Review article

TENDINOPATHY OF TENDO ACHILLIS

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In the past three decades, the incidence of overuse injury has risen because of greater participation in recreational and competitive sporting activities.¹ Excessive repetitive overload of tendo Achillis is regarded as the main pathological stimulus which leads to tendinopathy.² In a recent study, however, 31% of 58 patients with this condition did not participate in vigorous physical activity.³

In this article, we have concentrated on tendinopathy of the main body of tendo Achillis. We have not dealt with Haglund's condition, insertional tendinopathy, or with lesions of the myotendinous junction.

Anatomy. The gastrocnemius muscle merges with the soleus to form tendo Achillis.⁴ It has a round upper part and is relatively flat in its distal 4 cm. Its fibres spiral through 90°, increasing the release of stored energy during locomotion.⁵

Tendo Achillis is enveloped by a paratenon, originating from the deep fascia of the leg, the fascia cruris. Blood flow in the tendon increases considerably with exercise, probably due to the marked rise in the negative tissue pressure in the peritendinous space.⁶

Histology. Tenocytes and tenoblasts comprise 90% to 95% of the cellular elements of the tendon. The extracellular matrix is composed of collagen and elastin fibres, ground substance such as proteoglycans and organic components such as calcium.⁷ The collagen fibrils are bundled into fascicles containing blood, lymphatic vessels and nerves, and have recently been shown to interchange between the fascicles.⁷ The latter, which are surrounded by the endotenon, group together to form the gross structure of the tendon. It is enveloped by a well-defined layer of connective tissue, the epitenon.

Biomechanics. Actin and myosin are present in the tenocytes.⁸ Tendons are stiff and resilient, with high tensile

strength.^{7,9} In the male, tendo Achillis has a larger cross-sectional area than in the female, with greater stiffness and ruptures at a higher maximum force. In younger individuals the tendon has a significantly higher tensile stress before rupture and lower stiffness.¹⁰ Loading can reach up to 9 kN during running, corresponding to 12.5 times the body-weight, 2.6 kN during slow walking, and less than 1 kN during cycling.¹¹⁻¹⁴

The tendon loses its wavy configuration when it is stretched by more than 2%. As the collagen fibres deform, they respond linearly to increasing loading.^{7,15} The normal wavy appearance of the tendon is regained if the strain placed on it remains at less than 4%. At levels of strain greater than 8%, macroscopic rupture will occur.^{7,9}

Aetiology of tendinopathy of tendo achillis

The aetiology remains unclear.^{1,2} Tendinopathies have been linked to overuse, poor vascularity, lack of flexibility, genetic make-up, gender, endocrine or metabolic factors¹⁶ and, recently, to the use of quinolone antibiotics.^{17,18}

Excessive loading of the tendon during vigorous physical training is regarded as the main pathological stimulus.¹⁹ The tendon may respond to repetitive overload beyond physiological threshold by either inflammation of its sheath or degeneration of its body, or by a combination of both.²⁰ Damage to the tendon can occur even if it is stressed within its physiological limits since frequent cumulative micro-trauma may not leave enough time for repair.¹² Micro-trauma can result from non-uniform stress within tendo Achillis as a result of different individual contributions in force from the gastrocnemius and the soleus, producing abnormal concentrations of load within the tendon, frictional forces between the fibrils, and localised damage to fibres.²¹

Tendinopathy has been attributed to a variety of intrinsic and extrinsic factors. Vascularity, dysfunction of the gastrocnemius-soleus, age, gender, body-weight and height, deformity of the pes cavus and lateral instability of the ankle are considered to be common intrinsic factors. Excessive movement of the hindfoot in the frontal plane, especially a lateral heel strike with excessive compensatory pronation, is thought to cause a 'whipping action' on tendo Achillis predisposing to tendinopathy.¹⁹ Marked forefoot

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varus is common in patients with Achilles tendinopathy.²² Changes in training pattern, poor technique, previous injuries, footwear and environmental factors such as training on hard, slippery or slanting surfaces are extrinsic factors which may predispose the athlete to tendinopathy.^{19,23}

Many factors are associated with the pathogenesis of a tendinopathy, including tissue hypoxia and consequent changes in the tendon induced by free radicals due to ischaemia-reperfusion injury,²⁴ and exercise-induced hyperthermia.²⁵ Furthermore, a tendon strained repeatedly to more than 4% of its original length loses elasticity, and is at an increased risk of a subsequent break in its collagen structure.⁷

