The radiological and MRI appearances of 24 knees with patellar tendonitis resistant to conservative therapy were analysed to identify the characteristic MRI appearance and to determine if the patellar morphology was abnormal. A significant thickening of the tendon was found in all cases; this was a more reliable diagnostic feature than a high signal within the superior posterior and central aspect of the tendon at its proximal attachment.

The site of the lesion shown by MRI is more compatible with impingement of the inferior pole of the patella against the patellar tendon than a stress overload of the tendon. There were no significant differences in the length of the patella, inferior pole or length of the articular surface when the patellar morphology was compared with that of a matched control group.

Patellar tendonitis is a common condition affecting the proximal attachment of the patellar ligament to the inferior pole of the patella, and is found particularly in those engaged in sporting activities such as basketball, volleyball and triple, long or high-jumping. It has been called ‘jumper’s or basketballer’s knee’ (Blazina et al 1973).

It presents with pain, swelling and localised tenderness of the central portion of the proximal attachment of the patellar tendon adjacent to the inferior pole of the patella (Stanish and Curwin 1984) and may result in functional limitation or, in severe cases, rupture of the tendon (Blazina et al 1973). The condition has been classified into four stages (Blazina et al 1973; Table I). It must be differentiated from similar disorders such as Sinding-Larsen-Johannson’s disease (Sinding-Larsen 1921; Johansson 1922), Osgood-Schlatter’s disease (Osgood 1903; Schlatter 1903), chondromalacia patella, prepatellar or infrapatellar bursitis, synovial plicae or fat-pad entrapment (King et al 1990; Johnson, Eastwood and Witherow 1993). Plain radiography is usually unhelpful in diagnosis and assessment, particularly early in the disease (Roels et al 1978). Later radiological signs include non-specific abnormalities such as soft-tissue swelling, periosteal reaction at the anterior surface of the patella (Khan and Wilson 1987), calcification in the patellar tendon (Martens et al 1982) and elongation of the inferior pole of the patella (Blazina et al 1973; Roels et al 1978). CT can detect intraligamentous abnormalities, but Mourad, King and Guggiana (1988) have shown that ultrasound can be as effective in detecting patellar tendonitis, with the advantages of being relatively inexpensive, readily available and avoiding ionising radiation. It is generally accepted as the preferred imaging technique (Fritschy and DeGautard 1988; King et al 1990), but is dependent on the skill of the operator to show thickening of the tendon and a characteristic heterogeneous echogenicity; chronic cases show a poorly defined outline (Fritschy and De Gautard 1988; King et al 1990). Isotope bone scanning can detect focal increased activity at the inferior pole of the patella or tibial tuberosity (Khan and Wilson 1987), but cannot examine the patellar tendon specifically. MRI gives excellent soft-tissue contrast, anatomical definition and additional information about the other bony and ligamentous structures around the knee (Bodne et al 1988; Davies et al 1991; El-Khoury et al 1992). The MRI signs of patellar tendonitis include an increased

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>1</td>
<td>Pain only after sports</td>
</tr>
<tr>
<td>2</td>
<td>Pain at the beginning of sports disappearing after a warm-up but reappearing with fatigue</td>
</tr>
<tr>
<td>3</td>
<td>Constant pain at rest and with activity</td>
</tr>
<tr>
<td>4</td>
<td>Complete rupture of the patellar tendon</td>
</tr>
</tbody>
</table>

**Table I. Classification of patellar tendonitis according to Blazina et al (1973)**

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intraligamentous signal intensity on both short and long TE sequences and indistinct ligamentous margins (Bodne et al. 1988; El-Khoury et al. 1992). The dynamic sequencing of static MR scans has been found to be useful in the assessment of patellofemoral instability but has not yet been applied to patellar tendonitis.

We have performed a prospective, controlled study to define and quantify the characteristic MRI appearance and to analyse the patellofemoral morphology in patellar tendonitis using plain radiography, MRI and dynamic MRI along with several new imaging sequences with the knee in extension and in flexion.

