Chapter 1

Tendinopathy: A Major Medical Problem in Sport

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Tendinopathy is a major medical problem associated with sports and physical activity in active people over 25 years of age. It can be defined as a syndrome of tendon pain, localized tenderness, and swelling that impairs performance. The clinical diagnosis is determined mainly through the history, although the exact relationship between symptoms and pathology remains unknown. In chronic tendinopathy, there is an increasing degree of degeneration with little or no inflammation present. Increasing age results in decline of the ultimate load of the muscle–tendon–bone complex, ultimate strain, as well as modulus of elasticity and tensile strength of the tendon.

The management of tendinopathy is often based on a trial and error basis. Controlled motion and exercise are the cornerstone of treatment, as human tendon tissue responds to mechanical loading both with a rise in metabolic and circulatory activity as well as with an increase in extracellular matrix synthesis. These changes contribute to the training-induced adaptation in biomechanical properties whereby resistance to loading is altered, tolerance towards strenuous exercise can be improved, and further injury avoided. Using eccentric exercise has been shown to be effective. Extracorporal shock wave therapy and sclerosing therapy are fairly recent and promising techniques but more research is needed. Surgery is also an option but return to sports after surgery is reported in only 60–85% of cases. It should be noted that those reports with higher success rates following surgery are associated with a poor overall methodologic scoring system.

The treatment of tendinopathy is difficult and can be frustrating. Recovery takes time as there is no quick fix. The patient's return to sports after surgery may take a long time to avoid re-injury. Thus, tendinopathy remains one of the most challenging areas in orthopedic sports medicine. In this chapter, we review topics in tendinopathy including the incidence of injury in sports, factors associated with its development, pathology, diagnosis, and clinical management.

Introduction

Tendinopathy, a major medical problem associated with sports and physical activity, is a generic descriptor of the clinical conditions in and around tendons resulting from overload and overuse. As it is difficult to determine the pathologic changes for most patients, investigators today use a classification system based on pain and function. Tendinopathy can be defined as a syndrome of tendon pain, localized tenderness, and swelling that impair performance. This condition is aggravated by additional physical and sports activity.

The physiology of tendinopathy remains largely unknown. The condition may be called tendinosis, which is defined as intratendinous degeneration (i.e. hypoxic, mucoid or myxoid, hyaline, fatty, fibrinod, calcific, or some combination of these) resulting from a variety of causes such as ageing, microtrauma, and vascular compromise (Jósza & Kannus 1997). It is often detected when the clinical examination is supplemented with magnetic resonance imaging (MRI) and/or ultrasonography which shows changes in the tendon tissue. The syndrome

can include inflammatory changes in the tendon sheath and terms such as tenosynovitis, paratenonitis, and peritendinitis have been used. Others further suggest that the terms tendinosis and paratendinitis should not be used until after histologic examination (Maffulli *et al*. 1998).

Diagnosis is mostly determined through the history, although the exact relationship between symptoms and pathology remains unknown (Curwin & Stanish 1984). Pain during activity is the cardinal symptom in tendinopathy and swelling is often present. Tenderness on palpation will support the diagnosis. MRI and ultrasonography can be used to verify the diagnosis.

The most well-known problems are Achilles tendinopathy, patellar tendinopathy, quadriceps and hamstrings tendinopathy, rotator cuff tendinopathy, tennis and golf elbow epicondylitis, as well as paratendinopathy in the wrist and hand (de Quervain disease). These issues are discussed in this book. The findings can, to a large extent, be applied to most chronic tendon problems around the ankle, knee, hip, wrist, elbow, and shoulder joints.

Tendon has a unique structure. Its parallel collagen fibers are suitable for the tendon to resist high tensile load without excessive elongation. The muscle–tendon–bone complex is involved in almost every human motion. Some of the strongest tissues in the body are the Achilles and the patellar tendons. Nevertheless, these tendons can still be frequently injured and the unanswered question is why are they so prone to injury.

