Patellar tendinopathy

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Patellar tendinopathy is a common and significant syndrome encountered in sports medicine. Referring to a clinical condition characterized by activity-related, anterior knee pain associated with focal patellar-tendon tenderness [1–3], patellar tendinopathy is believed to result from repeated loading of the knee extensor mechanism, and is thus most prevalent in sports involving some form of jumping [4]. In recognition of this association with jumping, patellar tendinopathy was first described as and is commonly referred to as ''jumper's knee'' [4–8]. This term is misleading, however, as the condition is found in a wide variety of sports people, including those who do not participate in sports involving jumping [4,9–11].

Another traditionally popular term to describe the clinical condition is ''patellar tendinitis.'' Histopathological studies, however, have consistently shown the pathology underlying patellar tendinopathy to be degenerative rather than inflammatory [10,12–14]; thus, the suffix -itis, implying the presence of inflammation, is inaccurate. To describe the histopathological presentation of the condition the term ''tendinosis'' is preferred [2,3,10]. This distinction is important, as the correct labeling and understanding of the pathology has repercussions for management and the outcome expectations of both the clinician and athlete [2].

To clarify the terminology surrounding the syndrome, it has been advocated that the term patellar tendinopathy be used clinically to describe overuse conditions of the patellar tendon [1–3]. Alternative terms such as tendinosis and tendinitis should only be applied following pathologist examination of tissue biopsies, as these refer to distinct histopathological conditions that cannot be assessed clinically. In line with these recommendations, the term patellar tendinopathy is used in

the following discussion, which overviews current understanding of the pathology, pathophysiology, and pathogenesis of patellar tendinopathy, and discusses aspects of its clinical examination, imaging, and management.

Pathology and pathophysiology

The predominant pathological feature of patellar tendinopathy is tendinosis, typically in the deep posterior portion of the patellar tendon adjacent to the lower pole of the patella [13]. Tendinosis is characterized by progressive tissue degeneration with a failed reparative response and the complete absence of inflammatory cells [10,12,14]. Macroscopically, this makes the afflicted region of the tendon soft and gives it a yellow-brown, disorganized appearance—an appearance that is commonly labeled ''mucoid degeneration'' [15–18]. This contrasts with the normal appearance of a glistening, stringy, parallel-organized, white tendon.

When viewed microscopically, the pathological region is distinct from normal tendon, with both matrix and cellular changes. Instead of clearly defined, parallel, and slightly wavy collagen bundles, tendinopathy is associated with relative expansion of the tendinous tissue, loss of the longitudinal alignment of collagen fibers, and loss of the clear demarcation between adjacent collagen bundles [4,10, 12–14,19]. The tissue has lost its normal reflective appearance under polarized light, and there is gradual and increasing separation of collagen fibers that distorts the normally dense homogenous polarization pattern [4,10]. Occasional clefts in the collagen suggest microtears that may be interpreted as microscopic partial ruptures [20]. In addition, there are frequently focal regions of intratendinous calcification [10,21–24]. The latter may arise due to traction injury to the inferior pole of the patella [10,24]; however, recent evidence has shown the calcification to have formed discretely via endochondral ossification [21].

Multiple cellular changes coexist with the matrix changes in tendinopathy. The most obvious of these changes is hypercellularity resulting from an increase in cellular proliferation [25]. There is atypical fibroblast and endothelial cellular proliferation [12,19,26], and extensive neovascularization [10,14,19,22,27]. These changes represent an attempt at healing. The collagen-producing tenocytes lose their fine spindle shape, and their nuclei appear more rounded and sometimes chondroid in appearance, indicating fibrocartilaginous metaplasia [13]. Of note is the consistent finding of a clear absence of inflammatory cells [4,10,12,14].

Despite the absence of inflammatory cells in patellar tendinopathy, mediators in the inflammatory cascade appear to be involved in its pathophysiology. Of particular note is the involvement of cyclooxygenase-2 (COX-2). COX-2 is an inducible enzyme that rate-limits the production of proinflammatory prostaglandins, such as PGE_2. In tendons harvested from patients with patellar tendinopathy, both the tendon tissue itself and harvested cells expressed higher levels of COX-2 than healthy control patellar tendons [12]. In addition, the harvested cells produced greater in-vitro quantities of PGE_2 [12], although this has yet to be confirmed in

vivo [28]. The potential involvement of inflammatory pathways in patellar tendinopathy has implications with regard to the use of anti-inflammatory agents in its management (discussed later).