Changes in expression of the genes regulating cell-cell and cell-matrix interactions have been described, with down-regulation of metalloprotease 3 mRNA in samples of the tendon. Levels of type-I and type-III collagen mRNA have been found to be significantly higher in the tendinopathic compared with 'normal' samples.²⁶

Using *in vivo* microdialysis, intratendinous measurements have shown elevation of the levels of glutamate in tendinopathy with no abnormal elevation of inflammatory prostaglandin PGE₂.^{27,28}

The term 'tendinosis' has been in use for more than two decades to describe collagen degeneration in tendinopathy.²⁹ Most clinicians, however, still use the term 'tendinitis', thus implying that the fundamental problem is inflammatory. We believe that 'tendinopathy' should be used to describe the clinical conditions in and around tendons arising from overuse,³⁰ using the terms 'tendinosis' and 'tendinitis' only after histopathological confirmation of the condition.³⁰

Pain

Classically, pain was attributed to inflammation. Athletic tendinopathies are degenerative non-inflammatory conditions and so the combination of mechanical and biochemical causes of pain has become more attractive.³¹ Degeneration of the tendon with mechanical breakdown of collagen could explain the pain, but clinical and surgical observations challenge this view.³¹ Chemical irritants and neurotransmitters may generate pain in tendinopathy. High concentrations of the neurotransmitter glutamate have been found in patients with tendinopathy of tendo Achillis.²⁷ Substance P and chondroitin sulphate may also be involved in producing the pain.³¹

Histology

Tendinosis can be viewed as a failure of the cell matrix to adapt to trauma because of an imbalance between degeneration of the matrix and its synthesis.³² Macroscopically, the affected portions of the tendon lose their normal glistening white appearance and become grey-brown and amorphous.

Fig. 1

Fusiform swelling in the tendon of a 38-year-old male hockey player with an eight-month history of tendinopathy resistant to conservative management. Note the classical location of the swelling.

Fig. 2

Photomicrograph of Achilles paratendinopathy in a 33-year-old male runner with a 12-month history of tendinopathy resistant to conservative management. The appearance is disordered and there is haphazard evidence of vascular ingrowth and hypercellularity. The normal, well-ordered appearance of the collagen fibres has been disrupted (haematoxylin and eosin $\times 120$).

Thickening can be diffuse, fusiform or nodular³¹ (Fig. 1). The paratenon can be involved in the early phases of tendinopathy, and may present as 'peritendinitis crepitans' because of adhesion between the tendon and the paratenon. Histologically, the major lesion in chronic tendinopathy "is a degenerative process characterised by a curious absence of inflammatory cells and a poor healing response",³³ with non-inflammatory intratendinous degeneration of collagen, disorientation and thinning of fibres, hypercellularity, scattered vascular ingrowth, and increased interfibrillar glycosaminoglycans^{7,16,32-34} (Fig. 2). Frank inflammatory lesions

and granulation tissue are infrequent, and, when found, are associated with rupture of the tendon.³⁵ An angioblastic reaction is present, with random orientation of blood vessels, sometimes at right angles to the collagen fibres.³⁶ There are at least six different subcategories of collagen degeneration,¹⁴ but that in tendo Achillis is usually either 'mucoid' or 'lipoid'.¹ In mucoid degeneration, the affected region softens, losing its normal glistening white appearance and becoming grey-brown. Light microscopy reveals collagen fibres which are thinner than normal, with large mucoid patches and vacuoles between the fibres. The amount of ground substance staining with Alcian Blue is increased. Lipoid degeneration describes an abnormal intratendinous accumulation of lipids,⁹ with loss of the characteristic hierarchical structure of collagen fibres.^{7,35}

Although there may be histological changes of tendinosis within the tendon, they are often clinically silent and only appear when rupture occurs. They may, however, coexist with symptomatic paratendinopathy.²³ The general pattern of intratendinous degeneration is common to ruptured and tendinopathic tendons, with a greater degree of degeneration in the former. It is therefore conceivable that there is a common, as yet unidentified, pathological mechanism which has acted on the tendons, causing tendinosis and the clinical picture of tendinopathy.³⁶

In the paratenon, mucoid degeneration, fibrosis and vascular proliferation with a slight inflammatory infiltrate have been described.^{23,37-44} Astrom and Rausing³³ found virtually no evidence of paratenonitis in their series of patients with tendinopathy of tendo Achillis. These differences may be explained by the fact that the studies of Kvist et al⁴²⁻⁴⁴ did not show pathology of the tendon itself and were performed in active, younger patients.