Biomechanically, tendons are subjected to very high loads—thus causing tendons to be injured and result in pain that could lead to chronic pathology. Repair and remodeling are unusually slow and are not evident in great amounts. In addition, the injury takes a long time to heal. In acute tendon injury, however, repair tissues are present in the healing process. Biomechanical changes include a decrease in ultimate load and strain, decrease in tensile strength, and changes in collagen cross-links (Józsa & Kannus 1997).

Tendinopathy is to a large extent a work and sports-induced problem. In the initial phase of tendon injury, there is often inflammation present. However, in a more chronic setting, there is an increasing degree of degeneration, especially with increasing age, with little or no inflammation. Most inflammatory changes take place in the paratenon and are accompanied by areas of focal degeneration within the tendon (Backman *et al*. 1990). It would be uncommon for tendon degeneration to occur without some inflammatory changes around the tendon.

The etiology and pathology are still not well known and therefore the management of tendinopathy is often based on a trial and error basis. The treatment can vary from country to country, from clinic to clinic, and from clinician to clinician (Kannus & Józsa 1997).

Tendon and sport

Tendinopathy is thought to follow frequent actions involving quick accelerations and decelerations, eccentric activities, and quick cutting actions. Thus, the location of such injuries is sports specific.

Achilles tendinopathy can occur in sports activities such as running, jumping, or in team sports, such as football and team handball, as well as in racquet sports, such as tennis and badminton. The incidence in tennis players is 2–4% and 9% in dancers (Winge *et al*. 1989). The incidence in nonathletes is 25–30% (Åström 1997). However, for a long time, Achilles tendinopathy had been considered to be a running injury, with a high incidence, 6–18% of all injuries, especially during the running boom during the 1980s (Clement *et al*. 1984). The incidence seems to have decreased during the last 15 years, partly because of increasing awareness and prevention, but also perhaps a result of the improved quality and design of footwear. Still, there is a yearly incidence of 24–64% in runners (Hoeberigs 1992; van Mechelen 1995) while the prevalence in middle and long-distance runners is 7–11% (James *et al*. 1978; Lysholm & Wiklande 1987; Winge *et al*. 1989; Teitz 1997). The rate of Achilles tendinopathy among orienteering runners was 29% compared with 4% in the control group (Kujala *et al*. 1999).

Thirty percent of patients with Achilles tendinopathy have bilateral problems (Paavola *et al*. 2002b). In most cases, the problems are located to the medial side of the Achilles tendon while the lateral side is involved in every fifth case (Gibbon 2000).

Patellar tendinopathies are often associated with jumping sports such as basketball, volleyball, tennis, and high jump, as well as ice-hockey, football, downhill running, and weight lifting. Tennis players, baseball players, and golfers may develop lateral and medial elbow epicondylitis. Hamstring syndromes involving the proximal tendons are common in athletics. Rotator cuff tendinopathy occurs in throwing sports such as baseball, javelin, and team handball, as well as in volleyball, tennis, and gymnastics.

Known factors associated with tendinopathy

Many factors contribute to the development of tendinopathy: extrinsic factors, such as training errors and environmental conditions, as well as intrinsic factors, such as malalignment. In addition, increasing age as well as gender are also overall factors that can influence the development of tendinopathy.

Extrinsic factors

Tendinopathy can be caused by an interaction of extrinsic factors including training errors, such as excessive distance, intensity, hill work, technique, and fatigue, as well as playing surface, which seems to be predominant in acute injuries. Clinically, it is known that a change in load, training errors (linked to a change in load), changes in the environment, and changes or faults with equipment such as racquets can result in an onset of tendon symptoms (Jósza & Kannus 1997). Environmental conditions such as cold weather during outdoor training can also be a risk factor (Milgrom 2003). Footwear and equipment may also play a part. However, there is little evidence to support other extrinsic factors and further research is required.

Intrinsic factors

Intrinsic factors related to Achilles tendinopathy include the malalignment of the lower extremity, which occurs in 60% of Achilles disorders. There is an increased forefoot pronation (Nigg 2001), limited mobility of the subtalar joint, decrease of range of motion of the ankle (Kvist 1991), varus deformity of the forefoot, increased hind foot inversion, and decreased dorsiflexion with the knee in extension (Kaufmann *et al*. 1999). Other factors that have been discussed include decreased flexibility, leg length discrepancy, muscle weakness, or imbalance. Åström (1997) concluded in his PhD thesis that malalignments may not be of clinical importance. Biomechanical abnormalities may still be corrected but the clinical outcome has yet to be validated. We should remember that there is a lack of high-quality prospective studies, which limit the conclusions that can be drawn regarding these factors.