Pathogenesis

To prevent patellar tendinopathy and to develop appropriate treatment strategies when it does occur, an understanding of the pathogenesis is required. Unfortunately, the precise mechanism by which patellar tendinopathy develops is currently unknown. As with most overuse conditions, the development of patellar tendinopathy is likely to be due to a range of factors, with the relative contribution of each factor varying among individuals. These factors can be grouped into two categories: extrinsic and intrinsic.

Extrinsic factors are the most commonly indicted in the pathogenesis of patellar tendinopathy, with the most frequently reported causative factor being mechanical overload. For patellar tendinopathy to develop, repeated heavy loading of the tendon is required [6,29]. This explains its prevalence in sports involving some form of jumping, such as basketball and volleyball. In volleyball, a direct relationship exists between the number of training sessions (number of jumps) and the development of patellar tendinopathy [6].

As the characteristic lesion in patellar tendinopathy typically occurs in the deep posterior portion of the patellar tendon adjacent to the lower pole of the patella, it has been hypothesized that loading exposes this region to the most strain. This hypothesis has contrasting evidence, with two cadaver studies reporting opposing results [30,31]. One found greater strain in the anterior portion of the tendon [30], whereas the other reported greater strains in the posterior portion [31]. Although the latter study did find evidence to support the hypothesis of greater strains in the posterior portion of the tendon, the authors also found the posterior portion of the patellar tendon to be more adapted to loading, as evident by its enhanced mechanical properties. Thus, it is not clear whether patellar tendinopathy is purely a strain-related phenomenon.

Although extrinsic factors may be the most consistent causative factor in the development of tendinopathy, the development of patellar tendinopathy in some athletes while others with equivalent loading are spared signals that intrinsic factors must also contribute. Johnson et al [32] hypothesized that impingement of the inferior pole of the patella onto the tendon may contribute to the pathogenesis. This is supported by the findings of altered patella anteroposterior tilt [33] and a long inferior pole [18,34] in many knees with tendinopathy. Recent research, however, found no difference between symptomatic and asymptomatic knees in terms of the tendon-patella angle during flexion, suggesting that impingement is not an important contributing factor [35]. The long inferior pole found in some symptomatic athletes may merely represent a traction osteophyte caused by repeated high-tensile forces in this area [34], and may not have been a pre-existing contributory abnormality.

Other intrinsic factors that have been postulated as causes of patellar tendino-pathy include malalignment, patella alta, abnormal patellar laxity, and muscular tightness and imbalance [36]. Of these, only muscle tightness has prospectively been shown to be a predisposing factor. In particular, Witvrouw et al [36] found decreased flexibility of the quadriceps and hamstring muscles to be significantly associated with the subsequent development of patellar tendinopathy.

How extrinsic and intrinsic factors combine to trigger the generation of patellar tendinopathy is not established. It is possible that the pathological changes are initially triggered by matrix changes. Heavy loading may cause tensile failure of tendon fibers, resulting in microdamage. When this occurs, tenocytes must increase their production of collagen and matrix. This is a slow process, however, due to the inherently slow turnover rate of collagen. With further loading, an area of tendinosis may develop due to progressive microdamage and subsequent failed healing attempts.

An alternative to matrix-mediated changes is the possibility of cellular-triggered pathological changes. Recent research has shown a direct relationship between the amount of stress that tendon cells are exposed to and the induction of a stress-activated protein kinase, c-Jun N-terminal kinase (JNK) [37]. Although transient activation of JNK is associated with normal cell processes, persistent JNK activation has been linked to the initiation of programmed cell death or apoptosis. Although yet to be shown in patellar tendinopathy, increased cellular apoptosis has been shown in supraspinatus tendons with tendinosis [38].

