Normal tendon histology using (a) H&E stain at low power (x40). The tendon appears smooth and tenocytes and blood vessels are

inconspicuous, (b) Polarisation microscopy (x40) which shows tightly cohesive continuous collagen bundles with dense homogenous polarisation pattern. Normal "crimping" is evident. The corresponding tissue in an 18 year old male with jumper's knee is shown in (c) H&E stain at low power (x40) of abnormal tendon. There is loss of the normal smooth tendon appearance, an increased number of fibroblasts & myofibroblasts and blood vessels are prominent and (d) Polarisation microscopy (x20) demonstrating loss of the normal dense homogenous polarisation pattern, separation of collagen fibres and focal discontinuity of fibres.

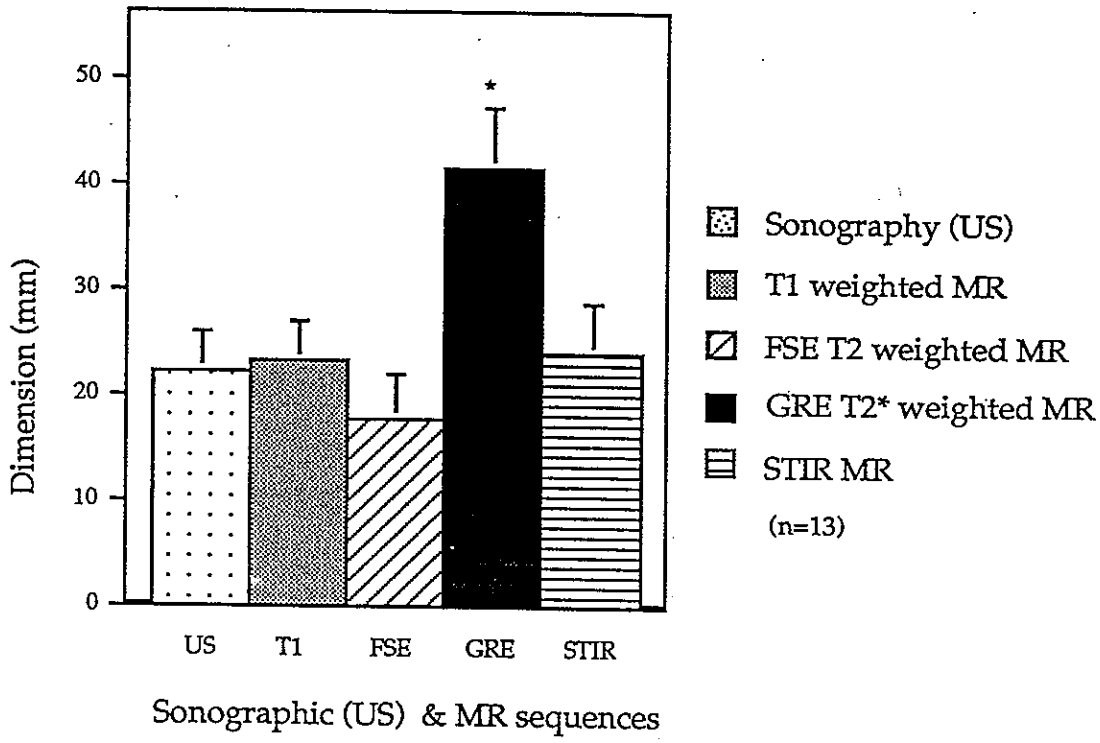
Figure 6. Histopathological changes found in an 18 year old male with jumper's knee. (a) H & E stain at medium power (x100) close to the patellar insertion site showing separation of collagen. (The intervening spaces can be shown to have an increased mucoid ground substance by Alcian Blue stain.) Tenocytes are plumper and more chondroid in appearance.

(b) H & E stain at high power (x200) in the same patient showing plumper tenocytes with chondroid appearance.

(c) H & E stain at higher power (x200) showing marked increase in cellularity in the pathological region of the tendon specimen.