PATIENTS AND METHODS

We included all patients who had been diagnosed as having chronic patellar tendonitis but failed to respond to treatment for six weeks by rest, anti-inflammatory medication, stretching exercises and physiotherapy. There were 18 men and one woman (24 knees); five men had bilateral symptoms. Their average age was 32.1 years (19 to 52) and their average height 187 cm (168 to 198). At the time of presentation they had had symptoms for an average duration of 15 months (2 to 60). Four patients had had a steroid injection (methylprednisolone acetate 80 mg). Ten patients had grade-3 symptoms, eight grade-2 and six grade-1 (Blazina et al. 1973; Table I).

Conventional radiography included anteroposterior, lateral in extension, lateral in 30° flexion and skyline patellar views in 30° of knee flexion. We performed MRI with a Siemens Magnetom Impact (1.0 T) superconducting MRI scanner (Siemens House, Bracknell, UK). All scans were obtained using a dedicated knee coil with fields of view

| Table II. Classification of the signal intensity (axial images) of the ligament |
|---------------------------------|-----------------------------------------------------------|
| 0                               | Normal tendon appearances                                 |
| 1                               | Increased high-signal intensity occupying more than 50% of the axial cross-sectional tendon width |
| 2                               | Increased signal intensity in less than 25% of the axial cross-sectional tendon width |
| 3                               | Increased high-signal intensity occupying more than 50% of the axial cross-sectional tendon width |
ranging from 160 to 210 mm. Five sequences were obtained:
1) Fast spin echo (FSE) dual echo sagittal (TR/TE, 4250/15, 105) (Table II).
2) Fast short TI inversion recovery (STIR) sagittal (TR/TE/ T1, 5300/60/100) (Fig. 1).
3) Either axial gradient echo T2-weighted transverse (TR/ TE 620/18; flip angle 30°) or axial FSE T2-weighted trans- verse (TR/TE, 4250, 90) (Fig. 2).
4) Spin-echo T1-weighted sagittal in full extension and flex- ion (60° due to the constraints of the scanner) (Fig. 3).
5) ‘Pseudo-dynamic’ flexion/extension sagittals, flash 2-D (TR/TE, 45/6, flip angle 90°). With the patient lying later- ally on the affected side, midline sagittal images were acquired in four positions ranging from full extension to maximum allowed flexion (approximately 60°). The static images were then displayed dynamically as a cineloop.

Patellar position was determined according to Insall and Salvati (1971). The greatest length of the patella was measured from the proximal to distal pole and the length of the patellar ligament from its origin on the lower pole of the patella to its insertion on the tibial tuberosity. The ratio of the length of the patellar ligament to patellar length was calculated. The femoral trochlear sulcus angle was measured on the plain skyline views of the patella (Merchant et al 1974) and from axial MRI. On the MR scans we measured the length of the non-articular inferior pole of the patella (P) and the length of the articular surface of the patella (AS) and expressed them as a ratio (P:AS ratio) (Fig. 4).

The patellar ligament was divided notionally along its length into thirds and for each third the midpoint AP thick-
pathology with no clinical evidence of patellar tendonitis or other patellofemoral problems. MRI in these patients was carried out in the same way as for those with patellar tendonitis.

Non-parametric statistical analysis used the Kruskal-Wallis and Spearman rank correlation tests using SAS version 6.07 on a UNIX computer. Parametric contingency table analysis and t-test statistics were undertaken using Statview 512 on a Macintosh IIci microcomputer.

RESULTS

Clinical. When scored on the International Knee Documentation Committee (IKDC) score two patients were nearly normal, five abnormal and 11 severely abnormal. On examination all had tenderness in the superior central part of the patellar tendon around the insertion to the lower pole.