History and clinical examination

A solid history and clinical examination are cornerstones to the diagnosis of patellar tendinopathy. Patellar tendinopathy presents subjectively as well-localized anterior knee pain related to activity levels [5,39]. Pain is usually insidious and gradual in onset, and may be precipitated by an increase in the frequency or intensity of repetitive ballistic movements of the knee. Initially pain may present as a dull ache at the beginning of or after strenuous activity. This initial symptom may be ignored as it warms up with further activity [40]. With continued use, however, pain can progress to be present during activity and can ultimately interfere significantly with performance. In some cases there is a constant ache at rest and night pain that disturbs sleep [10,27,34]. Other common complaints are pain when seated for long periods, and when ascending and descending stairs [29].

On clinical examination, the most consistent finding is patellar tendon tender-ness [29,41]. This is typically located at the inferior pole of the patella; however, it is influenced by knee position [42]. With the knee flexed to 90° the tendon is placed under tension, and tenderness significantly decreases and may disappear altogether. Thus, the patellar tendon should be palpated in relaxed full-knee extension. Pressure on the superior border of the patella should be applied to tilt the inferior pole anteriorly, enabling palpation of the tendon origin. Using this method, pain on palpation can reliably be graded as either mild, moderate, or severe [43]. Undue

significance should not be attached to mild pain in isolation of other signs and symptoms of patellar tendinopathy, as it may be a normal finding in active athletes [43,44].

In addition to palpation, other features to note on clinical examination are muscle size and functional strength. Patients with chronic symptoms may exhibit wasting of the quadriceps, with the vastus medialis obliquus portion most commonly affected. Overall thigh circumference may be reduced and calf atrophy may also be present. Functional strength testing of the quadriceps may be performed by asking the patient to perform 15 one-legged step-downs in which the non-weight–bearing foot is not allowed to touch the ground between cycles [45]. The work capacity of the calf can be assessed by performing single-legged heel raises. A jumping athlete should be able to perform a minimum of 40 raises [45]. During both activities the onset of fatigue and the quality of movement should be monitored, and both activities should be performed bilaterally.

To reproduce symptoms of patellar tendinopathy a useful functional test is the decline (30°) squat test. This places greater load on the patellar tendon than does a squat on level ground [45]. Objective measurement during this test can obtained by determining the number of decline squats before the onset of pain, and by asking the athlete to indicate the level of pain on a visual analog or verbal reporting scale. An alternative method of objectively assessing an athlete with patellar tendino-pathy is to implement the Victorian Institute of Sport Assessment (VISA) scale [46]. This scale provides a numerical index of the severity of patellar tendinopathy by assessing both symptoms and function. A maximum score of 100 indicates full, pain-free function.

The key differential diagnosis in patellar tendinopathy is patellofemoral pain syndrome. This is usually straightforward to differentiate, as the subjective and objective features of patellar tendinopathy are generally distinctive. In some cases, however, differential diagnosis may be difficult and the two conditions may coexist. One method to aid differentiation is to perform functional testing (ie, decline squat test) with and without the use of taping to influence the patellofemoral joint. At least, this may indicate whether the patellofemoral joint should also be treated.

In addition to patellofemoral pain syndrome, patellar tendinopathy needs to be differentiated from fat-pad syndromes, and assessment of other potential coex-isting conditions such as meniscal tears and cartilage degeneration may need to be considered where indicated [22]. Also, the potential of pain referral to the knee should not be ignored [47].

Imaging

Imaging can be used to confirm the clinical diagnosis of patellar tendinopathy with the techniques of choice being ultrasonography and magnetic resonance imaging (MRI). Both provide excellent anatomic representation of the patellar tendon, and histopathologic studies have shown that the characteristic tendino-

pathy appearances observed with both forms of imaging are due to the underlying tendon pathology [10,14,19,26,48,49].

Ultrasonography

Ultrasonography provides a readily available, quick, and inexpensive method of imaging the patellar tendon. The tendon is readily examined using high-frequency linear array transducers with the knee flexed or semiflexed, and by obtaining both longitudinal and transverse images [50]. In suspected cases of patellar tendinopathy, ultrasonography can be used to confirm the existence and location of intratendinous lesions. These lesions are reflected by decreased echogenicity, evident by either diffuse hypoechogenicity or a focal sonolucent region, typically in the deep posterior portion of the tendon adjacent to the lower pole of the patella [8,10,27,39,50–55] (Fig. 1). The decrease in echogenicity represents a decrease in the ultrasound attenuative properties of the tendon, resulting from the disruption of the collagen bundles. Other common findings on ultrasonography include tendon thickening [8,24,27,44,53,56], irregularity of the tendinous envelope [8,53], intratendinous calcification [8,24,44,51,57,58], and erosion of the patellar tip [8]. The primary disadvantages of ultrasonography are its operator-dependency and somewhat limited soft-tissue contrast [40].