Experimental models

Although the condition is common, there are few experimental models for its study and treatment. Backman et al⁴⁵ produced a paratendinopathy in anaesthetised rabbits by prolonged repeated contractions of the triceps surae for up to six hours per session, three times per week, for up to six weeks by electrical stimulation producing movements of the ankle.

In three-month-old male rats, eccentric exercise of the triceps surae muscle (30 stimulations/min for three times per week for one hour per session) under general anaesthesia induced inflammation of the epi- and paratenon, but changes in the tendon corresponding to chronic tendinosis did not develop in spite of the regime being carried out for 11 weeks.⁴⁶

More recently, rats have been used to produce acute tendinopathy of tendo Achillis by direct trauma.⁴⁷ Again in rats, prolonged administration of a proinflammatory cytokine (PGE₁) resulted in diffuse involvement of the extracellular matrix with derangement and degradation of the collagen fibres, a picture of more generalised tendinosis.⁴⁸

Clinical aspects

The history and examination play a key role in the diagnosis and management. The onset of pain, its duration, and aggravating factors should be documented. Thorough enquiry should be made regarding the relationship of pain to various activities, the intensity of training and technique. Details of previous treatments are also important.

The tendinopathy typically presents with pain located 2 to 6 cm proximal to the insertion of the tendon and felt after exercise. As the pathological process progresses, pain may occur during exercise, and, when severe, may interfere with the activities of daily living.⁴⁹ Runners experience pain at the beginning and at the end of a training session, with a period of diminished discomfort in between.⁵⁰

Clinical examination should start by the exposure of both legs from above the knees, and the patient should be examined standing and prone. The foot and the heel should be inspected for malalignment, deformity, obvious asymmetry in the size of the tendon, localised thickening, a Haglund heel and any previous scars. The tendon should be palpated to detect tenderness, heat, thickening, nodularity and crepitation.⁵¹ Its excursion is assessed. The 'painful arc' sign helps to distinguish between lesions of the tendon and paratenon. In paratendinopathy, the area of maximum thickening and tenderness remains fixed in relation to the malleoli from full dorsi- to plantar flexion, whereas lesions within the tendon move with movement of the ankle.³⁹ However, the paratenon is not necessarily involved when a patient presents with a tendinopathy. In those with a more chronic condition, the endurance of the gastrosoleus complex may be reduced.⁵¹

Imaging

Plain soft-tissue radiography is useful in diagnosing associated or incidental bony abnormalities,⁵² but MRI and ultrasound are both very effective in detecting the changes in Achillis tendinopathy. MRI provides extensive information on the internal morphology of the tendon and the surrounding structures, and is useful in evaluating the various stages of chronic degeneration and in differentiating between peritendinitis and tendinosis. Areas of mucoid degeneration are shown on MRI as a zone of high signal intensity on T1- and T2-weighted images.^{53,54}

Ultrasound is used routinely in Europe. It promptly identifies hypoechoic areas, which have been shown at surgery to consist of degenerated tissue, and increased thickness of the tendon^{55,56} (Figs 3 and 4).

Because of the high sensitivity of these methods of imaging,⁵⁷ an abnormality should be interpreted with caution and correlated with the patient's symptoms before making any recommendations as to management.¹⁶ Ultrasound is regarded by many as the primary method since it correlates well with the histopathological findings despite being operator-dependent.³ MRI studies should be per-

Fig. 3

Fig. 4

Figures 3 and 4 – Ultrasonographic appearance of Achilles tendinopathy in a 28-year-old male soccer player at presentation. Figure 3 – The longitudinal scan shows that the affected tendon (right side of the figure) is thicker than the asymptomatic contralateral side. The normal, well-ordered distribution of the fibrils is lost. There is also thickening of the paratenon. Figure 4 – The transverse scan shows an area of intratendinous calcification. There are scattered focal hypoechoic areas present over the whole thickness of the tendon.

formed only if the ultrasound scan remains unclear. The combination of imaging and clinical diagnosis enhances the efficiency of preoperative planning.⁵³ One of the main advantages of ultrasound is its interactive facility, which helps to reproduce symptoms by transducer compression and concentrates on the abnormal area.⁵⁸ Recent ultrasound studies have shown mild to moderate changes in both involved and uninvolved tendons, but their occurrence was not clearly related to the patient's symptoms.^{59,60}