Radiography. Plain radiography showed no periosteal reaction, patellar fragmentation, proximal ligament calcification or patellofemoral degeneration. There was fragmentation of the insertion of the ligament into the tibial tuberosity indicative of previous Osgood-Schlatter’s disease in both knees of one patient. Measurement of the Insall-Salvati index on lateral radiographs showed a mean patellar length of 50.3 mm (43 to 56), a mean length of the patellar tendon of 57.4 mm (45 to 72) and a mean patellar ratio of 1.15 (0.78 to 1.5). Six of the 24 knees (25%) had a patellar index of greater than the normal maximum of 1.2 (normal control mean 1.02; Insall and Salvati 1971; p < 0.001). On the skyline radiographs there was no evidence of patellofemoral subluxation or tilt. The mean congruence angle was -3.8° (+8 to -18) (normal control mean -6°; Merchant et al 1974). The mean femoral trochlear sulcus angle was 137° (123 to 148) (normal control mean 138°; Merchant et al 1974). No patient had a sulcus angle greater than the normal maximum of 150° (normal control mean 138°; Brattström 1964).

MRI. We used the midline patellar sagittal MR image to measure the dimensions of the inferior pole. The mean length of the non-articular inferior pole was 10.2 mm (2 to 22). The corresponding length of the articular surface was 36.5 mm (30 to 45). Thus the P:AS ratio was 0.28 (0.07 to 0.55). The corresponding measurements in the control group were a pole length of 10.2 mm (range 6 to 17; p = 0.97), articular length of 33.38 mm (range 31 to 45; p <0.01) and a P:AS ratio of 0.30 (range 0.22 to 0.5; p = 0.36).

An increased MRI signal within the proximal patellar ligament was detected in 19 of the 24 knees (79%); on the axial images it occupied the superior posterior and central aspect of the tendon in all cases (Fig. 5), but was not identified in any of the control cases. The increased signal extended into the lateral part of the tendon in five (26%) and into the medial aspect in five (26%). In nine knees there was a grade-1 signal and in ten a grade-2 signal showing a high intensity signal occupying between 25% and 50% of the tendon width. No grade-3 signals (occupying more than 50% of the tendon) were seen. The longitudinal extent of the increased signal measured from the axial images was less than 10 mm in one of the 19 abnormal scans (5%), between 10 and 20 mm in 11 (58%) and greater than 20 mm in seven (37%).

We compared the thickness of the patellar tendon with that of a matched normal group of 24 knees. In the patients the mean proximal thickness of the tendon was 8.5 mm (5 to 15) compared with 5.5 mm (range 4 to 7; p < 0.05) in the control group. In the middle third the mean thickness was 6.4 mm (4 to 12) compared with 5.4 mm (range 5 to 7; p < 0.001) in the control group, and the distal third 6.5 mm (4 to 10) compared with 6.25 mm (range 5 to 7; p < 0.01) in the
control group. In all 24 knees the thickness of the tendon in its proximal third on the midline scan was increased above the normal 3.7 mm reported by El-Khoury et al (1992).

In four of the knees (17%) with patellar tendonitis MRI did not show an area of increased signal although all demonstrated an increased thickness of the proximal patellar tendon (mean 5.4 mm). One of these scans also showed peritendinitis and another quadriceps tendonitis. Three of the MR scans (13%) demonstrated a previously unreported appearance of swelling, inflammation and oedema spreading upwards over the periosteum of the anterior inferior surface of the patella. In one case an asymptomatic degenerative medial meniscus was detected, and in another a medial synovial plica.

Pseudodynamic flexion-extension imaging did not reveal any abnormality which was not apparent on the static images other than kinking of the superior part of one tendon during flexion. The technique was poorly reproducible in most patients due to minor movements of the patellar tendon out of the plane of the scan.

DISCUSSION

Patellar tendonitis usually refers to an inflammatory condition of the infrapatellar portion of the patellar ligament, but has been used loosely by several authors to include inflammation of any part of the patellar ligament and even inflammation of the suprapatellar quadriceps tendon (Blazina et al 1973; Khan and Wilson 1987). Blazina et al (1973) popularised the term ‘jumper’s knee’ and patellar tendonitis usually affects athletes whose sport involves repetitive explosive extension or eccentric flexion of the knee. The term is a misnomer because there is no anatomical structure named the patellar tendon; better terms may be ‘incomplete patellar ligament tear’ or ‘chronic microtearing of the patellar ligament’ (El-Khoury et al 1992). They are anatomically correct, and also suggest the theoretical pathophysiology of the condition.

