ACHILLES TENDINOPATHY PART 1 – PATHOPHYSIOLOGY AND CLINICAL FEATURES

BY NAT PADHIAR, SPORTS PODIATRIST AND PODIATRIC SURGEON AND NAMITA ACHARYA, ROYCE CHAN, KARIN DAVINII ET AL ***SEE FULL LIST OF CONTRIBUTORS ON P.28***

This is the first of two reviews about the debilitating condition of Achilles tendinopathy. Understanding the anatomy and underlying pathology of this condition, including the presence or absence of neovascularisation, will markedly improve your ability to diagnose and treat the patients and athletes in your care. The information provided here will help you with your investigations, highlighting some of the pitfalls in diagnosis, and guide you towards the best clinical approach. The second part of this review will be published in the October 2010 issue of *sportEX medicine* and will deal with the management of this difficult tendinopathy.

INTRODUCTION

Achilles tendinopathy is classified as either insertional or non-insertional, where the pathology presents at the bone–tendon junction or further proximally, respectively. It is becoming increasingly common in both professional and recreational athletes and is now receiving more attention in research and practice. It is a frustrating condition, and its aetiology is not completely understood, although intrinsic and extrinsic factors, including biomechanical abnormalities and excessive mechanical overload, are reasonable explanations for this overuse injury. It is particularly common in runners, basketball players and other jumping athletes (1,2), however the non-athletic population is not completely exempt. In one study, 31% of 58 patients were sedentary (3).

The aim of this part of the review is to outline what is already known of Achilles tendinopathy, in relation to anatomy, pathophysiology, clinical presentation, investigations and treatments, so conclusions regarding the best clinical approach to this potentially incapacitating recalcitrant morbidity can be made.

Terminology

Tendinitis and *tendinosis*, previously used as descriptive terms for tendon pathology, suggested tendon inflammation and tendon degeneration with a failed healing response respectively. With *tendinitis* the involved tendon should show inflammatory features. Unfortunately, this term is now being used to refer to a clinical syndrome, as opposed to a specific histopathological entity. Misuse of the term has been generally accepted, but abandoning its use in conditions that are truly tendinitis may improve appreciation of the chronicity of the condition (4). *Tendinosis* is a histopathological entity affecting all components of the tendon, including collagen fibres, tenocytes and extracellular matrix (5,6). It was first used by Puddu et al (7) in 1976 to describe tendon degeneration without clinical or histological signs of intratendinous inflammation. In 1998, the term 'tendinopathy' was coined by Maffulli et al (4) as a generic descriptor of conditions in and around tendons that arise from overuse, encompassing pain, swelling and impaired performance (4). This umbrella term has been successfully used for over a decade to accurately describe tendon pathology and minimise the misuse of other descriptive terms.

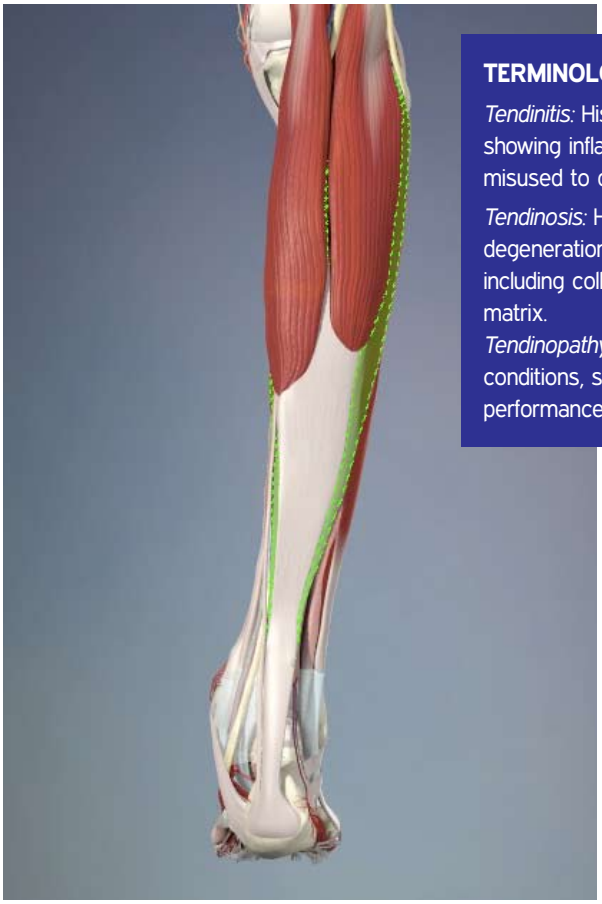


Figure 1: Anatomy of the triceps surae and Achilles tendon

TERMINOLOGY IN TENDON PATHOLOGY

Tendinitis: Histopatholgical description of tendons showing inflammatory features but sometimes misused to describe the clinical syndrome.

