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Patellar Tendinopathy and Patellar Tendon Rupture

Karim M. Khan, Jill L. Cook, and Nicola Maffulli

Introduction

Patellar tendon injuries constitute a significant problem in a wide variety of sports [1–4]. Despite the morbidity associated with patellar tendinopathy, clinical management remains largely anecdotal [5] as there have few well-designed treatment studies. This chapter will update the reader on management of 1) the patient with overuse patellar tendinopathy, and 2) the patient unfortunate enough to suffer the less common, but debilitating, condition of patellar tendon rupture.

Typical Clinical Scenario—Patellar Tendinopathy

In the patient with patellar tendinopathy, knee pain may arise insidiously. Those patients who recall when the pain began report that it started during one heavy training session or, less commonly, from one specific jump. In addition, they often remember a specific activity that seemed to make the pain worse. Pain is usually well localized to a small area over the anterior aspect of the knee region, and many patients have noticed tenderness at the inferior pole of the patella before they present for a medical examination.

Early in the clinical course, the patient's knee pain and discomfort may ease completely while exercising. In this case, the athlete often disregards the injury and does not seek treatment. With time and continued activity, however, pain worsens and limits sporting performance. Eventually, pain can develop during activities of daily living and can even be present at rest. Examination reveals tenderness at the junction of the patella and the patellar tendon. This clinical scenario has a number of names, including jumper's knee, patellar tendinopathy, patellar tendinosis, or patellar tendinitis. The preferred diagnostic term is patellar tendinopathy [6]. Palpation of the tendon attachment at the inferior pole of the patella has been the classic physical examination technique for detecting patellar tendinopathy, but mild tenderness at this site is not unusual in a normal tendon. Only moderate and severe tenderness is significantly associated with tendon abnormality as defined by ultrasonography. Thus, we suggest that mild patellar tendon tenderness should not be overinterpreted, and may be a normal finding in active athletes.

Patients with chronic symptoms may exhibit quadriceps wasting, most notably in the vastus medialis obliquus. Thigh circumference may be diminished, and calf muscle atrophy may be present. Testing the functional strength of the quadriceps may be done by comparing the ease with which the patient can perform 15 one-legged stepdowns on each leg. The athlete bends at the knee and then straightens again without letting the other foot touch the floor. Work capacity of the calf is assessed by asking the patient to do single-leg heel raises. Jumping athletes should be able to do at least 40 raises. It is important to monitor both the onset of fatigue and the quality of movement (e.g. control, as measured by wobbling), as either can be affected in the symptomatic limb.

In general, the clinical features of patellar tendinopathy are distinctive [7], and some authors have suggested that the diagnosis is straightforward [8]. Our clinical impression is that this is true in about three-fourths of the cases of patellar tendinopathy, but that in some cases patellofemoral joint syndrome and patellar tendinopathy may be difficult to differentiate, or the conditions may coexist.

Imaging Appearances

Magnetic resonance (MR) imaging and ultrasound (US) are the investigations of choice in the jumping athlete with knee pain (see Figures 18-1 and 18-2). Here we summarize the typical findings in a patient with patellar tendinopathy and we discuss the clinical utility of the imaging modalities.

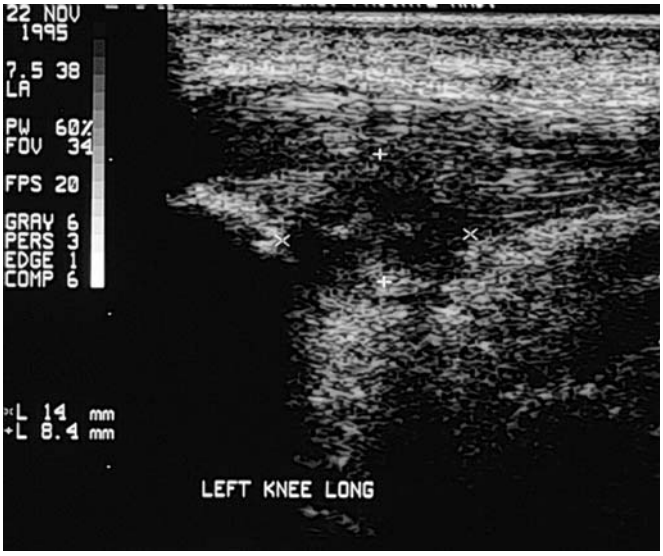

FIGURE 18-1. Longitudinal ultrasound scanning of a 25 year old basketball player with patellar tendinopathy. Note the area of hypoechogenicity.

MR Imaging

The abnormal patellar tendon contains an oval or round area of high signal intensity on T1 and T2 and the proton density–weighted images at the tendon attachment, or a focal zone of high signal intensity in the deep layers of the tendon insertion [9–11] (Figure 18-1). Tendons with patellar tendinopathy have increased anteroposterior diameter in the affected region [9,11,12].

The T2 weighted sequences (particularly the T2 weighted GRE sequences) have better sensitivity than the T1 weighted protocols [9,10,13]. However, the T1 weighted signal can image most cases of patellar tendinopathy.