Management

The natural history of the condition is still unclear. Recovery is slow as a result of low oxygen consumption, slow synthesis of structural protein, and continuing excessive load.⁶¹

Conservative. Encouragement of athletes and coaches to follow a sensible training programme can probably prevent Achilles tendinopathy.⁶² Seeking medical attention at an early stage may improve the outcome since treatment becomes more complicated and less predictable when the condition becomes chronic.^{37,63} At present, management is more an art than a science.¹⁶ The efficacy of conservative measures is debatable. A favourable long-term prognosis has been reported with a comprehensive series of conservative measures including relative rest and the use of anti-inflammatory drugs, physiotherapy and orthoses.^{41,59,60,64} Nevertheless, some authors argue that conservative management of chronic tendinopathy of tendo Achillis can be time-consuming and often unsatisfactory.⁶⁵

Abstinence from the activities which caused the symptoms is recommended in the acute phases. In mild tendinopathy, relative rest or only modified activities are pre-

scribed.⁶⁶ Repair and remodelling of collagen fibres are stimulated by loading of the tendon and therefore complete rest of an injured tendon can be counterproductive.

Cyriax⁶⁷ advocated deep friction massage for tendinopathy and paratendinopathy. In chronic tendinopathy, this should be accompanied by stretching to restore tissue elasticity and to reduce the strain in the muscle-tendon unit with movement of the joint. Augmented soft-tissue mobilisation is a new non-invasive technique of soft-tissue treatment which has been successfully used in chronic tendinopathy, probably by controlled application of micro-trauma which increases proliferation of fibroblasts.⁶⁸

Stretching is traditionally performed in a quasistatic mode by pulling, holding and releasing the gastrocnemius-soleus complex using a wall, stair, or 20° inclined board.²³

Eccentric strengthening of the gastrocnemius-soleus muscle and loading of the tendon are important for both prevention and conservative management of tendinopathy.^{69,70} Gentle strength training should be started early after injury to prevent disuse atrophy, and should not be painful.²³

Correction by orthotics can alter the biomechanics of the foot and ankle and relieve heel pain.⁷¹ They are commonly used, especially in runners, with success in up to 75%.⁷²⁻⁷⁴ A heel lift of 12 to 15 mm is classically used as an adjunct to the management of pain in the tendon.⁶⁴ To our knowledge, no randomised controlled trials have been performed to study the efficacy of this simple measure.

Cryotherapy is used for its analgesic effect, to reduce the metabolic rate of the tendon and to decrease the extravasations of blood and protein from new capillaries found in tendon injuries.⁷⁵

Therapeutic ultrasound may reduce the swelling in the acute inflammatory phase and improve healing of the ten-

Fig. 5

The same patient as in Figure 4. At operation, a diffusely thickened fibrotic paratenon is seen, with an area of tendon nodularity extending for about 2 cm.

don.^{76,77} Ultrasound also stimulates the synthesis of collagen in tendon fibroblasts and cell division during periods of rapid cell proliferation.⁷⁸

Several drugs, such as low-dose heparin, wydase and aprotinin, have been used in the management of peri- and intratendinous pathology,⁷⁹⁻⁸¹ but evidence of their long-term effectiveness is still unclear. Peritendinous injections with corticosteroids are still controversial, evidence for their effectiveness is missing, and there are no good scientific reasons to support their use.^{72,82} Intratendinous injections of corticosteroids are to be avoided.⁸³

Operative. Conservative management is unsuccessful in 24% to 45.5% of patients with tendinopathy of tendo Achillis, and surgery is recommended after exhausting periods of conservative management, often lasting for at least six months.^{60,65} Long-standing tendinopathy, however, is associated with poor results after operation, with a greater rate of reoperation before an acceptable outcome is obtained.⁶³

There are minor variations in surgical technique.^{3,38,40,84,85} The objective is to excise fibrotic adhesions (Fig. 5), remove degenerated nodules and make multiple longitudinal incisions in the tendon to detect intratendinous lesions and to restore vascularity, possibly stimulating the remaining viable cells to initiate a response in the cell matrix and healing.^{1-3,20,37,41,84} The defect can either be sutured in a side-to-side fashion or left open, which is our usual practice. Reconstruction procedures may be required if large lesions are excised.⁸⁶ Recent investigations have shown that multiple longitudinal tenotomies trigger neoangiogenesis with increased blood flow.⁸⁷ This results in improved nutrition and a more favourable environment for healing.