Tendinosis: Histopathological description of degeneration in all components of the tendon, including collagen fibres, tenocytes and extracellular matrix.

Tendinopathy: Umbrella term for all tendon overuse conditions, such as pain, swelling and impaired performance.

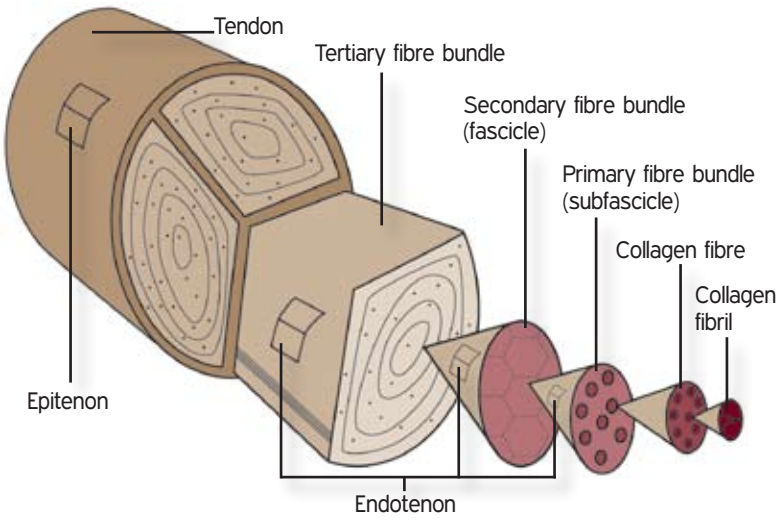


Figure 2: The hierarchical structure of tendon. Groups of fibres form the fascicles, which are surrounded by the endotenon, a loose connective tissue layer. The tertiary bundles of fascicles are surrounded by another connective tissue layer, the epitenon, and a double-layered covering, the paratenon (not shown)

ANATOMY

The Achilles tendon is the largest and strongest tendon in the human body (Fig. 1.). It is formed by the merging of the deep and superficial fasciae of the triceps surae and it inserts into the posterior aspect of the calcaneum where a deep retrocalcaneal bursa is frequently present (8).

The Achilles tendon lacks a true tendon sheath and is enveloped by paratenon, a double-layered membrane. The tendon itself consists of longitudinally orientated collagen fibrils interspersed with fibroblasts (Fig. 2.).

These collagen fibrils are bundled into fascicles, containing vasculo–neural and lymphatic systems, all of which are separated by connective tissue septations known as endotenon. These fascicles are then grouped together by epitenon, which in turn is surrounded by peritenon. This epitenon and peritenon constitute the layers of the paratenon membrane (8,9).

Around 90–95% of the cellular element of the tendon consists of tenocytes and tenoblasts, while the extracellular matrix consists of collagen, elastin fibres, proteoglycans and organic components such as calcium (10). Type 1 collagen fibres are the most abundant, followed by type 2. Together these collagen fibres account for 65–80% of the dry mass of the tendon, while elastin fibres only comprise 1–2% of this (11).

In general, blood to the Achilles tendon is supplied by the posterior tibial and peroneal arteries. Both have an input to three vascular territories: the midsection (supplied by the peroneal artery) and the proximal and distal sections (supplied by the posterior tibial artery) (12). The posterior tibial artery contributes the most to the vascular supply, which is why the midsection of the Achilles tendon is usually more hypervascular than the rest of the tendon (12,13). The nervous supply of the Achilles tendon mainly originates from surrounding muscles and the branches of the cutaneous nerves. Although they mainly terminate on the superficial aspect of the tendon, some nerves follow the vascular channels within the long axis of the tendon (14).

