



Conservative treatment of patellar tendinopathy

Jill L. Cook, Karim M. Khan and Craig R. Purdam

Patellar tendinopathy disrupts athletic careers in several sports and is resistant to many forms of conservative treatment. Outcome after conservative treatment has been minimally investigated, and the effect of these treatments on the pathology of overuse tendinopathy are not well understood.

The clinical assessment of patellar tendinopathy appears straightforward, but evidence suggests that the importance of imaging and palpation in diagnosis and ongoing assessment may be overestimated. There is a lack of clinically relevant research on which to base treatment. However, the principles of management for patellar tendinopathy derived from clinical experience include load modification, musculotendinous rehabilitation, and intervention to improve the shock absorbing capacity of the limb. The role of electrophysical agents, massage, and stretching in the treatment of patellar tendinopathy are also discussed. The progression of treatment is based on clinical grounds due to a lack of reliable subjective and objective tools to assess recovery.

The failure of some conservative programs could be due to either athlete compliance or practitioner expertise. The management of patellar tendinopathy is complex, and if the physiotherapist addresses all the principles of treatment, the chance of success could be increased.

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Jill L. Cook PhD,
PGManipPhys,
BAppSci (Phys),
Musculoskeletal
Research Centre, La
Trobe University,
Australia

Karim M. Khan PhD,
MD, Department of
Family Practice and
School of Human
Kinetics, University
of British Columbia,
Canada

Craig R. Purdam
MSportsPhys,
DipPhys, Australian
Institute of Sport,
Australia

Correspondence to:
J. Cook, Senior
Lecturer,
Musculoskeletal
Research Centre,
School of
Physiotherapy, La
Trobe University,
Bundoora Victoria
3083, Australia. Tel:
+61 3 9479 5789;
Fax: +61 3 9479
5768; E-mail: J.
Cook@latrobe.edu.
au

Introduction

Tendon pain due to overuse is a common injury in many sports (Kannus 1997b). The injury may limit, or even prevent, sporting participation for some time. For an athlete, any compromise to training and playing is harmful to their sporting career and health.

Treatment of tendon overuse is based on clinical guidelines that have their origin in tradition and supposition (Khan et al. 2000). Clinical decision making is difficult due to a paucity of understanding of, and research on, overuse tendinopathy. Consequently, athletes may experience lengthy and frustrating rehabilitation periods, with a relatively unpredictable outcome (Cook et al. 1997).

The patellar tendon is vulnerable to overuse injury due to repetitive landing loads and activities that involve changes of direction that are the essence of most sports (Kannus 1997a). Patellar tendinopathy is most commonly characterised by pain at the inferior pole of the

patella, although pain can also be at the tibial attachment and the attachment of the tendon to the superior pole of the patella (Blazina et al. 1973). The spectrum of presentation can range from a mildly irritating condition to an acute, irritable, inhibiting pain.

The pain associated with patellar tendinopathy can be recurring, often appearing several times in an athlete's career (Cook et al. 1997). Hence, reduction in pain may not indicate resolution of the pathology, and changes in load may provoke a recurrence of symptoms.

Loss of musculotendinous strength such as a prolonged layoff from training appears to expose the tendon to an increased risk of symptoms redeveloping.

Pathology

The pathology of overuse tendinopathy is well documented and similar in all tendons affected

in sport (Khan et al. 1999a). Tendons transmit load through an organized extracellular matrix composed mainly of Type I collagen. In the pathological state, this organized matrix is damaged, with consequent load intolerance. The pathology of tendinopathy underlies the difficulties associated with management, and explains the prolonged healing times and propensity for recurrence.

There are four main components of tendon pathology: deterioration of the collagen bundles, an increase in ground substance, activation of the cellular components, and vascular proliferation (Józsa et al. 1990). In the first component of tendon pathology, collagen is affected by disruption of the fibres (transverse disruption) or bundles (longitudinal separation), with the resulting gap filled with excess ground substance (Józsa et al. 1990). The longitudinal separation of collagen decreases the number of crosslinks between fibres, which together with a loss of fibre continuity, creates a significant decrease in the strength of the tendon (Eyre et al. 1984).