Figure 7. In an 18 year old male with jumper's knee, immunoperoxidase stain for smooth muscle actin at low power (x40) of the area close to the patellar insertion site reveals discontinuity of vascular proliferation and decreased myofibroblastic proliferation (bottom right-straight arrows) just prior to the area of maximal mucoid degeneration (top right-curved arrow).

Figure 1. Lengths (mean, SE) of the maximal sonographic hypoechoic region and MR abnormal signal (T1, FSE T2 & GRE* T2 sequences) in the craniocaudad (superoinferior) direction



* $p<0.05$ (GRE T2* versus sonography and the other MR sequences)

Figure 2. Sagittal (longitudinal) sonographic scan of the patellar tendon of an 18 year old man, showing focal hypoechoic region (arrowed) at the deep portion of the tendon adjacent to the lower pole of the patella.

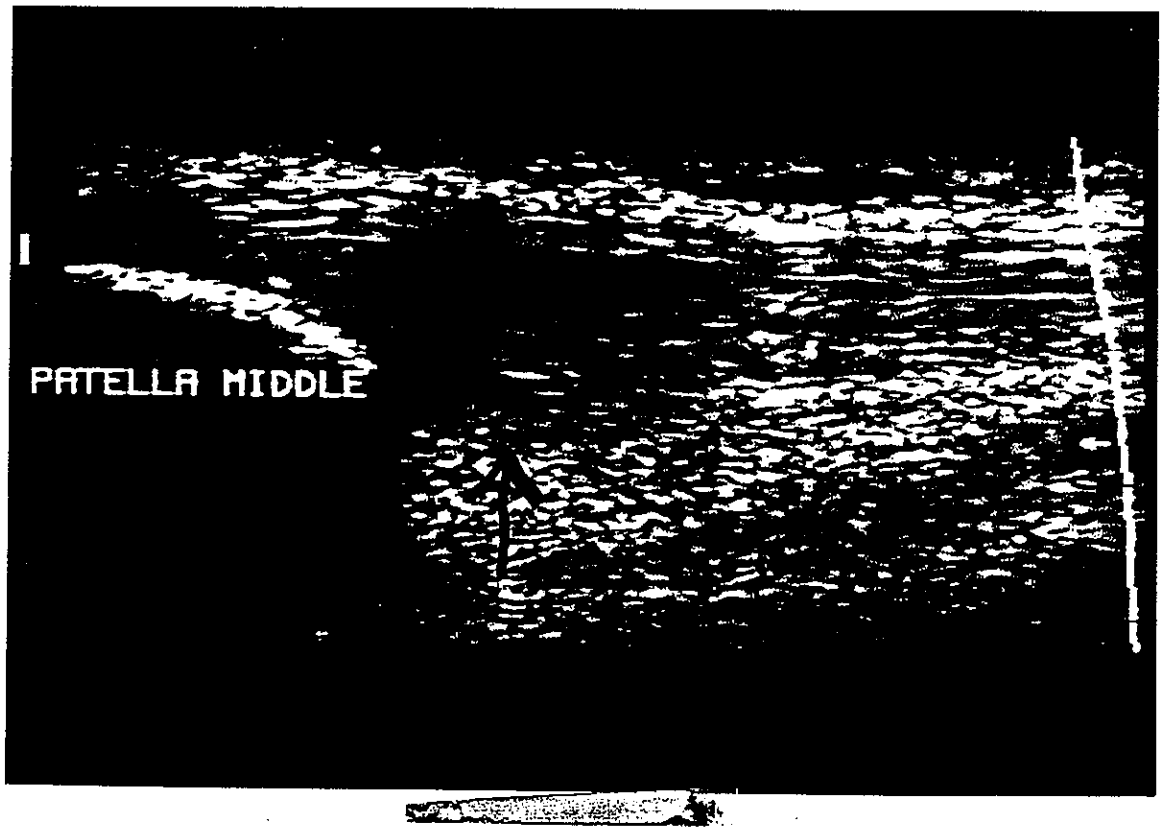


Figure 3. Sagittal MR images of an 18 year old man with jumper's knee. (a) T1 weighted SE image (400/20) showing increased signal (arrowed) relative to the tendon

(3b) GRE T2* image (800/30,with flip angle of 70°) showing marked hyperintense signal in the tendon at the attachment to the patella and an irregular poorly defined posterior margin of the patella (arrowed).