Magnetic resonance imaging

MRI provides high spatial resolution that allows detailed anatomic structures to be identified, and it provides high intrinsic tissue contrast that allows normal tendons to be distinguished from abnormal tendons. On MRI, patellar tendinopathy is characterized by a focal increase in signal within the tendon as well as an alteration in its size [14,19,26,27,32,34,59,60]. The later is necessary with some sequences, as the "magic angle" phenomenon associated with MRI can artificially increase signal intensity, resulting in false-positive findings [61–63]. El-Khoury
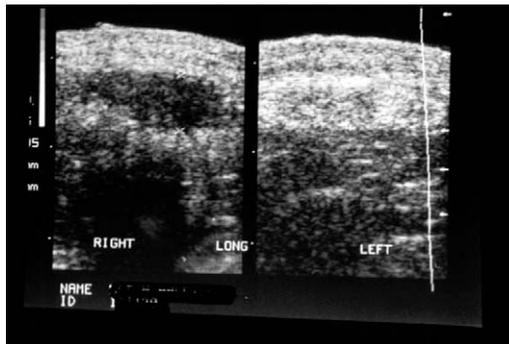

Fig. 1. Ultrasound appearance of patellar tendinopathy. On the left side of the figure, the right tendon shows a large area of decreased echogenicity (blackened area) associated with tendon thickening. On the right side, the left tendon shows a normal appearance of the patellar tendon.

et al [59] suggest an anteroposterior diameter cutoff point of 7 mm between symptomatic and asymptomatic tendons; however, more recent authors have shown considerable overlap and variation in tendon thickness [34,64,65]. The primary disadvantages of MRI are its relatively high cost, limited availability in some regions, and lengthy time for scanning.

Limitations of imaging

Although both ultrasonography and MRI are useful in imaging patellar tendinopathy, neither can be labeled as the gold standard for its diagnosis. Positive ultrasonography and MRI images for patellar tendinopathy have been shown in asymptomatic tendons [10,39,44,51,58,60,65–69]. Similarly, symptomatic tendons can have the imaging appearance of normal asymptomatic tendons [32,39,43,44,54,69]. Using currently available data, the sensitivity and specificity of ultrasonography can be calculated at 58% and 94%, respectively (Table 1). For MRI, the sensitivity and specificity can be calculated at 78% and 86%, respectively (Table 2). Thus, although ultrasonography and MRI may accurately reflect tendon morphology, the imaging appearance may not necessarily reflect clinical symptoms. Further confirming this, numerous authors have shown no correlation between the severity of tendinopathy symptoms on clinical grading systems and tendon appearance on ultrasonography [44,57,68].

Table 1
Results of ultrasonography assessment in symptomatic and asymptomatic patellar tendons

		Symptomatic tendons				Asymptomatic tendons			
	Total tendons	Total	+	−	+/−	Total	+	−	+/−
Cook et al [4]	57	57	57	0		0			
Cook et al [39]	176	19	15	4		157	34	123	
Cook et al [58]	374	0				374	71	303	
Cook et al [66]	46	0				46	18	28	
Davies et al [27]	16	16	13	0	3	0			
Fredberg and Bolvig [67]	98	3	3	0		95	15	80	
Fritschy and de Gautard [8]	47	32	32	0		15	nr	nr	
Khan et al [10]	63	28	28	0		35	6	29	
Khan et al [70]	23	8	8	0		15	8	7	
Lian et al [44]	81	30	23	7		51	12	39	
Myllymaki et al [54]	62	62	31	31		0			
Panni et al [68]	84	46	46	0		38	14	24	
Terslev et al [69]	18	4	3	1		14	12	2	
Summary	1145	305	259	43	3	840	190	635	0

Calculations
Sensitivity = 58%
Specificity = 94%
Positive predictive value (PV+) = 86%
Negative predictive value (PV−) = 77%
Abbreviations: +, positive ultrasonography finding; −, negative ultrasonography finding; +/−, unequivocal ultrasonography finding; nr, not reported.