The second component of tendon pathology involves ground substance, a combination of proteoglycan bodies and glycosaminoglycan chain. The ground substance is important, but sparse in normal tendon (Scott 1995). In tendinopathy there is both a large increase in ground substance and a change in the type of proteoglycan present. The small dermochondran sulphate of normal tendon is replaced by a larger chondroitin sulphate proteoglycan in tendinopathy (Benazzo et al. 1996).

As well, cellularity is increased in tendinopathy with tendon cells becoming active and producing both collagen and ground substance, presumably in an attempt to repair the tendon. Other cells (e.g. myofibroblasts) migrate into the tendon, but there is no evidence of an inflammatory reaction from such immigrant cells (Kraushaar & Nirschl 1999).

Although much of tendinopathy is hypercellular and blastic in nature, there are areas of tendon that are either devoid of cells (cystic tendinopathy) or have cells suggesting a decrease in cellular function (Józsa et al. 1982). Such cells have alterations of both their nucleus and mitochondria, resulting in a condition that has been termed hypoxic tendinopathy (Józsa

et al. 1982). Both the hypercellular and acellular areas of tendinopathy can be found in a single tendon, however, it is unclear what causes parts of each tendon to react differently.

Neovascularization is also seen in tendinopathy, although the patency and function of the vessels is questionable (Kraushaar & Nirschl 1999). The new vessels are tortuous, thick walled, have small lumen, and provide little evidence of blood flow within them. It has been suggested that neovascularization is a response to hypoxia, however there is no evidence of improved healing surrounding the newly vascularized areas. Although it is assumed that the new vessels grow into the area of tendinopathy, Kraushaar and Nirschl (1999) suggested they are produced by local metaplasia.

Despite the consistent nature of these four described features of tendinopathy, several other pathological processes can occur (Józsa et al. 1990). These other processes can be classified as either add-on features (e.g. calcific tendinopathy) or as further descriptions of the same process (e.g. mucoid, hyaline tendinopathy).

Tendinopathy repair

Many aspects of attempted tendon repair (e.g. cellular activation, neovascularization) are defined as pathology. Repair also involves the production of Type III collagen, that may or may not remodel into Type I collagen in an unknown timeframe (Maffuli et al. 2000).

The repair of overuse tendinopathy appears to be compromised by processes that are not as yet understood. Hence the tendon does not fully complete the repair process and tendinopathy could be defined as a failed healing response (Clancy 1989). The tendon is left with disorganized, loosely aggregated Type III collagen, separated by excessive ground substance, and interspersed with hypercellular and hypervascular areas. All of these structures compromise tendon function, and the physical, chemical and pharmacological stimulants that improve repair are unclear.

It is unknown if tendons progress to full healing, either returning to normal morphology or repairing with evidence of 'scar'. Diagnostic imaging (ultrasound, magnetic resonance

imaging [MRI]) reveals that tendons may remain abnormal for years after initial injury (Cook et al. 2001). Nevertheless, some tendons with abnormal imaging at baseline appear normal on imaging at followup (Cook et al. 2001). What is occurring histopathologically in the tendon when this happens is unknown.

Pathology of the patellar tendon

The pathology of patellar tendinopathy is identical to that outlined above for all overuse tendinopathies (Järvinen et al. 1997). As well, patellar tendinopathy has a propensity to develop cystic changes (Khan et al. 1996). Such cystic changes involve an acellular area of pathology comprising extracellular debris with no capacity for repair. The cyst is 'walled off' from areas of the tendon that are still cellular and thus capable of continued repair.

Patellar tendinopathy also involves pathology at the bone tendon junction (the enthesis), with an increase in depth of the fibrocartilage zone and a breakdown in the stratified transition from bone to tendon (Ferretti et al. 1983). The involvement of the enthesis is another example of an area that is poorly understood and researched.

The posterior (in the sagittal plane) and central (in the axial plane) part of the tendon suffers pathology most commonly, although medial and laterally placed lesions have been described on axial imaging (Cook et al. 1998). Involvement of the anterior aspect of the tendon, especially those fibres that extend over the patella itself, appears limited to those tendons with extensive changes on imaging. The biomechanical reason for this pattern of pathology is unclear, although the tendon attachment to bone may be less resilient to load than continuous tendon tissue (Uthoff & Matsumoto 2000).

Assessment of patellar tendinopathy

Clinical examination

The history and clinical examination of patellar tendon injury in most cases are straightforward, however, the objective diagnostic tests for tendinopathy may not be as valid and reliable

as they appear (Cook et al. 2000b, 2001).