(3c) T2-weighted FSE image (3000/110ef) with marked increased signal (straight arrow) in the tendon and increased signal in the lower end of the patella itself (curved arrow)

3 (d) Short tau inversion recovery (STIR) image (4000/17/140) with increased signal in the tendon (straight arrow) and the patella (curved arrow).

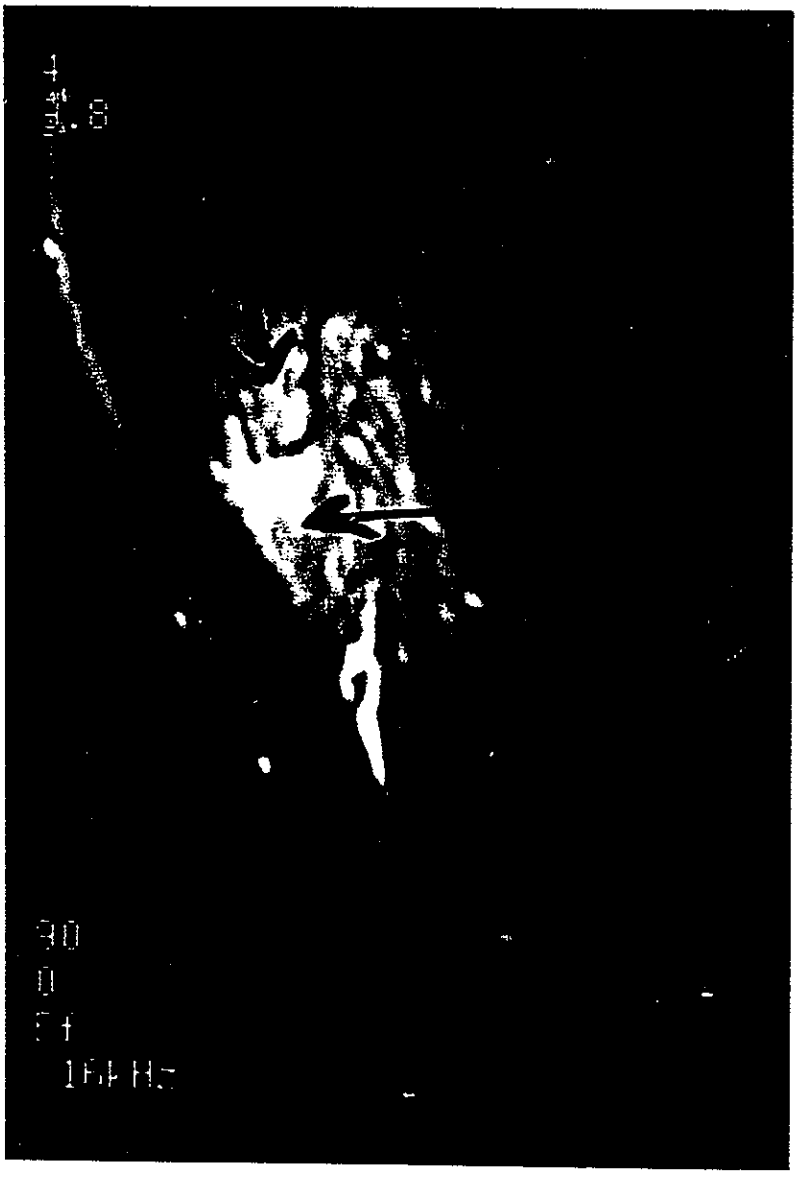


Figure 4. Axial T1 weighted image (600/20) of an 18 year old male patient with jumper's knee. This sequence localises the lesion in the axial plane and in this case the increased signal (arrow) arises from the medial aspect of the tendon.