Age

Even though the rate of tendon degeneration with age can be reduced by regular activity (Józsa & Kannus 1997), age is indeed a factor with respect to tendon injury. In patients less than 18 years of age, the problems are usually located in the muscle tendon junction or tendon insertion to bone. Younger tendons are smaller and can withstand more stress than older tendons (Ker 2002). Between 18 and 55 years of age there is an increasing incidence of tendinopathy in Achilles and patellar tendinopathy with clinical problems especially after 30–35 years of age. Patellar tendinopathy seems to have a similar prevalence in adolescents as adult athletes (Cook *et al*. 2000a). Shoulder injuries, especially rotator cuff problems, are increasingly frequent with increasing age especially in patients over 55 years of age. They account for 18% of cases.

Increasing age also results in changes in the structure and function of human tendons as the collagen fibers increase in diameter and decrease in tensile strength. The ultimate strain, ultimate load, modulus of elasticity, and tensile strength of the tendon also declines (Kannus & Józsa 1991). The most characterized age-related changes are degeneration of the tenocytes and collagen fibers and accumulation of lipids and calcium deposits (Kannus & Józsa 1991). Well-structured, long-term exercise appears to have a beneficial effect and minimize the negative effects on aging tendons (Tuite *et al*. 1997).

Gender

Achilles tendinopathy seems equally common in men and women (Mafi *et al*. 2001; Mafulli *et al*. 2005). However, larger cohort studies have found that there may be a decreased risk for females to develop patellar tendinopathy (Cook *et al*. 2000a).

In running, 60% of injuries occur in men. Many of these injuries involve tendinopathy and related issues. Women under 30 years are at the highest risk (Järvinen 1992). However, some studies have found no decreased risk of tendinopathy in females compared to males.

Work-induced tendinopathy, especially involving repetitive overloading of the muscle–tendon unit, is also common. This is characterized by being present in over 60% of women with a mean age of 41 years. The upper limb is involved in 93% of the cases of which 63% occur in the forearm and 24% is elbow epicondylitis. The remaining problems come from the neck, back, and lower limbs (Kivi 1984).

Pathology

In chronic tendinopathy, science has shown minimal or no inflammation (Arner *et al*. 1959; Puddu *et al*. 1976). This was verified by Alfredson *et al*. (1999), who found no physiologic increase in prostataglandin E_2 in the injured human Achilles tendon. Animal models also suggest that inflammation does not occur in the overused tendinopathy process. However, it is important to know that the absence of inflammatory mediators in end-stage disease does not mean that they were not present in early-stage disease. These mediators may still be a factor in the cause of tendinopathy. Mechanical load can increase the tendinosus and peritendinous levels of prostataglandins (Almekinders *et al*. 1993). Thus, inflammation could be associated with tendinopathy at some point in time.

Tendon impingement could also be a contributing factor to tendon pathology. For example, between the Achilles tendon and the calcaneal bone there is a retrocalcaneal bursa, which can be impinged between the Achilles tendon and the bony posterior aspect of the calcaneus. In some cases with bony prominence, impingement is likely, with resulting bursitis and tendinopathy. Another example of impingement may involve patellar tendinopathy (Johnson *et al*. 1996). However, others have found that the superficial attachment of the proximal patellar tendon is far stronger than its depth would indicate. There is pain in the early phases of landing with the knee in an extended position. Pain increases on palpation with the knee in full extension, making it less likely that such impingement could be a major contributing factor.

Where is the pain coming from in tendinopathy?

What causes tendon pain? Tendon degeneration with mechanical breakdown of collagen could theoretically explain the pain mechanism but clinical and surgical observations challenge this view (Khan *et al*. 2003). Structural changes are unlikely to be the main cause. Recently, the combination of biomechanical and biochemical changes has become a more plausible explanation for the cause of pain. Preliminary evidence shows that unidentified biochemical noxious compounds such as glutamate, substance P, or calcitonin gene-related peptide (CGRP) may be involved (Ackermann 2003; Alfredson 1999; Danielson *et al*. 2006).