Nerve endings of myelinated fibres function as specialised mechanoreceptors, detecting pressure and tension, while the unmyelinated nerve endings act as nociceptors, detecting pain (15).

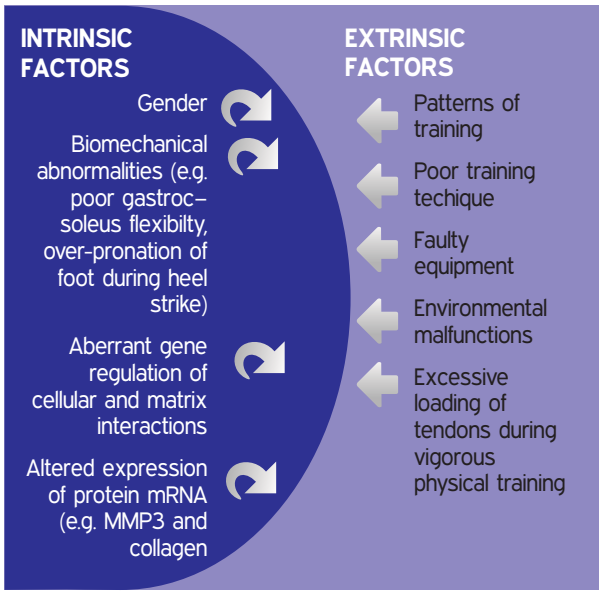


Figure 3: Aetiological factors in Achilles tendinopathy

AETIOLOGY

As mentioned before, the aetiology of Achilles tendinopathy is not completely understood, although the cause can be classified as either intrinsic or extrinsic. Extrinsic factors include changes in training patterns, poor technique, and equipment and environmental malfunctions (10,15,16). However, excessive loading of tendons during vigorous physical training is considered to be the main pathological stimulus for tendon degeneration. Tendons respond to repetitive overloading beyond the physiological threshold by inflammation of the tendon sheath, degeneration of the tendon body, or both (15).

Common intrinsic causes include biomechanical abnormalities, genetic factors and gender differences (14). Among the biomechanical factors are poor gastroc–soleus flexibility and over-pronation of the foot during heel strike. Thus over-pronation results in excessive motion of the hindfoot in the frontal plane and is thought to cause a 'whipping' action on the Achilles tendon, hence predisposing to tendinopathy (10,17). A prospective cohort study on male cadet officers identified that the strength of the plantar flexors and the amount of dorsiflexion excursion were significant predictors of an Achilles tendon overuse injury (18).

The genetic influence involves changes in the expression of genes regulating cell–cell and cell–matrix interactions, with downregulation of MMP3 mRNA in tendinopathic Achilles tendon samples. In addition, type 1 and type 3 collagen mRNAs have been found at higher levels in tendinopathic samples than in normal samples (19).

While appreciating the interplay of both extrinsic and intrinsic factors, in general it is the extrinsic factors that tend to cause acute Achilles tendon trauma, but a combination of both intrinsic and extrinsic factors is responsible for chronic tendinopathy (15).

HISTOPATHOLOGY

The histopathology can be classified as peritendinous or intratendinous (14) (see Fig. 4). Histologically, Achilles tendinopathy is characterised by non-inflammatory intratendinous collagen degeneration and a failed healing response. There is also scattered vascular ingrowth, hypercellularity, and increased levels of interfibrillar glycosaminoglycans (20,21).