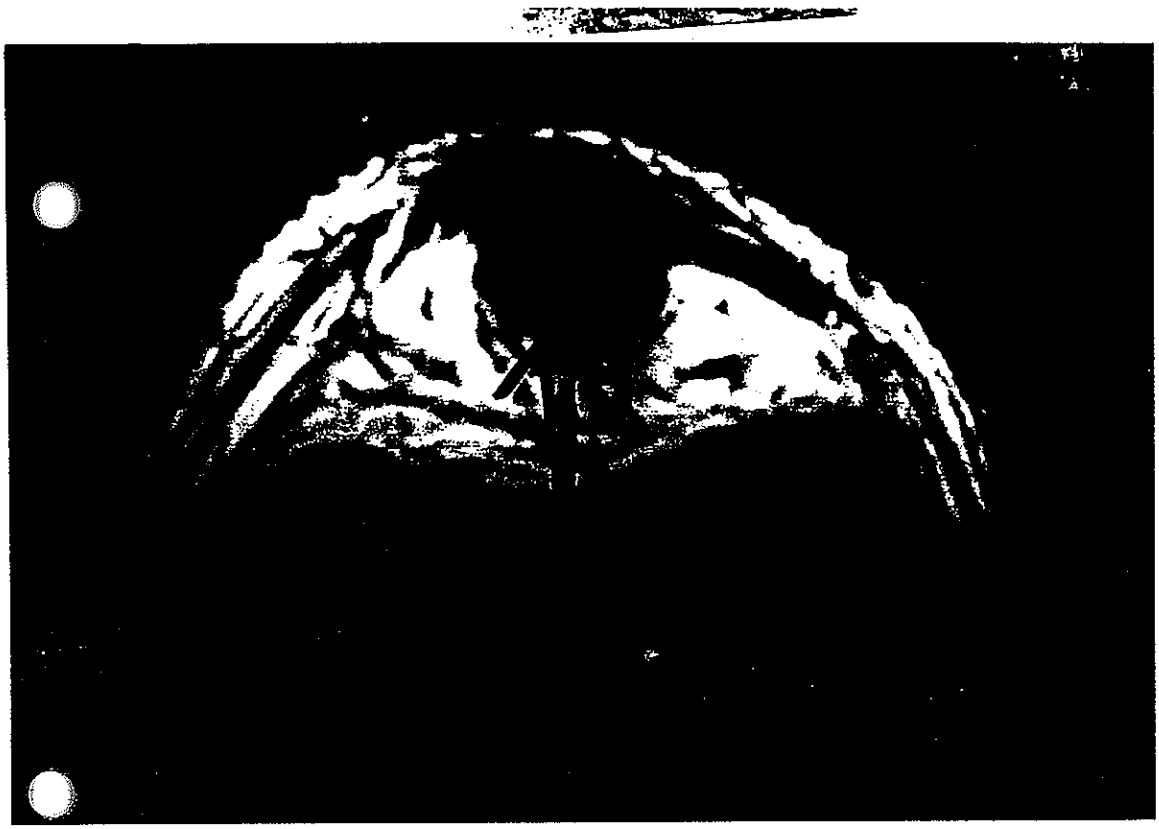