Neural factors such as sensory neuropeptides show that tendinous tissues are supplied with a complex network of neuronal mediators involved in the regulation of nociception vasoactivity and inflammation (Ackermann *et al*. 2003). In response to injury, the expression of neuropeptides is significantly altered, suggesting a synchronized mechanism in nociception and tissue repair. Further studies must be performed to determine if the neuronal pathways of tendon healing can be stimulated by specific rehabilitation programs or by external delivery of mediators and/or nerve growth factors.

Diagnosis of tendinopathy

In most cases, the patient's history should secure a correct diagnosis. Pain with activity, often combined with impaired performance, is the key component. The catchwords "Too much, too soon, too often" are often heard in the patient's history. Careful clinical examination will verify the diagnosis as the pain is often associated with localized tenderness and swelling.

Robinson *et al*. (2001) has developed a questionnaire, the Victorian Institute of Sports Assessment—Achilles questionnaire (VISA-A score), which can be used to evaluate the clinical severity of Achilles tendinopathy. This has also been used to evaluate the outcome of treatment (Peers *et al*. 2003) and can provide a guideline for treatment as well as for monitoring the effect of treatment (Silbernagel *et al*. 2005). This score is also useful for patellar tendinopathy (Frohm *et al*. 2004).

Ultrasonography is a valuable but not perfect tool for the diagnosis and evaluation of tendinopathy, particularly for patellar tendinopathy (Cook *et al*. 2000a). Ultrasonography is a very helpful guiding tool when minor biopsies are to be taken from the tendon tissue. MRI detects abnormal tissue with greater sensitivity than ultrasound (Movin 1998). In a recent observation, both ultrasound and MRI showed a moderate correlation with clinical abnormality at baseline (Peers *et al*. 2003). In Achilles tendinopathy, MRI was associated with clinical findings. However, it should be mentioned that tendon imaging findings can still be asymptomatic. Therefore, MRI must always be correlated to the clinical symptoms.

Management of tendinopathy—what do we know?

As the exact etiology, pathophysiology, and healing mechanisms of tendinopathy are not well known, it is difficult to prescribe a proper treatment regimen. So far, the given treatments are frequently based on empirical evidence and so recommended treatment strategies by different clinicians can vary greatly (Paavola *et al*. 2002b). Nevertheless, the first course would be to identify and remove those external factors and forces, especially excessive training regimens. Also, intrinsic factors, such as lack of flexibility, increased foot pronation, and impingement, should also be corrected.

Exercise—key to success in prevention and management

Exercise is an important factor in the prevention of tendinopathy but it has emerged also as a successful factor in the management of tendinopathy. Exercise combined with specified loading conditions will make the tendon more resistant to injury. Human tendon tissue responds to mechanical loading both with a rise in metabolic and circulatory activity as well as with an increase in extracellular matrix synthesis. These changes contribute to the training-induced adaptation in mechanical properties whereby resistance to loading is altered and tolerance towards strenuous exercise can be improved and injury avoided (Kjaer 2004). In other words, exercise during healing of an injury stimulates faster healing (Jósza *et al*. 1992).

The concept of using eccentric exercise in tendinopathy was introduced by Stanish and Curwin in 1984 and has been shown to function well in a number of studies (Curwin & Stanish 1984; Stanish *et al*. 1986; Fyfe & Stanish 1992; Alfredson & Lorentzon 2003). It has been shown to be superior to concentric training (Niesen-Vertommen *et al*. 1992) and effective in prospective randomized studies (Alfredson *et al*. 1998; Mafi *et al*. 2001; Alfredson & Lorentzon 2003). More than 80% of patients were satisfied with the treatment and could return to previous activity levels. However, the results for insertional injuries were not satisfactory. Curwin and Stanish (1984) stressed careful attention to the level of pain as a high level of pain or pain too early in the exercise program could cause worsening of the symptoms.