Differential diagnosis appears simple, but the anatomical complexity of the region contains several potential traps.

The history of patellar tendinopathy reveals increasing pain in the tendon under load, impaired athletic performance, and in severe cases, even pain with daily activities (e.g. ascending and descending stairs, squats) and night pain. In extreme cases, the knee may 'give way' under load. Previous episodes of symptoms in the same or opposite knee are common. A sudden onset of pain may not indicate acute partial tear, as quiescent tendinopathy exists in athletes (Cook et al. 1998), and sudden pain may be the first presentation of long term pathology (Cook et al. 2000b).

Examination of the quadriceps musculotendinous unit reveals pain and possible weakness with muscle testing, likely muscle wasting and limitation of simple functional tests. For example, the athlete may be unable to hop or squat fully and repeatedly without pain and may exhibit unloading patterns. These unloading strategies protect the affected joint or leg, using other joints or leg preferentially to unload painful or weak structures. This allows the athlete to continue to function with a compromised knee extensor mechanism function. Athletes with short term or mild symptoms may 'unload' the knee in landing and increase the use of hip and ankle joint and muscles. This landing pattern includes landing with a more rigid knee, and utilizing hip flexion, internal rotation, and adduction to absorb landing shock not absorbed satisfactorily at the knee. Athletes experiencing long-term symptoms, or those with severe pain, often unload the whole limb, landing and changing direction preferentially with the asymptomatic leg, and can present with more comprehensive weakness and dysfunction of the knee, hip, and ankle.

Tenderness to palpation is always present in patients with patellar tendinopathy, but recent data suggest that an athlete's tendon may be tender even in the absence of pathology (Cook et al. 2001). Similarly, palpation tenderness is not a particularly useful indicator of clinical improvement as tenderness may persist beyond clinical recovery.

Quantifiable subjective and objective measures at baseline that can be used throughout rehabilitation are essential. The Victorian Institute of Sport Assessment (VISA) score is a reliable and valid subjective measurement for monitoring the progress of rehabilitation (Khan et al. 1998, Visentini et al. 1998). It is an eight-question scale that assesses pain, function, and sporting participation out of a possible maximum score of 100 points (Fig. 1). It quantifies the symptoms and dysfunction in patellar tendinopathy, and is a valuable tool to assess recovery (Khan et al. 1999b).

A further clinically valuable tool is the decline squat (Fig. 2), a test reported to discriminate knee extensor function better than previously used loading tests (Cook et al. 2000a). The 25 degree decline decreases calf contribution to the squat, and by keeping the trunk upright (to minimize gluteal function), and completing it on a single leg (to minimize unloading), the knee extensors are loaded maximally. By quantifying the pain (verbal 10-point scale) and recording the knee flexion angle at which this pain occurs, it is a reliable and simple reassessment tool.

Differential diagnosis

As the patellar tendon is surrounded by complex anatomical structures, differential diagnosis can be difficult. Tendon pain is typically well localized, so vague diffuse pain must be examined and investigated thoroughly. In particular, pain from the patellofemoral joint should be suspected.

Inferior pole patella chondropathy mimics patellar tendinopathy almost perfectly, and is almost impossible to clinically differentiate from tendon pain. Magnetic resonance imaging (MRI) may provide clues to this diagnosis.

Although well documented (McConnell 1991, Duri & Aichroth 1995), the role of the fat pad in the production of inferior pole patellar pain has been little researched. Clinical experience suggests taping this site can be effective in pain reduction, but there is insufficient evidence to show that this pathology contributes substantially to infrapatellar pain.

As the patellofemoral joint and patellar tendon are intimately anatomically related it

appears plausible for infrapatellar pain to arise from both structures. Clinically, however, this is uncommon, as the elite athlete appears to be more vulnerable to tendon injury than to abnormal patellofemoral joint function. Conversely, the recreational athlete probably places insufficient load on the tendon to cause injury but commonly suffers patellofemoral joint problems. The muscle weakness and dysfunction found in patellar tendinopathy may cause secondary patellofemoral pain, so the practitioner should attempt to assess cause and effect accurately. Obviously some athletes may have both conditions simultaneously; the older athlete will have both tendon and joint aging that can predispose them to both conditions (Ippolito et al. 1975).