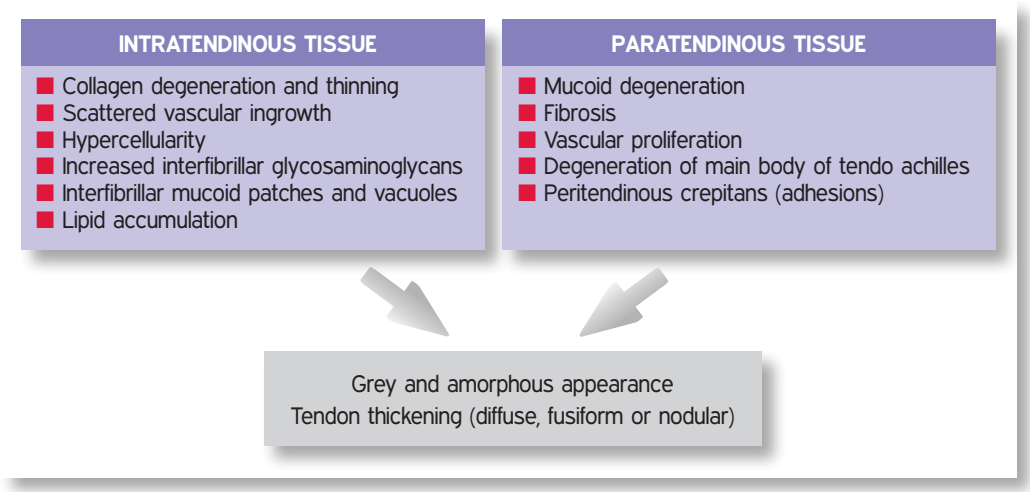


Figure 4: Histopathological signs of tendinopathy

The degeneration is either mucoid or lipoid, microscopically characterised by thinning of collagen fibres, interfibrillar mucoid patches and vacuoles, or an intratendinous accumulation of lipids, respectively (20). These degenerative changes are a constant feature of Achilles tendon rupture, emphasising that tendinosis is an important precursor and cause of tendon rupture (22).

The peritendinous tissue can be involved in the early stages of tendinopathy and can show histological signs of mucoid degeneration, fibrosis and vascular proliferation, either alone or in combination with degeneration of the main body of the tendo Achilles (23). Paratenon involvement occasionally presents as 'peritendinous crepitans' and is due to adhesions between the tendon and the paratenon (10). The occurrence of these histopathological changes results in macroscopic alterations in the appearance of the affected portion of the tendon, whereby the tendon begins to appear grey and amorphous from a previously normal glistening white appearance. In addition, there is diffuse, fusiform or nodular thickening of the tendon (24).

Pain is the main symptom of Achilles tendinopathy. However the pain mechanism is not completely understood. It was traditionally thought that pain originated from associated inflammation or from separation of collagen fibres. Although these mechanisms were widely accepted in the past, numerous studies have now provided data that are inconsistent with either theory (25). An in vivo microdialysis study by Alfredson recently identified an abnormal increase in glutamate levels in patients with painful Achilles tendinopathy (26). However, this biochemical finding needs to be correlated with histopathological findings and pain in Achilles tendinopathy before glutamate can be pinpointed as the real cause of the pain.

CLINICAL FEATURES

Achilles tendinopathy usually presents as morning stiffness, swelling and pain in the mid-portion of the tendon. The pain is usually exacerbated by eccentric loading exercises (4). Studies have shown that the duration of symptoms in Achilles tendinopathy patients can vary from a few months to a few years (27). It has been suggested that the pain in the early stages is bearable and does not affect daily activities, so patients only seek medical attention if the pain severely affects ability to carry out daily activities as the disease progresses (28). The pain is thought to be related to vasculo–neural growth in the tendon (29), however there is some evidence to show that the degree of neovascularisation does not correlate with the severity of symptoms (30).

Symptomatic Achilles tendons are caused by an abnormality in the kinetic chain of the lower limb, therefore the hips, back, knees, ankles and both Achilles should be examined. It is important to understand the origin of the abnormal biomechanics because this helps determine the target joint for intervention.

EXAMINING THE ACHILLES

As with other orthopaedic physical examinations, it is generally agreed that the Achilles should be examined by looking, feeling and moving (31) (Fig. 5).

- **Inspection:** Observe the Achilles from the sides and dorsal aspects for local erythema and swelling. Compare both Achilles (20). Inspect the foot for malalignment, for over-pronation, forefoot varus and pes planus. Over-pronation was present in 61 of 109 runners in a study by Clement et al (32) and was reported to predispose

LOOK

- Inspect the Achilles from the sides and back. Compare the Achilles of both legs.
- Is there any redness or swelling?
- Is there any malalignment of the foot, or over-pronation?
- Is there varus of the forefoot or pes planus?

FEEL

- Can you feel any areas of thickness, especially around the distal two-thirds of the tendon?
- Does the skin feel hot?
- Is there any tenderness?
- Can you feel any crepitus or nodules?

MOVE

- Are dorsiflexion and plantarflexion of the ankle joint as you would expect?
- Does the subtalar joint move correctly (actively and passively)?