In clinical practice, MR scans can identify the exact location and extent of tendon involvement, and help exclude other clinical conditions such as bursitis and chondromalacia [11]. Surgeons use MR to assess the severity of patellar tendon disease and determine how much tendon to excise [9,11].

Disadvantages of MR include cost, and the slow, often incomplete resolution of signal changes after surgical intervention [14,15]. Whether MR abnormalities occur in asymptomatic patellar tendons has not been examined, but other tendons contain high signal abnormality on MR in nearly a quarter of young volunteers [16].

Abnormal signal without change in size must be interpreted with caution, as the normal patellar tendon has a range of appearances due to technical factors and intrinsic fiber differences [17]. In particular, the "magic angle" phenomenon can result in false positive high signal intensity on GRE T2 weighted images of normal tendon [10,18,19].

Ultrasonography (c)

Sonographic studies in athletes with the clinical features of patellar tendinopathy should include both knees using high-resolution, linear array, 10 or 12 mHz ultrasound transducers. It is vital that the examination is performed with the probe exactly perpendicular to the tendon to avoid a false positive image due to artifactual hypoechogenicity [20]. Sonographic appearances in jumper's knee reveal a focal hypoechoic area (Figure 18-2) combined with various amounts of swelling of the surrounding tendon. Hyperechoic regions within the tendon correspond with dystrophic ossification on histopathology [10].

A proportion of asymptomatic athletes have sonographic hypoechoic regions in their patellar tendons. Among volleyball players, 24% of asymptomatic knees contained patellar tendons with hypoechoic regions on US [21]. Similarly, 15% of basketball players with no past history of knee pain had abnormal tendon morphology on US [22]. Comparable findings have been reported in

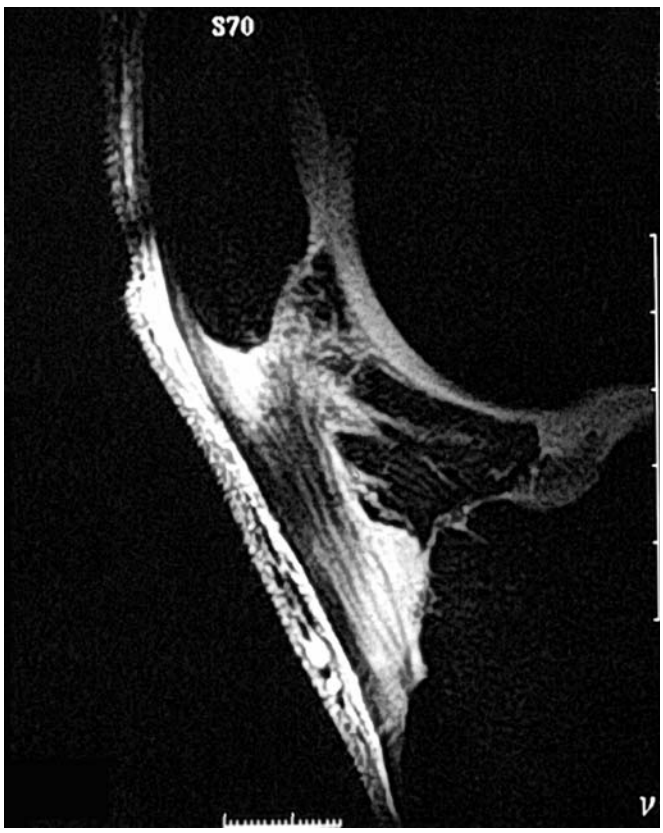

FIGURE 18-2. The same patient at MRI scanning.

asymptomatic recreational athletes [23]. Furthermore, longitudinal studies found that hypoechoic US regions did not predict subsequent development of symptoms in athletes [24], but conferred relative risk of patellar tendinopathy in 16- to 18-year-old basketball players [25]. These data suggest prudence when considering ultrasound appearance as a guide to prognosis and management [26]. A sonographic hypoechoic region is not, of itself, an indication for surgery [24,25,27]. Surgeons have used US to accurately locate the area of tendinopathy to allow correct placement of the scalpel blade when performing multiple percutaneous longitudinal tenotomies [28].

Conservative Management of Patellar Tendinopathy

Given the degree of morbidity associated with chronic tendon problems, and the extent of knowledge in certain areas of medical treatment, there is a surprising lack of scientific rationale for tendon treatment [1]. Conservative and operative treatments of tendinopathies vary considerably among surgeons and across countries. Unfortunately, "there is little scientific evidence for the majority of treatments proposed and used for chronic tendon problems [14]." Thus, the treatment outlines suggested below are, at best, "empirical." We discuss conservative management of the athlete with patellar tendinopathy under 5 broad headings: 1) decreasing load on the tendon; 2) eccentric strengthening; 3) cryotherapy and physical modalities including ultrasound and laser; 4) remedial massage; and 5) pharmacological management.