Imaging

Tendon imaging provides excellent morphological detail of the tendon. Tendon pathology has characteristic appearance on both MRI and ultrasound (US) (Fig. 3). Both imaging modalities are effective in showing tendon swelling, an increase in water content (held within the increased ground substance), and changes at the bony insertion (Read & Peduto 2000). There is high correlation between imaging changes and the histopathology described above in tendons from surgical series (Yu et al. 1995, Khan et al. 1996, Green et al. 1997).

The relationship of abnormal images to the clinical and symptomatic status of the tendon is poor. Although imaging confirms that tendon pathology exists, abnormal imaging exists in athletes' tendons without pain (Miniaci et al. 1995, Cook et al. 1998). Hence, imaging alone does not confirm that the source of the pain is the tendon, and clinical assessment skills are imperative in providing an accurate diagnosis.

Similarly, imaging does not predict clinical outcome in patellar tendinopathy. Although it is tempting to assess an US and assign symptom severity and likely outcome to the image, there is no evidence that this is clinically appropriate. Recent research indicates that imaging bears little relationship to symptomatic outcome. Imaging can resolve, remain unchanged, or expand without predicting the symptoms of patellar tendinopathy (Khan et al.

1. For how many minutes can you sit pain free?
 0 mins 100 mins

2. Do you have pain walking downstairs with a normal gait cycle?
 Strong severe pain No pain

3. Do you have pain at the front of the knee with full active non-weightbearing knee extension?
 Strong severe pain No pain

4. Do you have pain when doing a full weightbearing lunge?
 Strong severe pain No pain

5. Do you have problems squatting?
 Strong severe pain No pain

6. Do you have pain during or immediately after doing 10 single leg hops?
 Strong severe pain No pain

7. Are you currently undertaking sport or other physical activity?
 0 Not at all
 4 Modified training ± modified competition
 7 Full training±competition but not at the same level as when symptoms began
 10 Competing at the same or higher level as when symptoms began

8. Please complete **EITHER A, B or C** in this question.
 •If you have **no pain** while undertaking sport please complete Q8a only.
 •If you **have pain** while undertaking sport but it does not stop you from completing the activity, please complete Q8b only.
 •If you **have pain** that stops you from completing sporting activities, please complete Q8c only.

8a. If you have no pain while undertaking sport, for how long can you train/practice?
 Nil 0-5 mins 6-10 mins 11-15 mins >15 mins
 0 7 14 21 30

OR

8b. If you have some pain while undertaking sport, but it does not stop you from completing your training/ practice, for how long can you train/practice?
 Nil 0-5 mins 6-10 mins 11-15 mins >15 mins
 0 4 10 14 21

OR

8c. If you have pain that stops you from completing your training/practice, for how long can you train/practice?
 Nil 0-5 mins 6-10 mins 11-15 mins >15 mins
 0 2 5 7 10

TOTAL VISA SCORE

Fig. 1 Victorian Institute of Sport Assessment (VISA) score.



Fig.2 Decline squat.

1997, Cook et al. 2000b, 2000c, in press). Clinical decision making in patellar tendinopathy is exactly what the name suggests – clinical. Imaging results should not impact on this decision-making process in any way.

Principles of rehabilitation

Evidence for treatment

There are few studies on the conservative treatment of patellar tendinopathy. In vitro studies indicate the efficacy of some treatments such as frictions (Gehlsen et al. 1999), therapeutic ultrasound (Ramirez et al. 1997), and exercise (Almekinders et al. 1993) in the management of tendinopathy. Clinical studies of the patellar tendon have been very limited in number and in scope but indicate that eccentric

exercise, frictions, and iontophoresis may be effective forms of treatment.

Although eccentric exercise is the hallmark of most tendinopathy rehabilitation programs, few studies have investigated clinically relevant exercise programs. Eccentric exercise using isokinetic machines have been shown to cause strength improvements (Jenson & Di Fabio 1989). Pain reduction with eccentric drop squat exercises is less predictable (Cannell et al. 2001). Eccentric exercise in the Achilles tendon has shown to be very successful clinically (Alfredson et al. 1998, Alfredson & Lorentzon 2000).