Figure 5: Examining the Achilles

Achilles tendinopathy especially among individuals who participate in repetitive action sports.

- **Palpation:** Palpate the Achilles for tendon thickness (most commonly in the distal two-thirds of the tendon (33)) as well as for temperature, crepitus, tenderness and nodules.
- **Movement:** Thoroughly assess dorsiflexion and plantarflexion of the ankle joints and the mobility of the subtalar joint both actively and passively. In Kvist's study, 60% of the athletes had impaired dorsiflexion and subtalar joint movement (34).

Special tests

There are two special tests. First, the toe raise – or the calf raise – which can be performed to assess the strength of the calf muscles because calf muscle weakness will cause abnormal biomechanics in the Achilles. Clement et al (32) highlighted that 41 out of 109 runners had weakness in their calf muscles and had an increased risk of developing Achilles tendinopathy (32). The second validated method of assessing pain and function of the Achilles tendon (35) is the Victorian Institute of Sport Assessment questionnaire for Achilles tendinopathy – the VISA–A.

Differentials

As the symptoms are non-specific, there are a few conditions that present in the same way. These include Achilles tendinitis, paratendinitis, partial or complete rupture, retrocalcaneal bursitis, Achilles bursitis and referred pain. The crucial difference is that the pathophysiology in Achilles tendinopathy does not involve inflammation, so non-steroidal anti-inflammatory drugs will not alleviate symptoms like they do for the other conditions. The onset of the symptoms also helps differentiation, for example sudden onset of severe pain suggests Achilles rupture. Further investigations are needed to confirm the diagnosis.

INVESTIGATIONS

Öhberg et al (28) found neovascularisation in all their symptomatic Achilles tendinopathy specimens, but none in asymptomatic normal tendons. Due to the significance of such neovascularisation, power Doppler and colour Doppler have been used extensively in other studies and in clinical practice.

Ultrasound scans have also been used to demonstrate sonographic differences in normal and pathological tendons. Data from many studies suggest that deterioration of neovascularisation is associated with an improved clinical prognosis, so ultrasound is an ideal tool for grading, capable of showing the disappearance of neovascularisation during ankle dorsiflexion. This further supports the mechanism whereby eccentric exercise permanently damages the neovascularisation process and accompanying formation of new nerves (30). Since the publication of Alfredson et al's (36) results, eccentric loading exercises are the mainstay of conservative management of this tendinopathy.

Other diagnostic modalities include tendon biopsy and magnetic resonance imaging (MRI). Aström et al (37) showed that tendon biopsy detected all cases of tendinopathy and MRI and ultrasound had comparable good detection rates. Another study (38) produced similar results, with ultrasound detecting 23 out of 34 cases of Achilles tendinopathy, and MRI detecting 19 out of 34. In this study, ultrasound had a higher positive predictive value (0.65) than MRI (0.56), in contrast to a lower negative predictive values of 0.68 and 0.94, respectively.

While many studies demonstrate the occurence of neovascularisation in symptomatic Achilles tendinopathy, there are varying opinions about the absolute presence of neovascularisation. This may be because of differences in methodology and the types of participants examined in such studies.