Decreasing Load on the Tendon

Load can be reduced on the tendon by reducing overall activity, and by improving the efficiency of lower limb biomechanics to protect the patellar tendon. Athletes who present with patellar tendinopathy for the first time have their best opportunity to make a full recovery by resting from competition and undergoing thorough rehabilitation before returning pain-free to their sport. This approach is advocated even in elite regular competitions (e.g. an NBA season, premier league soccer, world league volleyball), but a compromise may be necessary immediately prior to one-off tournaments such as world championships or Olympic games. Nevertheless, this conservative approach is often unacceptable to players (who have little pain) and to coaches (who are paid to have short-term goals). A poor alternative may be a period of rest taken at the end of a season, but by that time the tendon is likely to have a much greater degree of tendinosis. In jumping sports, forces generated in landing are substantially greater than those that produce the jump [29]. Therefore, correcting biomechanics improves the energy-absorbing capacity of the limb, both at the affected musculoskeletal junction and at the hip and ankle. The ankle and calf are critical in absorbing the initial landing load, and any functional compromise of these structures increases the load transmitted to the knee [29]. About 40% of landing energy is transmitted proximally [30]. Thus, the calf complex must absorb a major portion of the load that would otherwise be transmitted proximally to the patellar tendon–quadriceps complex. Compared with flat-foot landing, forefoot landing generates lower ground reaction forces; if this technique is combined with a large range of hip or knee flexion, vertical ground reaction forces in landing can be reduced by a further 25% [31].

These data suggest that the practitioner should assess the patient's static alignment and functional biomechanics [32]. Anatomic variants that predispose to patellar tendinopathy are listed in Table 18-1. Some anatomic abnormalities, such as pes planus, may be evident during static assessment, but others, such as excessively rapid pronation, may only be evident during dynamic evaluation. Shoe orthoses are one method of correcting some biomechanical faults. Some physicians, but not us, routinely recommend knee braces.

Biomechanical abnormalities arise from functional as well as anatomic abnormalities. Low flexibility of the quadriceps, hamstrings, iliotibial band, or calf has the potential to restrict range of motion at the knee and ankle joints and is likely to increase the load on the patellar tendon. Hamstring tightness, detected by a decreased sit-and-reach test, is associated with increased prevalence of ultrasonography-proven patellar tendinopathy [25]. Weakness of the gluteal, lower abdominal, quadriceps, and calf muscles leads to fatigue-induced aberrant movement patterns that may alter the forces acting on the knee. It is imperative that proximal and distal muscle groups be assessed in patients with chronic patellar tendinopathy.

Table 18-1. Anatomical characteristics associated with patellar tendinopathy

Limb or joint	Anatomical characteristic
Foot	Excessive range of pronation, excessively fast pronation (even within a normal range), pes planus, rigid cavus foot, poor dorsiflexion (e.g., due to anterior impingement syndrome)
Knee	Hyper- or hypomobile patella leading to poor mechanism of patellofemoral movement, tight band between illiotibial band and patella
Thigh	Tight iliotibial band
Hip	Coxa vara, femoral anteversion

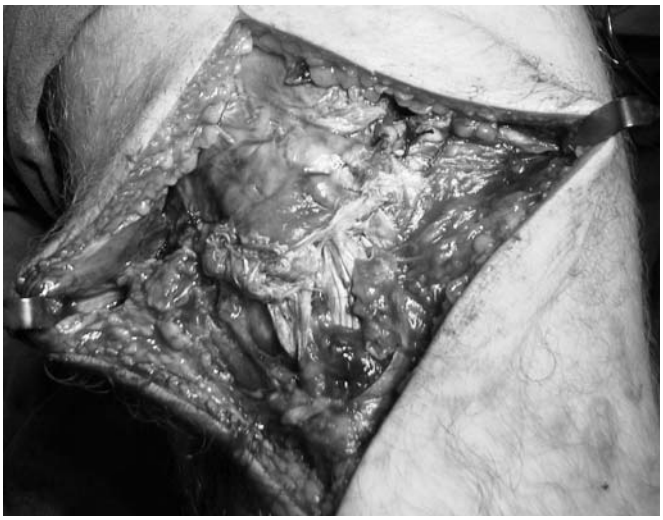

FIGURE 18-3. Typical appearance of ruptured patellar tendon at surgery.

Eccentric Exercise Protocol

Eccentric strengthening has long been recognized as the keystone to successful management of tendinopathies [33,34]. Well-designed studies have demonstrated the efficacy of strengthening as a treatment for both Achilles and adductor tendinopathy [35–37], but there are few published studies of strengthening for the patellar tendon [33,38].

Eccentric loading of the patellar tendon may well be essential to promote clinical recovery [33,39,40]. The key exercise was a drop squat from standing to about 100 to 120 degrees of knee flexion. Patients perform 3 sets of 10 repetitions per session (one session daily) [33,40]. The proposers of this treatment program emphasized the principles of training specificity, maximal loading, and progression [41,42]. In basketball and volleyball players, specificity is achieved by using jumping activities as the rehabilitative exercise. Maximal loading occurs when patients feel pain in their tendons during the third set of 10 repetitions. Progression is achieved by increasing the speed of movement or by increasing the external resistance, again using pain as a guide. Ice is used to cool the tendon after the eccentric training.