In a study by Penderghest et al. (1998) phonophoresis did not improve pain perception in tendinitis (sic) more than stretching, strengthening, and ice. Similarly, Pellechia et al. (1994) reported that iontophoresis improved pain and functional outcome measures more than phonophoresis combined with frictions and other modalities. It is difficult to draw any clinically relevant conclusions from so few studies and further research is clearly needed.

There is, however, strong evidence that the inflammation paradigm is inaccurate and the popular treatments of rest, anti-inflammatory medication, and ice may no longer be appropriate. Few studies have investigated overuse tendinopathy but histopathology at end-stage disease has not demonstrated any inflammatory reaction (Yu et al. 1995, Green et al. 1997). The current description of tendon pathology as degenerative or as a failed healing response does not however help clinicians, as it is unknown what treatments, if any, may affect degeneration or improve healing. Hence, there are no treatments that are known to directly and positively affect pathologic tendon tissue.

Principles of rehabilitation

Without sufficient mechanistic evidence on which to base treatment, the principles of rehabilitation are inevitably clinically based. The priority of most physiotherapy is to decrease pain associated with function. However, how we treat tendinopathy if the cause of the pain is not known, is less clear. Tendon studies have shown that structural changes do not always equate to pain, hence physiotherapy directed at improving tendon

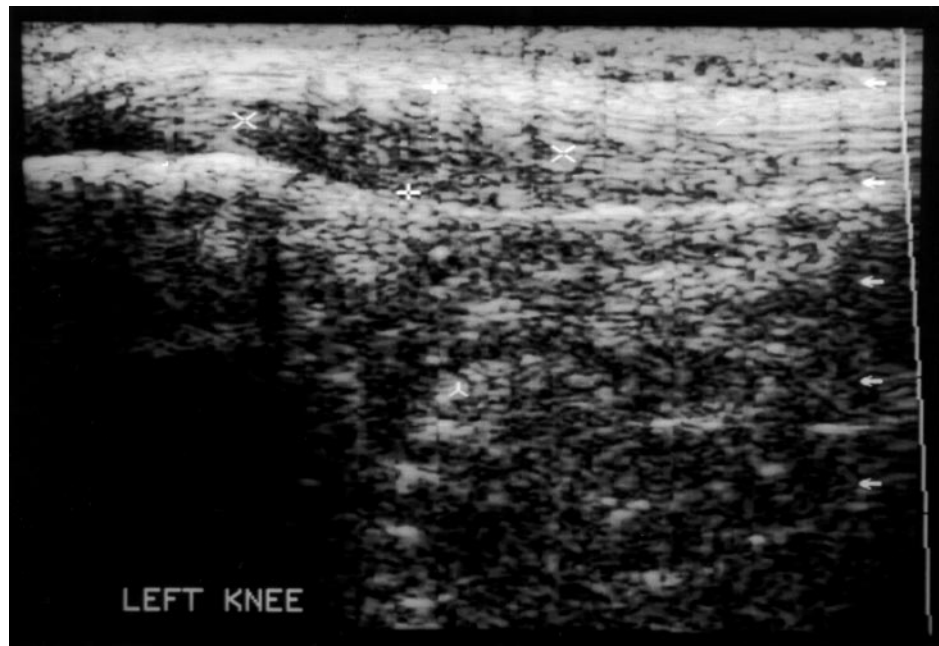


Fig. 3 Classic US images (sagittal view). This imaging appearance is seen in tendons with and without symptoms, and does not indicate the likely outcome.

structure may not affect pain. Evidence is mounting for a biochemical cause of pain, with high levels of glutamate (a neurotransmitter) found in achilles tendinopathy. Such evidence has been found by both direct measurement and exclusion of other possible causes (Alfredson et al. 1999, Khan & Cook 2000)

The acceptable level of pain in a rehabilitation program has been recently debated with some programs incorporating higher levels of pain than previously considered acceptable (Curwin & Stanish 1984, Alfredson et al. 1998). As both programs appear to be successful, the effect of pain on rehabilitation is unknown.

A second priority of physiotherapy is to improve function of both the individual and affected tissue. Once again this is problematic in tendinopathy if it is unclear what affects the repair process either positively or negatively. We can, however, improve musculotendinous function by prescribing eccentric exercise programs even if it remains unclear how improved function alters pain. Eccentric control is a crucial part of most sports and it is possible to hypothesize that improved musculotendinous function reduces the peaks of tendon load experienced in sporting

activities. Alternatively, (Banes et al. 1995), in the laboratory setting, has shown that mechanical loading stimulates cellular protein synthesis and upregulation of nuclear protein. It has been speculated that eccentric exercise may provide a similar stimulus in vitro (Khan & Cook 2000).