INVESTIGATIONS FOR ACHILLES TENDINOPATHY

- Ultrasound
- Tendon biopsy
- Magnetic resonance imaging

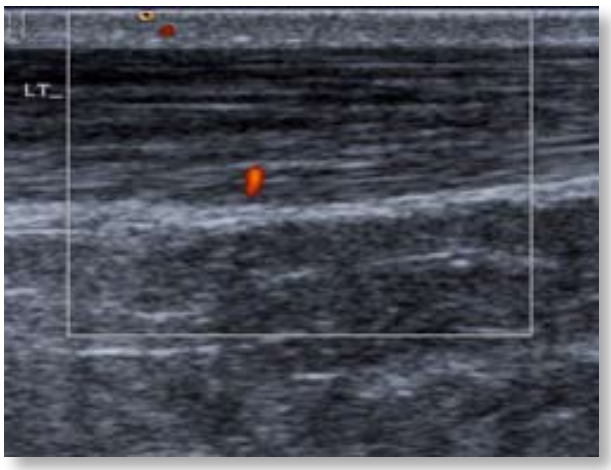


Figure 6: Ultrasound image of thickening of Achilles tendon

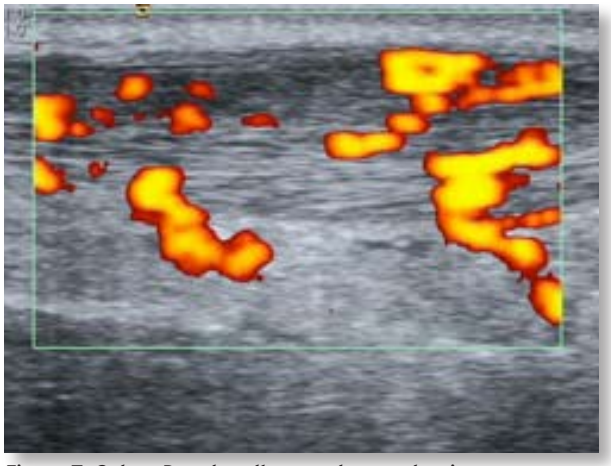


Figure 7: Colour Doppler ultrasound scan showing neovascularisation of Achilles tendon

ULTRASOUND IN ACHILLES TENDINOPATHY

Features found on ultrasound

Achilles tendinopathy can manifest in several ways on ultrasound, showing features such as:

- diffuse or focal tendon thickening (Fig. 6)
- loss of the parallel contours
- changes in reflectivity
- neovascularisation (Fig. 7).

It is generally agreed (30,39,40) that the main distribution of tendon thickening is in the distal two-thirds of the tendon, but Gibbon et al (41) showed that tendon thickening was more common in the proximal two-thirds of the tendon. The

> "TENDINOPATHY – THIS UMBRELLA TERM HAS BEEN SUCCESSFULLY USED FOR OVER A DECADE TO ACCURATELY DESCRIBE TENDON PATHOLOGY AND MINIMISE THE MISUSE OF OTHER DESCRIPTIVE TERMS"

only discrepancy in Gibbon et al's methodology was the position used during the examination. Their patients were in a prone position with the ankle in full dorsiflexion whereas patients in the other studies were prone with the ankle in a neutral position, resting off the end of the bed. Full ankle dorsiflexion would disrupt neovascularisation and the measurement of the tendon thickness, but it would not affect the position of the thickening. Gibbon et al's (41) findings are well supported by a large sample size of 118 patients, of whom a significant proportion (109 out of 118 tendons) showed thickness in the proximal two-thirds. The reasons for the varying observations are unknown.

Presence of neovascularisation

Two studies by Öhberg and colleagues (28,42) and one by Alfredson et al (29) produced results showing that neovascularisation (Fig. 7) was always present in the Achilles tendinopathy cases they examined and always absent in normal tendons. All three studies were case–control studies with significant sample sizes (28, 41 and 25 patients, respectively) and both the control and the Achilles tendinopathy groups were well matched in terms of their baseline characteristics. If proved to be correct, then the absolute presence of neovascularisation could be used as a cardinal diagnostic sign. Unfortunately many other studies have found this not to be the case. Leung et al found no neovascularisation in 14 out of 30 cases (40), Peers et al showed no neovascularisation in 22 out of 25 (43), de Vos et al in 40 out of 67 (30) and Richards et al in 45 out of 55 (44).

One possible explanation for these discrepancies might relate to the ultrasound imaging. Ultrasound has a higher positive predictive value of 0.65 and a lower negative predictive value of 0.68 (38), which means there is probable chance of acquiring false-positive and false-negative results. In addition, the studies in question used different ultrasound equipment (models and transducers) and frequency settings as shown in Table 1. Such differences might have contributed to reduced image resolution, thus affecting the results. Despite differing results from de Vos et al (30), Peers et al (43) and Richards et al (44), their methodologies was sound and even involved extra precautions to minimise obscuring of smaller blood vessels. De Vos et al (30), for example, used minimal probe pressure, and Richards et al (44) and Peers et al (43) set the power Doppler to a pulse repetition frequency of 800–1000 Hz and adjusted the colour gain to exclude signals from normal vessels.