Table 2
Results of MRI assessment in symptomatic and asymptomatic patellar tendons

	Total tendons	Symptomatic tendons				Asymptomatic tendons			
		Total	+	−	+/−	Total	+	−	+/−
Davies et al [27]	16	16	14	0	2	0			
Johnson et al [32]	38	24	19	5		14	0	14	
Khan et al [10]	63	28	28	0		35	nr	nr	
Major and Helms [60]	34	0				34	8	26	
McLoughlin et al [64]	15	15	15	0		0			
Popp et al [19]	11	11	11	0		0			
Schmid et al [35]	51	19	18	1		32	23	9	
Shalaby and Almekinders [34]	29	12	7	3	2	17	4	5	8
Yu et al [14]	11	11	11	0		0			
Summary	252	120	109	9	2	132	35	54	0

Calculations
Sensitivity = 78%
Specificity = 86%
Positive predictive value (PV+) = 93%
Negative predictive value (PV−) = 61%
Abbreviations: +, positive ultrasonography finding; −, negative ultrasonography finding; +/−, unequivocal ultrasonography finding; nr, not reported.

In addition to being unable to reflect clinical symptoms, ultrasonographic and MRI appearances cannot be used to distinguish outcome following intervention for tendinopathy. Ultrasound images remain both qualitatively and quantitatively abnormal 12 months after surgery, even in athletes who have returned pain-free to full competition [24]. With longer follow-up after surgery, no correlation exists between the area of the hypoechoic region and either function or time after surgery [57]. In terms of MRI, tendon appearance does not return to normal after successful surgery, and thus it is not able to distinguish patients whose surgical outcome was excellent from those whose outcome was poor [24]. Consequently, imaging does not appear to have a major role to play in monitoring outcomes following intervention for tendinopathy.

As tendons can have asymptomatic lesions on imaging, and these lesions in symptomatic tendons have been shown to be an area of histological degeneration, it is reasonable to question whether imaging can be used to predict future prognosis. At this stage this is not completely clear, although based on current longitudinal data using ultrasonography a trend does appear (Table 3). In one study, a 4.2 times greater risk of developing symptoms was identified in asymptomatic tendons with imaging abnormalities than in those without such abnormalities [51]. Because asymptomatic tendinosis can lead to spontaneous tendon rupture [23], the relevance of asymptomatic lesions on imaging needs further investigation.

As imaging does not reflect symptoms or indicate outcome, it remains a supplemental aid to clinical examination in the assessment of patellar tendinopathy. Imaging is very sensitive to abnormal tendon morphology [24]. Given the

Table 3
Ability of ultrasonography to predict future prognosis

Reference	Total tendons	Follow-up time	Ultrasonography at baseline	Clinically symptomatic at follow-up
Cook et al [51]	52	16 months	10 abnormal	3
			42 normal	3
Cook et al [66]	46	4 years	18 abnormal	4
			28 normal	2
Fredberg and Bolvig [67]	98	11 months	18 abnormal	3
			80 normal	0
Khan et al [70]	46	>12 months	23 abnormal	8
			23 normal	2
		Totals	69 abnormal	18 (26%)
			173 normal	7 (4%)

high positive-predictive value of imaging (Tables 1,2), it is of use in patellar tendinopathy, as it can confirm the clinical diagnosis and increase the overall likelihood of diagnosis. If imaging reveals characteristic features of patellar tendinopathy that fit the clinical presentation, then treat as such; however, if imaging is normal, then other causes of anterior knee pain need to be further considered. Imaging should not be used to determine management, and it does not appear to have a major role postoperatively. Whether imaging findings in asymptomatic tendons are predictive of future prognosis should be a focus of further research.