There was increased collagen synthesis and cross-sectional area after a session with eccentric exercise (Magnusson & Kjaer 2003; Crameri *et al*. 2004). An ongoing study indicated that weekly overload eccentric exercises, using weights up to 300 kg in a specifically constructed machine called Bromsman, might be effective in the management of tendinopathy (Frohm, personal communication). These studies support the results from previous studies by Woo *et al*. (1982) which showed prolonged exercise is effective and results in an increase in the cross-sectional area of extensor tendons as well as its mechanical properties.

A well-defined exercise regimen should be the cornerstone of treatment of tendinopathy. Knowledge of the tendon in response to tensile loading can be used successfully to manage most tendon overuse injuries (Stanish *et al*. 1986; Magnusson & Kjaer 2003). However, it should be pointed out

that exercise alone does not solve all cases of tendinopathy.

Patients with long-standing Achilles tendinopathy may be instructed to continue Achilles tendon loading activity with the use of a pain monitoring model during the treatment (Silbernagel 2006). This model may help the patient as well as the clinician to handle the pain and determine how the exercises should progress.

Physical therapy

Physical therapy is important. Regular attention to the problem in combination with hands-on techniques enables the patient to feel more relaxed and better as a whole. Some rehabilitation techniques such as eccentric training can be very difficult for the patient to perform properly without supervision. Although the scientific evidence is limited and in some cases controversial, modalities such as heat, ultrasound, electrical stimulation, laser stimulation, and acupuncture may be effective. Systematic reviews of the effect of ultrasound to treat soft tissues in general showed little efficacy except in the treatment of calcific tendinopathy and lateral epicondylitis (van der Windt *et al*. 1999).

Recently, some newer treatment techniques have been introduced. Extracorporal shock wave therapy has been reported effective (Rompe *et al*. 1996) and shows a comparable outcome to surgery of patellar tendinopathy (Peers *et al*. 2003). Sclerosing therapy focusing on destroying the new vessels and accompanying nerves showed very promising short-term clinical results (Alfredson & Öhberg 2004). As a whole, much more research is needed before these new procedures can be recommended routinely.

Corticosteroid injections

Corticosteroid injections are designed to suppress inflammation. As tendinopathy is not an inflammatory condition, the value of injection may be to reduce adhesions between the tendon and sheath as well as to medicate pain in the short term. However, the adverse effects of corticosteroids may be recognized and caution is needed. A corticosteroid injection may inhibit collagen synthesis and delay tendon healing. Partial ruptures are also common (Åström 1998). In addition, peritendinous injections may also have deleterious effects (Paavola *et al*. 2002). In conclusion, it is fair to say that the biologic basis of the effect of corticoid injections and benefits are largely lacking. Above all, one should avoid injections into the tendon substance.

Surgical management

It should be remembered that a surgical incision, in itself, results in a strong healing response. Clinicians have suggested needling, coblation, percutateous tenotomy, arthroscopic debridement, percutaneous paratenon stripping, open tenotomy, paratenon stripping, and tendon grafting (Maffulli *et al*. 2005). However, the surgical technique as such in open surgery is probably not very important in the management of tendinopathies. Furthermore, endoscopic arthroscopic techniques have seemed to gradually take over.

As the physiologic, biomechanical, and biologic base of the effect of surgery are not clearly understood, it is not possible to establish the relationship between operative treatment and the healing and reparative process. In general, a return to sports after surgery has been reported in 60–85% of cases. Again, we caution that the higher the reported success rates of surgery, the worse the overall methodologic score in published paper (Tallon *et al*. 2001).

Healing and return to sport take time

Patients' return to sports following surgical management of tendinopathy may take a long time. The rule of thumb for their return has been no increase of symptoms. First time ailments may require 2–3 months to recover but chronic cases may take 4–6 months in the case of Achilles tendinopathy, 6–8 months for patellar tendinopathy, and 8–12 months for rotator cuff tendinopathy.