Some authors [41,42] contend that athletes often do not need to refrain from sport during the strengthening program, and that athletes become asymptomatic after 6 to 8 weeks exercising [33]. Under close clinician supervision to adjust the program as needed [41,42], this regimen brought complete relief to 30% of patients and marked decrease in symptoms to a further 64% [33]. The remaining 6% had worsening symptoms. A randomized pilot study found a nonsignificant trend for eccentric strengthening to be superior to a concentric training program in patellar tendinopathy [38]. Given the natural history of patellar tendinopathy, these results are encouraging.

Prescribing Eccentric Exercise: Practical Issues

Therapists often have concerns as to when and how they should begin a strengthening program. Even athletes with severe patellar tendinopathy should be able to begin some exercise, at the very least standing calf strength and isometric quadriceps work (see Figure 18-4). On the other hand, the athlete who has not lost appreciable knee strength and bulk can progress quickly to the speed part of the program. Table 18-2 presents a strength program embracing the activities and timelines that our clinical experience has shown to be effective.

Both pain and the musculotendinous unit's ability to do work should guide the amount of strengthening activity. If pain is a limiting factor, then the program must be modified so that the majority of the work is relatively pain free, and does not cause delayed symptoms, commonly pain the morning after exercise.

A subjective clinical rating system such as the Victorian Institute of Sport Assessment (VISA) score [43–45]

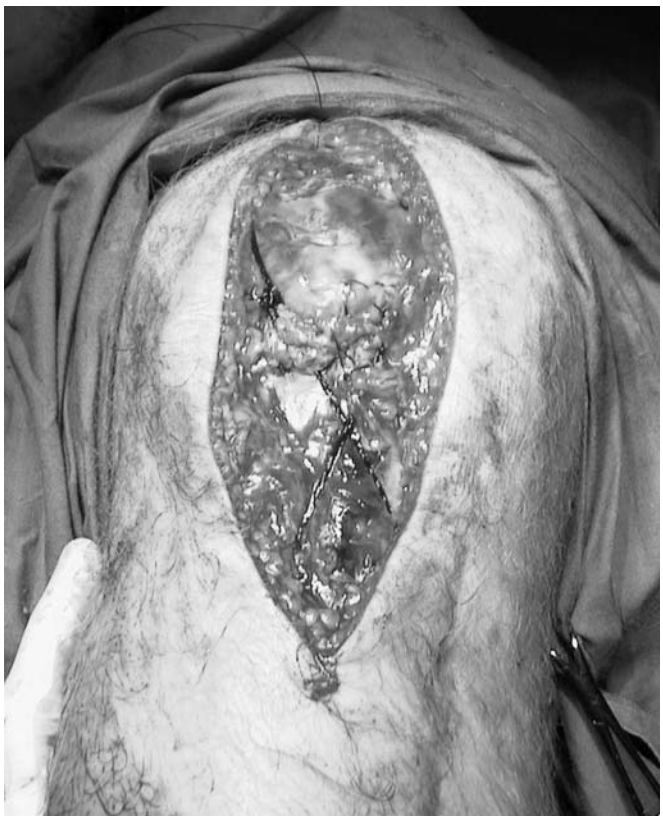

FIGURE 18-4. Repaired patellar tendon with end-to-end suture and figure-of-eight reinforcement with a figure-of-eight strong absorbable material passed through a tunnel in the anterior tibial tuberosity and over the superior pole of the patella.

Table 18-2. Outline of strengthening program for management of patellar tendinopathy

Timing	Type of overload	Activity
0–3 months	Load endurance	Hypertrophy and strengthen the affected muscles, focus attention on the calf as well as the quadriceps, gluteals
3–6 months	Speed endurance	Weightbearing speed-specific loads
6+ months	Combinations dependent on sport (e.g., load, speed)	Sports-specific rehabilitation

(a numerical scale for assessing the severity of jumper's knee) helps both the therapist and the patient to measure progress, and it allows early detection of any worsening of symptoms.

If pain is under control, then the practitioner supervising the program should monitor the control and quality with which the patient performs the exercises. Progression to the next level of the program should only be performed if the previous work load is easily managed, pain is controlled, and function is satisfactory.

Because athletes with patellar tendinopathy tend to "unload" the affected limb to avoid pain, they commonly have not only weakness, but also abnormal motor patterns that must be reversed. Strength work must progress to single leg exercises, as bilateral exercises only offer options to continue to unload the tendon. Some physicians and therapists maintain that quadriceps-only exercises such as leg extensions have a place in the rehabilitation of patellar tendinopathy, specifically to load the quadriceps exclusively, not allowing the calf and glutei to "take over" the exercise. Similarly, we have found that squats performed on a 30-degree decline board are effective in reducing the influence of the calf group in retarding knee flexion such as occurs in a normal squat done with the heels fixed. The therapist can help the patient progress by adding load and speed to the exercises, and then endurance can be introduced once the patient can do these exercises well. After that, combinations such as load (weight) and speed, or height (e.g. jumping exercises) and load can be added. These end-stage eccentric exercises can provoke tendon pain, and are recommended only after a sufficiently long rehabilitation period and when the sport demands intense loading. In several sports it may not be necessary to add height to the rehabilitation program at all, whereas in some sports (volleyball, for example), it is vital.