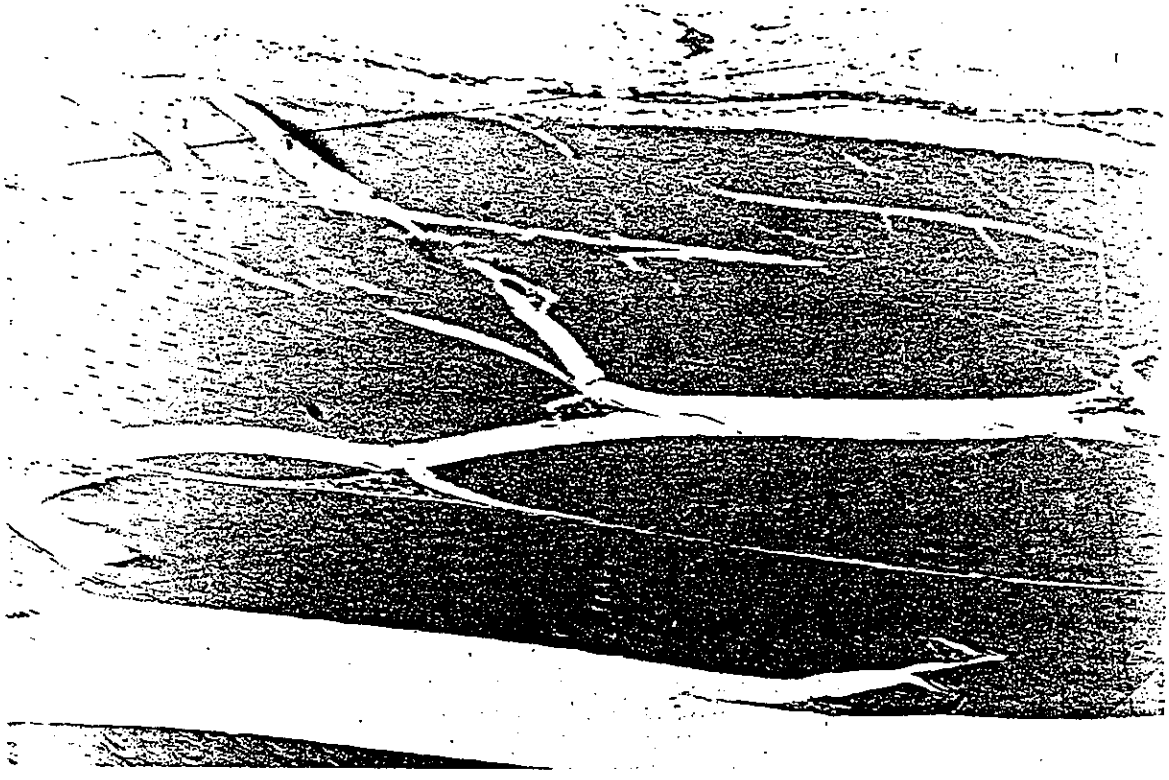