Rehabilitation

Although standard eccentric exercises may offer adequate rehabilitation for some tendons, many patients with patellar tendinopathy do not respond to this prescription alone (Cannell et al. 2001). As the knee is the middle joint of a kinetic chain (between the ankle and hip) the influence and effect of joints and muscles above and below the knee must also be considered.

Given that patellar tendinopathy affects high-level athletes more than recreational sportspeople (Torstensen et al. 1994), the demand for successful rapid recovery is high. Monitoring and modification of training and competition as well as appropriate rehabilitation must be undertaken. Athlete and coach compliance are essential components of a successful program.

There are several clinical principles that must be applied when treating patellar tendinopathy. These principles hold for both the athlete with new and mild symptoms as well as those with long term and intense symptoms. Specifically, these clinical principles are changing the load on the tendon, strengthening the musculotendinous unit and improving both motor patterning and shock absorption in the leg.

It is important to acknowledge that there is little evidence for this rehabilitation program that follows. This approach has been based on the authors' clinical experience.

Reduction of abusive load

It is imperative to reduce abusive load if the ability of the musculotendinous unit to function is decreasing due to pain. This principle can be applied in a wide spectrum of ways: from a reduction in training volume or intensity to rest from all training. It is vital to find the baseline training level that does not provoke the tendon, and to take this as the point to start the rehabilitation process. It is rare that the tendon will need complete rest, as rest diminishes tendon strength (Kannus et al. 1997) and therefore should be avoided.

Improving musculotendinous function

Both the knee extensor unit and those areas affected by unloading of the knee (the ankle and hip) must be strengthened. Strengthening should incorporate all the components necessary for sporting function such as strong eccentric contraction, fast eccentric contraction, rapid change from an eccentric to concentric contraction, endurance, landing from a height, and combinations of these constituents.

The usual process of regaining function is to initially strengthen and hypertrophy weak muscles (up to 3 months). The gradual addition of speed to the program (up to 6 months) is followed by sport specific movement patterns (up to 12 months). A gradual return to training and competition follows.

Competition generally places more load on the tendon than training (Zernicke et al. 1977). Although it can be challenging to reduce players' training loads after the competitive season begins, this is normally necessary during the initial return to playing sport after

tendinopathy. Often the first season of competition after a sustained lay-off with patellar tendinopathy requires several 'rest' days, where the player does weights and activities that do not provoke the tendon. To prevent recurrence, it is critical to maintain a strong emphasis on strength work for at least 12 months after return to sport, as motor patterning and unloading tendencies appear to persist for long periods.

Improvement in pain and function often plateau at one, or both, of two stages of rehabilitation. The first of these is the stage of introduction of speed training and the second is the introduction of drills that emphasize changes of direction and sudden stops. The athlete can usually progress if exercises are adequately modified or load is sustained for an extended period when symptoms first develop.

Improving shock absorbing capacity of the limb

In the closed kinetic chain, the knee joint and patellar tendon function as a secondary shock absorber, as the ankle joint, foot, and calf complex are the primary ground contact points (McClay et al. 1994). Hence any factors that compromise the function of these structures increase the load on the knee.

Athletes involved in jumping activities are very susceptible to repeated ankle injury (Hickey et al. 1997) and consequent joint degeneration and impingement (Brodelius 1961). This decreases available range of ankle dorsiflexion and joint range to absorb shock.

Similarly, calf strength is compromised by ankle injury and by unloading patterns. Rehabilitation must, therefore, correct limited ankle joint range of movement and calf weakness.

Hip strength is less important than knee and ankle strength, but motor pattern changes with long-term symptoms will reduce strength in both the abductors and extensors of the hip. Although there are good strategies to improve abductor strength, little attention is paid to the extensor strength, and functional exercise prescription is problematic due to the muscle strength and endurance needed.

Retraining motor patterns

Although improving strength allows better motor patterns, poor patterns may become

habitual and thus, correcting motor patterns throughout rehabilitation is essential. This should start with the simplest of exercises and be reinforced continually, through to return to sport.