From Table 1 it is clear that the studies using colour Doppler reported 100% neovascularisation, which implies that this modality might be more sensitive than power Doppler. Richards et al (44) compared power Doppler to colour Doppler for detecting neovascularisation in Achilles tendinopathy patients. Their results conclusively showed that power Doppler was more accurate, detecting 191 vessels compared to 44 vessels with colour Doppler, leading them to claim that power Doppler was more accurate. Since the publication of their paper in 2005 two of the four of the chosen studies used power Doppler in preference. No other studies have challenged this finding. Leung et al (40), de Vos et al (30), Peers et al (43) and Richards et al (44) have shown with power Doppler that all normal tendons have no neovascularisation, but not all symptomatic Achilles tendinopathy tendons have neovascularisation. These results are comparable across studies because the subjects were similar in age (mid-40s) and the same transducer was used (5–12 MHz) except by de Vos et al.

TABLE 1: ULTRASOUND SCANNER MODELS AND TRANSDUCERS USED IN THE NEOVASCULARISATION STUDIES

Study	Model	Type of probe (frequency)	Symptomatic patients with neovascularisation
Alfredson et al (29)	Siemens Sequoia 512	Colour Doppler (8–13 MHz)	100%
Öhberg et al (28)	Siemens Sequoia 512	Colour Doppler (8–13 MHz)	100%
Öhberg and Öhberg (42)	Siemens Sequoia 512	Colour Doppler (8–13 MHz)	100%
de Vos et al (30)	Unspecified Siemens model	Power Doppler (8–13 MHz)	63%
Peers et al (43)	HDI 5000 Philips	Power Doppler (5–12 MHz)	88%
Leung et al (40)	HDI 5000 Philips	Power Doppler (12–5 MHz)	47%
Richards et al (44)	HDI 3000 Philips	Power Doppler (5–12 Mhz)	82%
Sengkerij et al (45)	Unspecified Siemens model	Power Doppler (8–13 MHz)	70%

> NEOVASCULARISATION IN ACHILLES TENDINOPATHY IS ABNORMAL AND ASSOCIATED NERVES SHOW NEUROGENIC INFLAMMATION. LOCAL ANAESTHETIC IN THE VESSELS REMOVES PAIN AND THEREFORE REMOVING VESSELS MIGHT IMPROVE SYMPTOMS

It does raise the issue of the intra- and inter-observer reliability, however. Tests conducted by Öhberg et al (42) showed excellent intra-observer reliability, which increases with the degree of experience of the radiologist. Sengkerij et al (45) reported excellent inter-observer reliability for power Doppler for detecting neovascularisation.

There are several reasons why not all Achilles tendinopathy tendons show neovascularisation in these studies. Richards et al (44) theorised that neovascularisation might go undetected in tendons that are less than 6.5 mm thick. As the other studies included painful tendon thickening among their diagnostic criteria for Achilles tendinopathy, but failed to state the actual tendon thicknesses, it is not possible to test this theory.

Furthermore, among the studies that showed 100% neovascularisation, Öhberg et al (28) scanned patients before asking them to participate in the study, and Alfredson et al (29) verified the diagnosis using ultrasound. In both cases, it is unclear just what was found on ultrasound – if the presence of neovascularisation was part of the diagnosis (and subsequently part of the the inclusion criteria) then both studies were subject to selection bias.

> "IF PROVED TO BE CORRECT, THE ABSOLUTE PRESENCE OF NEOVASCULARISATION COULD BE USED AS A CARDINAL DIAGNOSTIC SIGN. UNFORTUNATELY MANY OTHER STUDIES HAVE FOUND THIS NOT TO BE THE CASE"

Assessing progress of therapy

To determine clinical improvement, most studies use the VISA–A questionnaire or visual analogue scales and patient satisfaction questionnaires as outcome measures. In contrast, Öhberg et al (42) were unique in using the degree of neovascularisation as part of their ultrasound assessment of pathological features of Achilles tendinopathy. They graded neovascularisation according to three levels:

- 0 for no vessels,
- (+) for 1–2 vessels
- + for several irregular vessels.

They concluded that eccentric exercises were an effective therapy for Achilles tendinopathy and that a normal tendon appearance on ultrasound (together with the absence of neovascularisation) is associated with clinical improvement. Such conclusions appear reliable because the sample size was adequate and because the methodology, ultrasound equipment and techniques were identical to the other studies. However, future research is needed to add support to their findings.