So, why does it take such a long time? As tendons have limited vascularity and a slow metabolic rate, the tenocytes that produce collagen have a very slow turnover rate of 50–100 days (Peacock 1959). Healing and remodeling of the Achilles tendon of the rabbit requires 4 months (Williams 1986) and

complete regeneration of the tendon is never achieved. If a tendon is given inadequate time to repair, tenocytes may undergo apotosis because of excessive strain. So, the message here is that even though the athlete would like to return to their sport quickly, the clinician must advocate that time for healing be allowed in order to avoid re-injury. Recovery from tendinopathy often takes time and there is no quick fix. Such treatment regimens often lead to frustration and re-injury. The use of controlled exercise programs can be effective but a clear understanding of the mechanisms is largely lacking.

Future directions

Aging and exercise influence the degenerative process, which has an important role in the development of tendinopathy. However, regular exercise appears to have a beneficial effect on aging tendons and may delay the degenerative changes. Therefore, more studies need to be performed in order to define a quantitative relationship: what would be the appropriate amount of usage and at what stress levels for the maintenance of homeostasis. Meanwhile, it is also important to recognize the large differences in the development of tendinopathies between normal and diseased tendons. Because these tendinopathies come from different origins, they do not have the same cell phenotypes and will respond to the mechanical and biologic milieu differently (Fig. 1.1). Therefore, different strategies are needed for the appropriate management of these tendinopathies. As such, appropriate strategies for management and maintenance of tendon homeostasis and tendon remodeling must be developed to avoid tendinopathy.

Many athletes are at risk for developing tendinopathy. Activities such as basketball, triple jump, tennis, badminton, and running continue to be the main sports that increase risk. Downhill eccentric activities can also be a problem. With increasing demands, intensified training, and more frequent athletic contests, it is vital to analyze these sports and activity schedules in order to design preventive programs.

Exercise and load are the key parameters for an ideal and effective treatment. Too much load and activity too early may generate pain and more severe injury as well as significantly delay the healing process. During passive static stretching, the mechanical properties of the muscle–tendon unit are affected during the actual stretch maneuver and stress relaxation occurs. However, this mechanical effect appears to disappear rapidly within minutes. Stretching produces gains in maximal joint range of motion and habitual stretching may improve maximal muscle strength and the height of a jump. The currently available evidence does not support the notion that stretching prior to exercise can effectively reduce risk of injury. However, stretching includes exercise and load, and may favorably affect the homeostatis of tendons. As such, the effect of stretching on tendons needs to be further studied.

Tendon healing after injury will not result in a normal tendon. The results may be often functionally

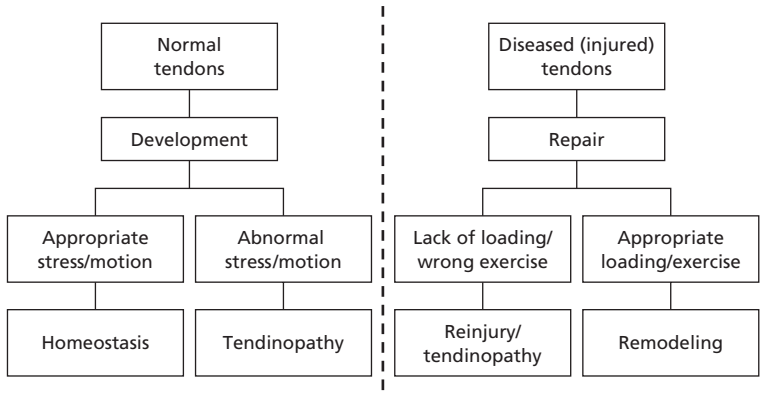

Fig. 1.1 The different pathways for the development of tendinopathies between normal and diseased tendons. As such, different strategies are needed for the appropriate management of these tendinopathies as the cell phenotypes as well as the biologic and mechanical milieu are not the same.

satisfactory despite its biomechanical weakness and morphologic inferiority. The effect of growth factors and gene therapy with the aim to "normalize" the healing tendon are intriguing and seem promising but are far from being ready for clinical use. However, there are exciting possibilities on prevention of tendon degeneration as well as promoting tendon regeneration after injury.

Tendinopathy is without doubt one of the most difficult areas in orthopedic sports medicine. The research on this common problem needs to be intensified in the coming years. This IOC workshop was therefore an important catalyst as it allowed many front line researchers to get together, share their knowledge, and discuss future directions that can lead to solving the enigma of tendinopathy.

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