The exercise program must help to correct aberrant motor patterns such as stiff landing and pelvic instability. For example, athletes must learn to perform weightbearing exercises within a functionally required range with appropriate pelvic control.

Failure in rehabilitation strength programs can stem from many sources. They include too rapid a progression of rehabilitation; inappropriate loads (not enough strength or speed work, eccentric work too early or aggressively, insufficient single leg work); too many electrotherapeutic modalities; and lack of monitoring patients' symptoms during and after therapy. Rehabilitation and strength training must also continue through the return to sports, rather than ending immediately on return. Finally, plyometrics training can be performed inappropriately, not tolerated, or done unnecessarily.

Cryotherapy and Physical Modalities

To control initial tissue response to tendon injury, most clinicians advise rest, cryotherapy and anti-inflammatory medication (see below). Cryotherapy is thought to act by decreasing blood flow and metabolic rate, thereby limiting tissue damage. Electrical modalities that have been used in patellar tendinopathy include ultrasound, heat, interferential therapy, magnetic fields, pulsed magnetic and electromagnetic fields, transcutaneous electrical nerve stimulation (TENS), and laser. The background to the use of these modalities is explained elsewhere [1,39]. Their true effects remain unknown. Whether or not modalities are beneficial in tendinopathy is an area of disagreement. One of us (NM) generally includes cryotherapy, laser and magnetic fields, and pulsed magnetic and electromagnetic fields as part of treatment [39].

Remedial Massage

Remedial massage aims to decrease load on tendons by improving muscle stretch. Deep friction massage may activate mesenchymal stem cells to stimulate a healing response [45].

Pharmacotherapy

The main pharmaceutical agents used to treat patellar tendinopathy have been nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids. After discussing these, we review the data regarding novel agents for treating tendinopathies.

Although the biological basis for using NSAIDs in tendinopathies is not obvious [1,46], these drugs are undoubtedly the most commonly used symptomatic therapy. In the only double-blind, placebo-controlled study of NSAIDs in tendinopathy, piroxicam did not benefit patients with Achilles tendinopathy [47]. A study of topical ketoprofen in patellar tendinopathy showed that the drug reached target tissue, but the authors did not assess clinical outcome [48]. Although the use of "anti-inflammatory" medication seems paradoxical in a degenerative condition, NSAIDs act in ways beyond their well-known anti-inflammatory mode [49,50]. For

example, in human cartilage *in vitro*, some NSAIDs stimulate and some inhibit glycosaminoglycan synthesis [51]. If this also proves to be the case *in vivo* in tendons, it would provide a mechanism whereby NSAIDs could influence extracellular matrix. Thus, it would appear premature to rule out any potential benefit of this class of medication merely because tendinopathy is not an inflammatory condition.

Injection and infiltration of corticosteroids by means such as iontophoresis has a dramatic effect on symptoms arising from inflamed synovial structures [52]. However, the role of corticosteroids in management of tendinopathy remains controversial [53,54]. A key point is that, after injection, a tendon is at increased risk of rupture until appropriate strengthening has been undertaken [55]. Recently, aprotinin has been trialed in the management of patellar tendinopathy [56]. Aprotinin is a polyvalent inhibitor of the proteases—collagenase, elastase, metalloprotease, kallikrein, plasmin, and cathepsin C [57]. At least in the short term, aprotinin (2 to 4 injections of 62,500 IU with local anesthetic in the paratendinous space) seems to offer better chances of pain relief than corticosteroids. However, patients with an insertional tendinopathy fared less well than those with tendinopathy of the main body of the tendon. As aprotinin is an anti-inflammatory agent, its administration is probably only warranted in athletes with relatively short duration of symptoms.

Using a combination of all of the above conservative management modalities, 33 of 42 athletes who presented to a specialist clinic within a few weeks from the onset of symptoms were able to return to their sport 6 months after the first visit. In the 9 patients presenting with Blazina's Stage 3 disease, conservative measures failed, and surgery was necessary [58].

Surgical Management

Patellar tendon surgery [26,59–67] is generally performed when the patient has not improved with at least 6 months of conservative management. A variety of surgical methods for treatment of jumper's knee have been described [68]. These include: drilling of the inferior pole of the patella; resection of the tibial attachment of the patellar tendon with realignment [69]; excision of macroscopic degenerated areas [61,62]; repair of macroscopic defects [65]; scarification (i.e. longitudinal tenotomy/tenoplasty of the tendon) [70]; percutaneous needling [71]; percutaneous longitudinal tenotomy [28]; and arthroscopic debridement [72]. Surgical technique is based on surgeon's opinion and experience, as the pathophysiology of patellar tendinopathy is not known.