Figure 5. Normal tendon histology using (a) H&E stain at low power (x40). The tendon appears smooth and tenocytes and blood vessels are inconspicuous,



5 (b) Polarisation microscopy (x40) which shows tightly cohesive continuous collagen bundles with dense homogenous polarisation pattern. Normal "crimping" is evident.

The corresponding tissue in an 18 year old male with jumper's knee is shown in (5c) H&E stain at low power (x40) of abnormal tendon. There is loss of the normal smooth tendon appearance, an increased number of fibroblasts & myofibroblasts and blood vessels are prominent and

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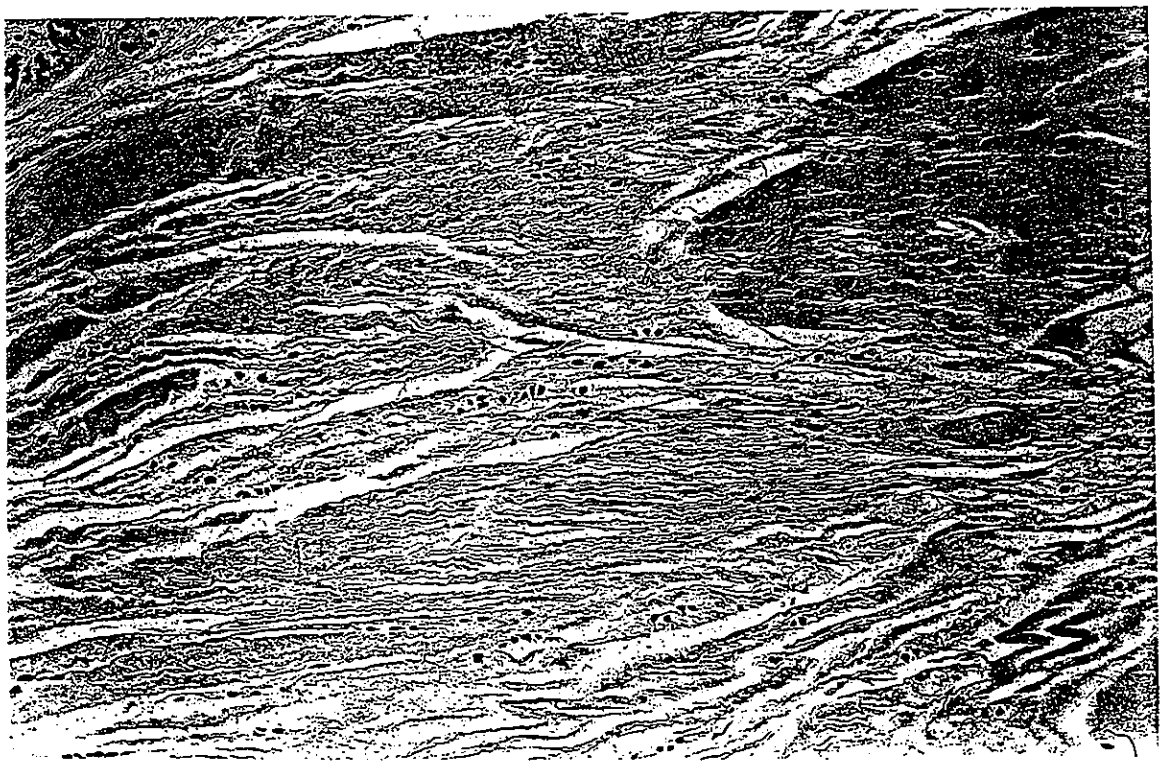
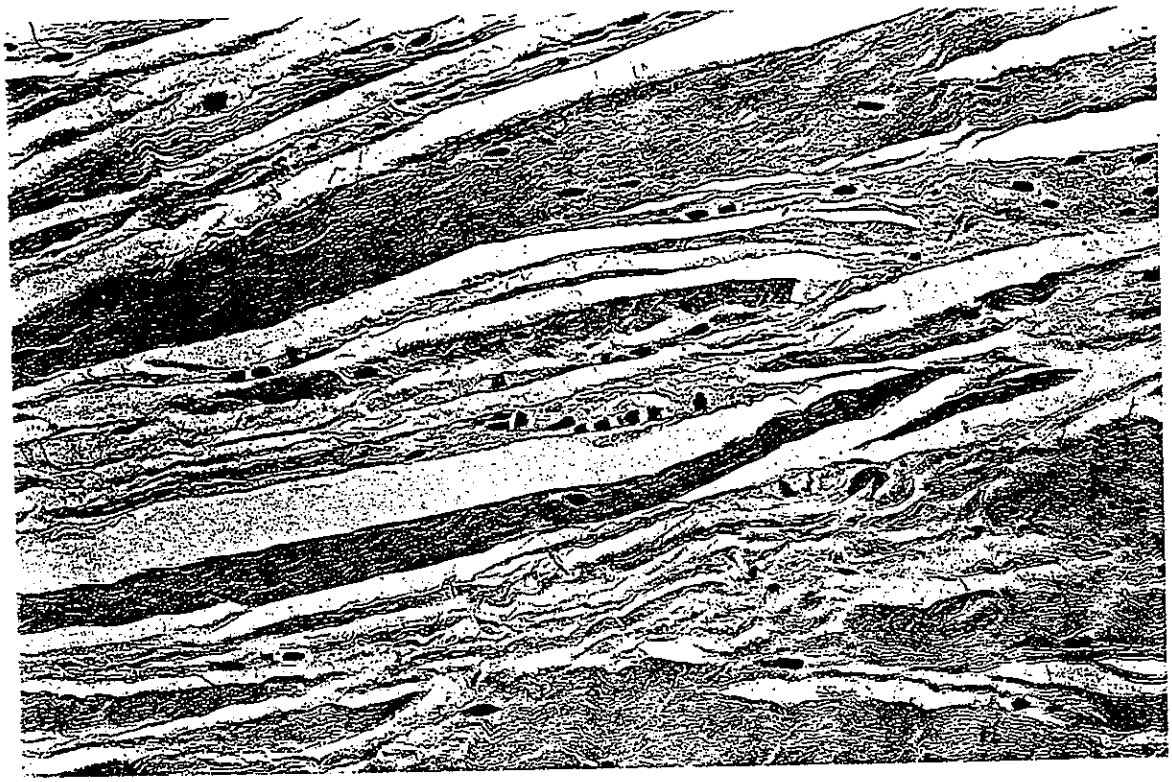
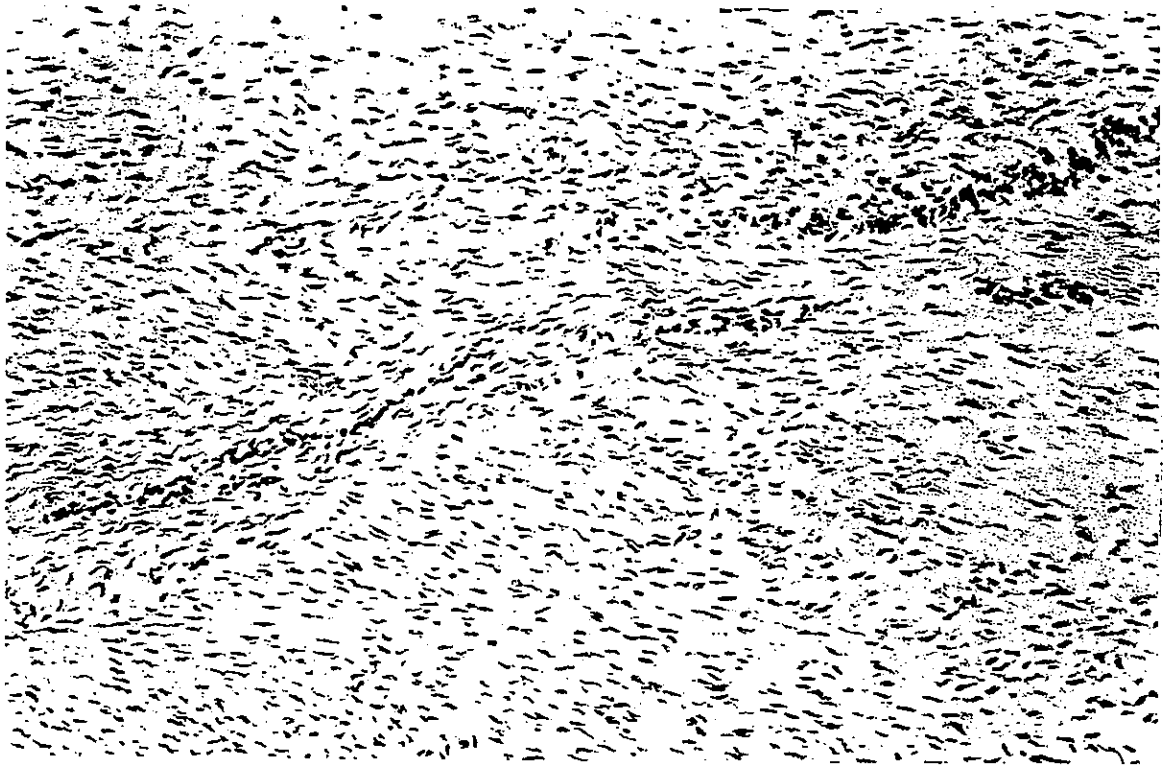


6(b) H & E stain at high power (x200) in the same patient showing plumper tenocytes with chondroid appearance.



6(c) H & E stain at higher power (x200) showing marked increase in cellularity in the pathological region of the tendon specimen.



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