Conservative treatment

Despite the morbidity associated with chronic patellar tendinopathy, there is a surprising lack of scientific evidence directing the management of this condition. This lack of evidence results from a dearth of methodologically sound, randomized-controlled trials of clinically implemented treatments, and has resulted in vastly contrasting treatment choices among clinicians. In spite of this, what is currently agreed upon is that initial management should be conservative rather than surgical. This reasoning is based on the facts that the time course of recovery with appropriate conservative management is equivalent to that following surgery, and that the outcome of conservative management is equal to, if not better than, that following surgery [2].

For appropriate conservative management, the aforementioned underlying pathology of patellar tendinopathy needs to be understood by both the treating clinician and athlete. The pathology is degenerative by nature and this degeneration was most likely taking place before the onset of symptoms. This means that the pathology is typically quite advanced before clinical presentation. The advanced degeneration before the onset of symptoms, combined with the slow metabolic rate of tendon, means that recovery can be prolonged. In chronic cases, this recovery

can take in the vicinity of 4 to 6 months [2]. In athletes with a short duration of symptoms, recovery to full sporting capacity may take 2 to 3 months [2]. It is with these latter athletes that special care needs to be taken, as they may be able to warm up the injury, enabling full sporting capacity, further mechanical overload, and further tendon degeneration.

The focus of any conservative management program should be to deload the tendon and to encourage collagen synthesis, maturation, and strength [2]. At all times, progression through the program should be directed by the athlete's symptoms. Common reasons why conservative treatment programs fail are too rapid a progression through rehabilitation, lack of monitoring of an athlete's symptoms during and after therapy, and inappropriate loads [45].

Relative load reduction and biomechanical correction

As patellar tendinopathy is predominantly a strain-related phenomenon, initial conservative management should involve some form of load reduction to limit progression of the pathology. Given the detrimental effects of complete immobilization, load reduction should be achieved by relative rest rather than complete cessation of activity. Relative rest means that the athlete may be able to continue playing or training, if it is possible to reduce the amount of loading through modification of pain-provoking activities and reduction in total training hours.

In addition to changing training activities and durations, patellar tendon loading may be reduced through biomechanical correction. Correcting biomechanics of the lower limb kinetic chain can improve its energy-absorbing capacity and redistribute forces from the knee and patellar tendon. Biomechanical correction can be as simple as training how to land so that greater load is absorbed by distal and proximal joints. When landing, the ankle and calf are critical in absorbing the initial load and reducing load being transmitted to the knee [71]. Approximately 40% of landing energy is transmitted proximally, and thus a functioning calf complex is required to absorb the major portion of the initial load [45]. Similarly, functioning of the hip complex is important. When a large range of hip flexion is combined with forefoot landing, vertical ground reaction forces can be further reduced [72].

To assist distal and proximal joints in absorbing more load, correction of anatomical and functional abnormalities may be needed. Inflexibility of the quadriceps, hamstrings, iliotibial band, or calf has the potential to restrict range-of-motion at the knee and ankle, and to increase the load on the patellar tendon. Similarly, weakness of the gluteal, lower abdominal, quadriceps, and calf muscles may lead to fatigue-induced aberrant movement patterns that may alter forces acting on the knee [45]. Forces on the knee may also be influenced by foot mechanics, and thus shoe orthoses may be indicated in some athletes.

Strengthening exercises

Although mechanical loading is implicated in the etiology of patellar tendinopathy, loading is known to be beneficial to tendon health, and strengthening

exercises are recommended in its management. Exercise may influence the structure, chemical composition, and mechanical properties of a tendon [73]. Animal studies have shown loading of tendon improves collagen alignment and stimulates collagen cross-linkage formation [74]. In tendinopathy, mechanical loading may speed repair, as it increases tenocyte metabolism [75].

Clinical studies point to the efficacy of eccentric strengthening regimes in the treatment of tendinopathies [76–78]. This has been supported by recent scientific evidence [79,80], although studies in patellar tendinopathy are currently lacking [9]. Initial exercises should focus on strength and endurance gains before progressing to speed gains. Both pain and the ability of the musculotendinous unit to do work should guide the amount of strengthening activity, and during all exercises quality of movement should be emphasized.