Patellar tendon surgery has a rather unpredictable outcome. A review of 23 papers found that the outcomes of surgery varied between 46% and 100% [73]. In the 3 studies that had more than 40 patients, authors reported combined excellent and good results of 91%, 82%, and 80% in series of 78, 80, and 138 subjects, respectively. The mean time for return to preinjury level of sport varied from 4 months to greater than 9 months. A long-term study of outcome in patients who underwent open patellar tenotomy for patellar tendinopathy showed that only 54% were able to return to previous levels of sport activity [72]. In 2 prospective studies that evaluated time to return to sport, most subjects required more than 6 months, and often 9 months, to return to full sporting competition [15,28]. Unfortunately, several factors confound the analysis of outcome of surgery [73]. Surgeons differ in their diagnostic criteria, selection of cases for surgery, the actual operation performed, as well as in their postoperative protocols. Different types of surgery result in differences in the amount of bone either excised or drilled from the inferior pole of the patella, the margin of normal tissue excised around the macroscopically degenerative tissue, the use or avoidance of longitudinal tenotomies, and the type of closure, if any, of the tendon after surgery. Intersurgeon technical ability is another major factor whose influence has never been studied.

Recently, we have shown that the scientific methodology behind published articles on the outcome of patellar tendinopathy after surgery is poor, and that the poorer the methodology the higher the success rate [73]. Obviously, improving study design would provide clinicians with a more rigorous evidence-base for treating patients who have recalcitrant patellar tendinopathy.

Conclusions

Patellar tendinopathy is a degenerative, not an inflammatory, condition of the patellar tendon, most likely a result of excessive load bearing. Clinical assessment is the key to diagnosis, although the presence of US or MR abnormalities increases the likelihood that the patient's symptoms arise from the patellar tendon. Imaging appearances should not dictate management, which, for the time being, remains based on clinical experience, rather than scientific rationale.

A variety of management modalities exist, including correcting perceived underlying biomechanical problems, local physical modalities such as ice, and, when the patient is pain free, a graduated strengthening program emphasizing functional exercises including eccentric training. Eccentric training appears most promising, but well-designed controlled studies are awaited. To prescribe exercise effectively requires thorough assessment of the patient's functional capacity and a skillful approach to gradually increasing the demand that the athlete imposes on the tendon.

Clinical experience suggests that, in some patients, peritendinous corticosteroid or aprotinin infiltration may be warranted as an adjunct to other appropriate treatments. Surgery is indicated after a 6- to 9-month trial of appropriate conservative management. Open patellar tenotomy is the conventionally accepted surgical treatment of insertional patellar tendinopathy, but often requires 6 to 12 months rehabilitation. Arthroscopic debridement has been proposed, and, although randomized controlled trials are lacking, this procedure may permit earlier return to sport than traditional open surgery, even though both techniques have an equal success rate at 12 months [72].

Rupture of the Patellar Tendon

Rupture of the patellar tendon is relatively infrequent. It is usually seen in active patients around the age of 40 [74], but, with the increased participation in sports of all ages, it is not uncommon to see this injury in older patients.

The vast majority of ruptures of the patellar tendon are unilateral, although bilateral ruptures have been described [75]. In these instances, a rupture may occur during less strenuous, nonathletic activity [76].

Biomechanics of the Extensor Mechanism

During active knee extension, forces generated in the quadriceps muscle complex are transferred via the patellar tendon to the proximal tibia. Forces generated in the patellar tendon while ascending stairs are approximately 3.2 times body weight [77], and the force necessary to cause a patellar tendon rupture in a weight lifter approached 17.5 times body weight [78]. The force required to rupture a tendon weakened by systemic disease is much lower, although no data are available.

Etiopathogenesis of Patellar Tendon Rupture

Tensile overload of the extensor mechanism usually leads to a transverse fracture of the patella, which is considered the weakest link [75]. Patellar tendon rupture due to indirect trauma is probably the end stage of chronic tendon degeneration [79]. In a study of 53 patellar tendons, all the ruptured tendons exhibited pathologic changes, whereas such changes were detected in only one third of intact tendons from age-matched control subjects [80].

In a review of 13 athletes with chronic jumper's knee resulting in tendon failure, 10 sustained a patellar tendon rupture (one bilateral), and 3 had a quadriceps tendon rupture [79]. Patients younger than 25 presented more severe symptoms of patellar tendinopathy before rupture, and older patients complained of less severe symptoms. Probably, more advanced degeneration is required to critically weaken a younger tendon. Less advanced microtrauma, combined with the natural increase in stiffness resulting from ageing, probably lead to rupture in older athletes. Patients with preexisting systemic disorders, such as systemic lupus erythematosus, rheumatoid arthritis, chronic renal failure, and diabetes mellitus, are susceptible to patellar tendon ruptures during non-strenuous activity [75]. Patellar tendon ruptures are also seen in patients on long-term systemic corticosteroids [81], who are at greater risk of bilateral ruptures [75,81]. A rupture may also occur after local corticosteroid injection close to the tendon for chronic patellar tendinopathy [82]. In the series by Kelly et al. [79], 8 of 13 patients treated for jumper's knee received 2 to 3 corticosteroid injections in or around the patellar tendon before rupture.