Histological examination of specimens with patellar tendinitis shows chronic inflammation, mucoid degeneration, fibrinoid necrosis and synovial proliferation within the deep aspect of the patellar insertion (Martens et al 1982; Ferretti et al 1983; Stanish and Curwin 1984; Bodne et al 1988; Davies et al 1991). The cause is generally considered to be chronic stress overload resulting in microscopic tears of the tendon and degeneration. Biomechanical forces within the patellar ligament may reach 8000 N when landing from a jump, up to 9000 N during fast running and 14 500 N during competitive weight-lifting, compared with a force of 500 N experienced in level walking (Zernicke, Garhammer and Jobe 1977; Stanish and Curwin 1984; Stanish, Rubinovich and Curwin 1986).

The suggested pathogenesis of stress overload and tension failure, however, does not explain why this is related solely to the deep aspect of the central portion of the tendon, as shown in our study. Tension failure would be expected to affect the insertion throughout its width, and the superficial aspect more than the deep surface (Fig. 5). The rationale of surgical treatment which releases the central portion of the tendon from the inferior pole of the patella is also debatable, since it must increase the stress on the residual intact part of the tendon. Good results have been reported, however, after such treatment (Roels et al 1978; Martens et al 1982), which suggests that perhaps the usual theory of pathogenesis may be flawed.

Plain radiography is of minimal value in confirming the presence of tendonitis (Blazina et al 1973; Roels et al 1978; Martens et al 1982; Khan and Wilson 1987), but our study of the patellofemoral morphology has shown that the mean length of the patella was greater than normal in our patients, although their greater average height may account for the difference.

MRI results showed that the femoral sulcus angle was normal and that there was no patellofemoral tilt or subluxation. The Insall-Salvati patellar height index was increased in 25% of our cases, but the length of the non-articular inferior pole was not abnormal. An elongated inferior pole has been reported in patellar tendonitis (Roels et al 1978), but may be a sporadic finding.

We found a significant increased thickness of the upper third of the patellar tendon in all cases compared with our control group and with the normal group described by El-Khoury et al (1992). This was most obvious in the superior-central region and can reliably be used to confirm the diagnosis. The characteristic inflammatory lesion in the superior, posterior and central aspect of the patellar tendon was closely related to the inferior pole of the patella and never involved more than 50% of the width of the tendon. There were no such lesions or increased thickness in our control group. The inflammatory lesion (79%) was a less reliable indicator than the increased thickness, probably because MRI showed a normal signal intensity but some thickening in chronic cases with no acute inflammatory reaction.

In three knees (13%) MRI showed previously unreported extensive inflammatory changes extending upwards to cover the anterior surface of the inferior aspect of the patella. These changes may be related to the periosteal reaction at the anterior surface of the patella which is sometimes seen on plain radiography (Khan and Wilson 1987), possibly the result of spreading periosteal inflammation. Pseudodynamic MRI proved difficult to perform and did not yield any significant additional relevant information.

Laduron et al (1993) has recently suggested that patellar tendonitis may result from an impingement or ‘conflict’ between the deep fibres of the proximal patellar tendon and the lateral part of the femoral trochlea when the knee is fully extended. Analysis of our MR images showed suspicion of this impingement in only two of the 24 knees and we therefore discount this theory.

Our results refute some of the other theories of the pathogenesis of patellar tendonitis; we suggest that the cause is not stress overload but impingement of the inferior pole of the patella on the patellar tendon in flexion. Most of the
The patellar tendon does not insert into the inferior pole of the patella but continues over the anterior surface of the patella to be continuous with the quadriceps tendon (Fig. 5). In deep knee flexion the central portion of the patellar ligament is displaced around the prominent inferior pole of the tendon (Fig. 6). Such impingement could explain the characteristic site of the lesion, the normal patellofemoral joint morphology, and also why surgical release of the tendon or partial excision of the inferior pole of the patella are beneficial (Martens et al 1982). It also explains the site of the degenerative lesion (Figs 2, 5 and 6). This theory is not incompatible with the histological features of intratendinous fibrinoid necrosis, mucoid degeneration and chronic inflammation as described by Martens et al (1982).

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No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References