Maintaining fitness

Although not sport specific, maintaining some fitness is relatively easy in patellar tendinopathy. There are several activities that do not stress the tendon excessively. Cycling, stepper, and running in water are all excellent activities for cardiovascular fitness and they generally do not provoke tendon symptoms. Athletes appear to comply better with rehabilitation if they are able to participate in some fitness activity.

Electrophysical agents

As with exercise programs, there is little clinical evidence that the use of electrophysical agents improve clinical outcome in tendinopathy. The dosage (intensity, frequency and treatment time) for most of these modalities is not based on scientific rationale (Van der Windt et al. 2000). Clinically, magnetic field therapy and very low dose ultrasound may permit some chronically painful tendons to progress into rehabilitation, but the use of electrophysical agents should remain a low priority in treatment.

Massage and frictions

Regular quadriceps and calf massage are important as compliant muscle may allow the energy from landing to attenuate over a greater time. Massage may also decrease delayed muscle soreness associated with eccentric exercise.

Frictions are more difficult to prescribe, as intensity, frequency, and treatment time are poorly understood. Frictions can be provocative as well as beneficial and it is hard to ascertain the clinically appropriate time to use such techniques. Theoretically, tendinopathy may benefit from the mechanical stimulus of frictions.

Stretching

Although stretching is integral to most injury rehabilitation guidelines, it is not as important in patellar tendinopathy as improving muscle

function and motor patterning. Two muscles, however, warrant particular attention. First, hamstring tightness has been shown to correlate with abnormal US imaging in the patellar tendon in junior male basketball players (Cook 2000). Relieving hamstring tightness is also an integral part of the treatment of patellofemoral pain (McConnell 1986). Second, calf muscle flexibility may also be important, as calf tightness can limit ankle dorsiflexion, and in turn, decrease ankle shock absorption capabilities. Each athlete requires a stretching routine individualized to his or her needs. Other muscles that commonly need addressing are tensor fascia lata, psoas, and quadriceps.

Length of rehabilitation

Rehabilitation of patellar tendinopathy can be a lengthy process, particularly in those athletes with poor function and profound weakness. Athletes with long-standing (>12 months) and/or severe symptoms can rarely rehabilitate in less than 3 months, and often require in excess of 6 months to do so adequately.

Although athletes with minimal symptoms can usually make a rapid return to sport, any deficiencies in strength, abnormal loading pattern, and dysfunction must still be corrected. This is best done in the off-season or during pre-season time where load modification can be more easily achieved.

Progressing treatment

There are few reliable and quantifiable measures of progression of rehabilitation. The VISA score and decline squat (both described previously) are simple and adequate tests. As symptomatic improvement can occur without consequent changes in tenderness on tendon palpation and imaging, improved musculotendinous function under load is the only clinical indicator that should be used to show treatment progression is needed.

Failure of conservative rehabilitation

In some cases, tendon rehabilitation (as outlined) may improve function but not symptoms, or symptoms may prevent strength gains. This usually occurs relatively early in rehabilitation and means decisions must be made about further management such

as psurgery, injectable medication (e.g. corticosteroid, aprotinen) and retirement. Corticosteroid injections offer short term pain relief (up to 8 weeks) (Stahl & Kaufman 1997) that may permit rehabilitation to progress. Clinically, if one injection does not allow progress, it is unlikely that multiple injections will improve outcome.

Rehabilitation outcome may be compromised by poor athlete compliance or practitioner acumen. Athletes can retard rehabilitation, as they may find long term compliance difficult, especially if their symptoms are mild. Similarly, slow progression in rehabilitation can limit athlete motivation (Kolt 2000). Clinicians may fail to prescribe programs that have suitable levels of eccentric exercises or adequate load increases. A reliance on electrophysical agents rarely offers long-term improvement.

Conclusion

Patellar tendinopathy can be problematic and resistant to conservative treatment. Further research on the efficacy of conservative treatment protocols is clearly needed before therapists can have confidence in prescribing efficacious rehabilitation.

Treatment must be holistic, recognizing the pathology and addressing the consequent secondary problems that arise from dysfunction. Many athletes fail to respond to treatments that seek an easy, short term solution. Such treatment programs are usually based on flawed understanding of tendinopathy. The therapist must produce a management plan, a rehabilitation timetable, and protocol to suit the athlete and the particular level and commitments of the sport.

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