Patellar tendon rupture has been reported after some surgical procedures that disturb the midsubstance or insertion sites of the patellar tendon, such as total knee arthroplasty [83]. In these instances, no single technique consistently guarantees good results. Primary repair, possibly reinforced with autogenous hamstrings, and extensor mechanism allografts have been advocated [83]. Primary suturing of the torn tendon ends should be attempted if adequate local tissue is present. Allografts may be considered at a later stage as a method of salvage.

Patellar tendon rupture can ensue after anterior cruciate ligament reconstruction performed with use of the central third [84]. Proximal tendon rupture or avulsion of the distal pole of the patella usually occurs when patients are too active before complete healing of the graft harvest site. Primary tendon repair is the management of choice, and the rupture normally does not interfere with the result of the anterior cruciate ligament reconstruction.

History and Physical Examination

Normally, the patient has sustained a forceful eccentric contraction of the knee extensors against the full body weight, placing the knee in a flexed position (e.g. landing after a rebound, tripping up the stairs). Sudden pain with an associated tearing or popping sensation is experienced. Unassisted weightbearing is impossible. The patient usually presents with a tense hemarthrosis and inability to bear weight on the involved leg. Intra-articular injuries must be excluded. The most important sign in patellar tendon ruptures is the lack of active extension of the knee with inability to maintain the passively extended knee against gravity. If the rupture extends completely through the tendon and the retinacula, active extension is lost completely. Less commonly, if the rupture involves only the tendon, and most of the retinacular fibers remain intact, some extension will be possible [74]. A short while after the injury, there is often a palpable gap at the level

of the rupture, and the patella may be proximally displaced compared with the contralateral side. Passive knee flexion is markedly diminished because of pain.

When the diagnosis is delayed, the tendon defect may be obscured by consolidation of the haematoma and early scar formation. Some active knee extension can be possible, but an extensor lag will be evident. Quadriceps atrophy and proximal migration of the patella are usually seen. Weight bearing may be possible, but often with a forward flinging motion of the affected leg in the swing phase and feelings of knee instability during single-leg stance [85]. Stair climbing and rising from a chair are very difficult.

Imaging

Plain Radiography

Plain radiographs are cost-effective and are sufficient in most cases. On the lateral view, a patella alta is identified. One or more bone fragments may be seen if the tendon was avulsed.

Ultrasonography

High-resolution ultrasonography is effective to image the patellar tendon in both acute and chronic injuries [86]. In acute ruptures, hypoechogenicity is noted over the entire thickness of the tendon. In chronic tears, thickening of the tendon at the rupture site is seen, with disruption of the normal echo pattern.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is an expensive way of diagnosing acute and chronic patellar tendon ruptures [87]. The normal patellar tendon demonstrates a homogeneous low signal intensity throughout its course on proton-density images. The anterior and posterior margins are well defined. In patellar tendon ruptures, there is discontinuity of tendon fibers, waviness of the ends of the tendon, and an increase in signal intensity on sagittal T2 weighted images. Hemorrhage and edema may also be seen posteriorly to the infrapatellar fat pad. We do not recommend MRI to evaluate acute patellar tendon ruptures. However, it may be helpful in patients with neglected tears, when the diagnosis is uncertain or when associated intra-articular injuries are suspected.

Classification

Various authors have tried to classify patellar tendon ruptures on the basis of the location, configuration, and duration of the rupture. Some authors [88] grouped patellar tendon ruptures into 3 categories according to the location of the disruption: distal pole of the patella, tendon midsubstance, or tibial tubercle. Others [79] classified the ruptures according to the morphology of the tears: transverse, Z type (medial patellar avulsion with a lateral tubercle avulsion), and inverted-U type (medial and lateral portions ruptured from the tibial tubercle and the midportion avulsed from the patella). No study has been able to show a correlation between the type of rupture and the method of repair undertaken or the clinical outcome.

When bilateral ruptures were classified into midsubstance tears or tendon avulsions from the proximal or distal end, most cases of midsubstance rupture occurred in patients with chronic diseases. Proximal or distal tendon avulsions were usually seen in healthy patients with no systemic or local disease. Again, there is no correlation between the type of tear and the clinical result [81].

The only classification correlated with outcome takes into account the time between injury and repair. When repaired patellar tendon ruptures were divided into 2 categories based on the interval between injury and repair, immediate repair and delayed repair performed more than 2 weeks after injury [74], immediate repair was shown to be beneficial. Primary tendon repair was usually feasible in patients operated on immediately, whereas preoperative patellar traction and fascia lata autografts were often required for patients with a neglected rupture. Patients who underwent a delayed repair had greater loss of flexion and higher incidences of persistent quadriceps atrophy and strength loss.

Management

Conservative management is ineffective.