Pharmacological intervention

Anti-inflammatory agents are the most common pharmacological interventions in patellar tendinopathy, with the two most common agents being oral nonsteroidal anti-inflammatory drugs (NSAIDs) and local injection of corticosteroids. The use of both has been debated, considering that tendinopathy has a noninflammatory pathology. In a thorough review of the role of NSAIDs in the treatment of tendinopathy, Almekinders and Temple [81] found little evidence that they were helpful. In terms of corticosteroid injection directly into the tendon tissue, it has been found to inhibit collagen synthesis [82] and lead to cell death and tendon atrophy [83], and a reduction in load-to-failure [84].

Although inflammatory cells do not appear to be present in patellar tendinopathy, inflammatory pathways may still be involved, and thus anti-inflammatory agents may have a role in management. This potential role needs to be further explored. It is possible that NSAIDs benefit tendinopathy via alternate mechanisms, such as accelerated formation of cross-linkages between collagen fibers [85,86]. Similarly, corticosteroids, when injected peritendinous rather than intratendinous, could possess beneficial effects, mediated through effects on the connective tissue and peritendinous adhesions by inhibiting the production of collagen, other extracellular matrix molecules, and granulation tissue [87].

Electrophysical modalities

A range of electrophysical modalities have been employed to treat patellar tendinopathy. These include ultrasound, laser, and electrical stimulation. Currently there is only circumstantial evidence supporting the use of these modalities and further research is required. Ultrasound can stimulate in-vitro collagen production from fibroblasts [88,89], and increases mechanical strength return during repair of acute tendon injuries [90,91]. Laser has been shown in a rabbit Achilles tenotomy model to increase collagen content [92]. Biomechanical and biochemical measures of tendon healing were improved by a combination of ultrasound, laser, and electrical stimulation of rabbit Achilles tendons after tenotomy and suture repair

[93]. Whether beneficial effects of these modalities are present in degenerative patellar tendinopathy and in humans has not been investigated.

Cryotherapy

Icing may have a role in the management of patellar tendinopathy, particularly when applied post-loading. Icing reduces blood flow and may help to reduce the pathological neovascularization associated with tendinopathy. Whether it also reduces tenocyte collagen production requires consideration. Icing may also be used as an analgesic; however, this role should only be used following exercise, as it may mask symptoms enabling tissue overload [13].

Massage

Massage therapy is used in patellar tendinopathy to promote repair and to decrease adhesions between the tendon fibers [29]. Research in healing rodent tendons showed soft-tissue mobilization to increase fibroblast recruitment and promote healing [94,95]. Clinically, in tendinopathy the most effective form of massage appears to be digital ischemic pressure followed by deep transverse friction throughout the entire tendon. Massage should also be performed on both the calf and quadriceps muscles to maintain their compliance [29,45]. This may take the form of sustained myofascial tension.

Surgical treatment

Surgery for patellar tendinopathy is only indicated after a prolonged (6 months) and well-supervised conservative treatment program fails. Surgery may involve excision of degenerated areas, arthroscopic debridement, repair of macroscopic defects, multiple longitudinal tenotomies, drilling of the inferior pole of the patella, resection of the tibial attachment of the patellar tendon with realignment, percutaneous needling, or percutaneous longitudinal tenotomy [29,41]. As the pathophysiology of patellar tendinopathy is not known, the exact surgical technique chosen is based on the surgeon's opinion and experience [29]. There is no consensus as to the optimal surgical technique to use.

Surgery is not indicated in the initial management of patellar tendinopathy, as surgical outcomes are rather unpredictable and recovery can be extended. A review of 23 papers found that the outcome following surgery was either excellent or good in 46% to 100% of cases, with an overall success rate of 75% to 85% being a very-best-case estimate [96]. Thus, 15% to 25% of patients will experience persistent or recurrent tendon pain following surgery. The recovery following surgery, even with a good or excellent result, can take 6 to 12 months [40,57], and many athletes will not be able to return to their previous level of sport [97]. Consequently, surgery should only be considered after a thorough, high-quality, conservative management program has been attempted.

Summary

Patellar tendinopathy is a common and serious condition in athletes. Although there have been many advances in the understanding of the histopathology, imaging, and surgical outcomes in this condition in the past decade, successful management of athletes with patellar tendinopathy remains a major challenge for both the practitioner and patient. There is a definite need for further prospective studies into etiological factors and randomized controlled trials into treatment choices.

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