Patients are encouraged to bear weight as soon as possible. Greater protection is recommended in patients requir-

ing reconstruction of the tendon. Sport-specific training is started at three months, and competition is resumed after six months.

Outcome of surgery. It is remarkable how, for a condition which is relatively common, most studies do not report their methods of assessment, which makes it difficult to compare the results.⁸⁸ Many authors describe excellent or good results in up to 85% of cases, with most articles giving a rate of success of over 70%,⁸⁸ but this is not always observed in routine non-specialised clinical practice.^{63,88}

In the most comprehensive study to date,⁵⁹ 432 consecutive patients were followed up for five months after surgery. There were 46 (11%) complications in these patients, 14 of whom needed a further operation, although in most healing took place and they returned to their levels of preinjury activity (Fig. 6).

We have recently validated a self-administered questionnaire-based instrument to measure the severity of Achilles tendinopathy.⁸⁹ The VISA-A questionnaire is valid, reliable and easy to administer, and seems to be suitable for both clinical rating and quantitative research.

Other operative procedures

Percutaneous longitudinal tenotomy. We have used multiple percutaneous longitudinal tenotomies when conservative management has failed in patients with isolated tendinopathy with no involvement of the paratenon and a well-defined nodular lesion less than 2.5 cm long.⁹⁰ Ultrasound can be used to confirm the precise location of the area of tendinopathy. If the multiple percutaneous tenotomies are performed in the absence of chronic paratendinopathy, the outcome is comparable to that of open procedures. It is a simple procedure and can be performed in the

Fig. 6

Breakdown of the wound after percutaneous longitudinal tenotomies in a 55-year-old male jogger two weeks after the procedure. Conservative management was begun and the wound healed over the following three weeks.

Fig. 7

Breakdown of the wound after open exploration in a 42-year-old male fell runner three weeks after operation. The patient felt well after removal of the dressing, and went to run. He fell and disrupted the wound. Conservative management was elected, and the wound healed over eight weeks. By seven months after the procedure, he had returned to training.

clinic under local anaesthesia without a tourniquet, but attention to detail is necessary, since even in minimally invasive procedures complications are possible (Fig. 7).

Muscle transfer to the body of the tendon. Longitudinal tenotomies increase the blood supply of the degenerated area.⁸⁷ Recently, in a rabbit model, after longitudinal tenotomy we performed a soleus pedicle graft within the operated tendon, and showed that the transplanted muscle was viable and had integrated well within the tendon tissue three months after the transplant, without transforming into connective tissue. The transplanted muscle fibres integrated with the tendon but remained distinct from it. Hypervascularisation of the graft tissue, probably due to the operation, was also observed, together with neoangiogenesis up to three months after the operation.⁹¹

Future advances

With progress in molecular biology, it may be possible to identify the factors which influence the metabolism of the tenocytes and promote their natural healing process. The role of growth factors in the healing of tendons is still unclear, although basic fibroblast growth factor can stimulate healing by promoting cell proliferation and synthesis of the matrix.⁹² Application of the appropriate growth factors at certain periods during the process of repair may improve the result after tendon lesions. However, most of these growth factors are rapidly metabolised proteins, and their delivery is challenging and difficult. Transfer of growth factor genes into tenocytes may eliminate this problem by allowing continuous local release of growth factors at the healing site. Gene transfer for the targeted delivery of growth factors has been used successfully in animal studies.⁹³

Conclusions

Although tendinopathy of tendo Achillis has been studied extensively, there is a clear lack of properly conducted

scientific research to clarify its aetiology, pathology and optimal management. Most patients respond to conservative measures if the condition is recognised early, while continuance of the offending activities leads to chronic changes which are more resistant to non-operative treatment. Teaching patients to control the symptoms may be more beneficial than leading them to believe that Achilles tendinopathy is fully curable. Surgery usually involves removal of adhesions and degenerated areas, decompression of the tendon by tenotomy or measures to influence the local circulation. It is still debatable why tendinopathic tendons respond to surgery.³¹ We do not know whether surgery induces revascularisation, denervation or both, resulting in reduction of pain. It is also unclear how longitudinal tenotomy improves vascularisation. As the biology of tendinopathy is being clarified, more effective methods of management may emerge, improving the rate of success of both conservative and operative treatment.

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