Immediate Repair

Surgical repair to restore the extensor mechanism should be performed as soon after the injury as possible both in athletic and nonathletic patients, regardless of their age. A number of methods for immediate surgical repair have been described. We perform simple end-to-end, Bunnell-type repair with the use of heavy absorbable sutures through transosseous tunnels with a reinforcing cerclage suture of absorbable suture material or tape [79,81,88–91] (see Figures 18-3 and 18-4). The repair is performed under tourniquet control. A longitudinal midline incision is made from the midpatella to the tibial tubercle. Full-thickness skin flaps are lifted to expose the tendon and the adjacent retinacula. The tendon ends are debrided. If the injury involves an avulsion off the distal pole of the patella or tibial tubercle, the tendon is cleared of loose bone fragments too small for internal fixation. The medial and the lateral extent of the retinacular tear are identified, and repaired. Three or 4 heavy absorbable sutures are passed through a transverse hole approximately 1 cm posterior to the tibial tubercle, brought prox-

imally, and passed transversely through the quadriceps tendon close to the superior border of the patella in a figure-of-eight fashion. Tension is applied to the sutures, which are then clamped but not tied. In the North American literature, it is recommended that a lateral radiograph of the knee be obtained at this stage to assess the patellar height compared with that on preoperative radiographs of the contralateral knee. However, residual articular incongruity alone is not be the cause of persistent anterior knee pain in patients who undergo repair. We prefer to flex the knee to 90 degrees, test the tension of the sutures, adjust them as required, and tie them. The wound is closed in layers, and the knee is placed in a well-padded dressing, without using braces.

Postoperative Rehabilitation

Isometric quadriceps- and hamstring-strengthening exercises are begun on the first day after surgery, and we allow protected weightbearing with crutches from the second postoperative day. Active flexion and extension of the knee are started 2 weeks after the operation. Weightbearing progresses to full weightbearing without crutches by 4 to 6 weeks postoperatively, if good quadriceps control is shown.

Although isokinetic rehabilitation is very much in fashion, we prefer to undertake functional rehabilitation with isoinertial exercises, gradually progressing to a sport-specific functional rehabilitation program. Resumption of strenuous athletic activity is allowed 4 to 6 months postoperatively, when the patient should have a full range of knee motion and at least 85% of the strength of the contra-lateral extremity.

Delayed Repair

Simple end-to-end suture is difficult when the repair has been delayed more than 6 weeks [74,85,92]. The longer the delay between injury and repair, the greater the likelihood of quadriceps contracture and proximal patellar migration. Fibrous adhesions may form between the patella and the femur. Distally, the ruptured tendon ends retract and become embedded in scar tissue, and calcific intratendinous deposits may develop. Patients with a neglected rupture of several months' duration who present with superior patellar migration, loss of passive patellar mobility, and lack of full passive knee motion may require preoperative patellar traction [74]. This can be accomplished over several days to a few weeks with a 2 kg weight through a Steinmann pin placed transversely through the midportion of the patella. Lateral radiographs of the knee are taken weekly until the patella has been returned to its anatomical position.

Primary repair combined with autogenous graft augmentation using the fascia lata [74] or hamstring tendons [92] has been described, and should be attempted if sufficient tendon is left for repair. Carbon fibers [93] and nonabsorbable tape suture materials [74] have also been advocated. Achilles and patellar tendon allografts have also been used, mostly in salvage situations [94]. Allografts allow early, vigorous rehabilitation. However, there is a risk of disease transmission.

Rehabilitation after a neglected rupture is considerably more conservative than that after repair of a fresh rupture. A brace is used for 6 weeks, during which time gentle passive exercises are performed, followed by active range of motion exercises to regain full extension. Closed manipulation of the knee is occasionally required to increase the range of motion of the knee.

Results

Most patients undergoing early primary repair achieve nearly full return of knee motion and extension strength, although persistent quadriceps atrophy is common [74,88]. The only factor that appears to correlate with clinical outcome is the timing of repair. Patients who undergo delayed repair have greater persistent quadriceps atrophy.

No large series evaluates the results of reconstruction of a neglected rupture [85,92,94]. Patients who require pre-operative traction and the use of autogenous or allograft tendons may have a worse result than patients with sufficient tissue for an end-to-end repair [74].

Complications

Decreased quadriceps strength and loss of full knee flexion are the most common complication after a patellar tendon repair. Aggressive postoperative rehabilitation, with early range of motion and quadriceps exercises, should be implemented. Manipulation under anaesthesia may be considered if 120 degrees of flexion are not achieved by the second post-operative month. Arthroscopic debridement can be considered to achieve the last few degrees of extension if these are lacking despite appropriate rehabilitation. Quadriceps atrophy does not compromise the final return of strength, both subjectively and objectively [74,88].

Hemarthrosis necessitating either aspiration or formal drainage occasionally develops, and the use of a suction drain should be considered. Wound infection or skin dehiscence may occur, usually over the distal aspect of the wound, where the skin is relatively thin. This complication can be avoided by placing the skin incision lateral to the tibial tubercle, and by imparting less tension at the suture line. Rerupture is occasionally seen in patients who attempt to return to running or jumping activities before complete healing of the repaired tendon [74]. Revision is usually successful in reestablishing knee

motion and strength, as long as the repair is performed early. When the repair has been reinforced with a cerclage wire, wire breakage may occur. The wire may need to be removed because of skin irritation and wire extrusion. For this reason, we use absorbable sutures, rather than metallic wire, as the reinforcing material. Patella baja may occur, with resultant motion loss and the risk of patellofemoral degeneration if excessive tension on the sutures causes inaccurate coaptation of the tendon ends.

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