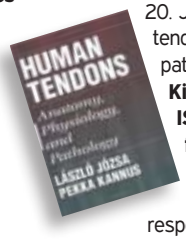
There is no consensus on how to quantify the degree of neovascularisation. Boesen et al (46) expressed neovascularisation as a pixel percentage, and Peers et al (43) expressed it as surface area of the signal on the power Doppler

images, but other studies simply state the presence or absence of neovascularisation. Boesen et al (39) modified Öhberg's grading system to five grades, numbered 0 to 4. In our opinion, the current methods are too crude or too impractical for a clinical setting, so a more comprehensive grading system should be used.

Overall ultrasound has a vital role in the diagnosis and monitoring of Achilles tendinopathy as it has comparable accuracy to other imagining modalities, is easily accessible, and exhibits a high inter- and intra-observer reliability (45).

References

1. Kvist M. Achilles tendon injuries in athletes. **Sports Medicine 1994;18:173–201**
2. Jarvinen TA, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: Etiology and epidemiology. **Foot and Ankle Clinics 2005;10:255–266**
3. Rolf C, Movin T. Etiology, histopathology and outcome of surgery in achillodynia. **Foot and Ankle International 1997;18:565–569**
4. Maffulli.N, Khan.K, Puddu.G. Overuse tendon conditions: Time to change a confusing terminology. **Arthroscopy 1998;14(8):840–853**
5. Jarvinen M, Jozsa L, Kannus P. Histopathological findings in chronic tendon disorders. **Scandinavian Journal of Medicine and Science in Sports 1997;7:86–95**
6. Khan K, Cook J, Kannus P. Time to abandon the *tendinitis* myth. **British Medical Journal 2002;324;626–627**
7. Puddu G, Ippolito E, Postacchini F. A classification of Achilles tendon disease. **American Journal of Sports Medicine 1976;4:145–150**
8. Fornage BD. Achilles tendon: US examination. **Radiology 1986;159(3):759–764**
9. Maffulli N, Benazzo F. Basic science of tendons. **Sports Medicine and Arthroscopy Review 2000;8:1–5**
10. Kader D, Saxena A, Movin T, Maffulli N. Achilles tendinopathy: Some aspects of basic science and clinical management. **British Journal of Sports Medicine 2002;36:239–249**
11. Kannus P. Structure of the tendon connective tissue. Scandinavian **Journal of Medicine and Science in Sports 2000;10:312–320**
12. Chen TM, Rozen WM, Pan WR, Ashton MW, Richardson MD, Taylor GI. The arterial anatomy of the Achilles tendon: **Anatomical study and clinical implications. Clinical Anatomy 2009;22(3):377–385**
13. Ahmed IM, Lagopoulos M, McConnell P, Soames RW, Sefton GK. Blood supply of the Achilles tendon. **Journal of Orthopaedic Research 1998;16(5):591–596**
14. Tan SC, Chan O. Achilles and patellar tendinopathy: Current understanding of pathophysiology and management. **Disability and Rehabilitation 2008;30(20/22):1608–1615**
15. Maffulli N, Sharma P, Luscombe K. Achilles tendinopathy: Aetiology and management. **Journal of the Royal Society of Medicine 2004;97:472–476**
16. Kannus P, Natri A. Etiology and pathophysiology of chronic tendon disorders in sports. **Scandinavian Journal of Medicine and Science in Sports 1997;7:78–85**
17. Khan KM, Maffulli N. Tendinopathy: An Achilles' heel for athletes and clinicians. **Clinical Journal of Sports Medicine 1998;8:151–154**
18. Mahieu NN, Witvrouw E, Stevens V, Van Tiggelen D, Roget P. Intrinsic risk factors for the development of Achilles tendon overuse injury: A prospective study. **American Journal of Sports Medicine 2006;34(2):226–235**
19. Ireland D, Harrall R, Curry V, Holloway G, Hacknet R, Hazelman B, Riley G. Multiple changes in gene expression in chronic human Achilles tendinopathy. **Matrix Biology 2001;20:159–169**



20. Jozsa LG, Kannus P. Human tendons: Anatomy, physiology, and pathology. **Champaign, IL: Human Kinet; 1997 (from £230.14). ISBN 0873224841. To Order from Amazon** http://bit.ly/99HZfZ
21. Leadbetter WB. Cell-matrix response in tendon injury. **Clinical Journal of Sports Medicine 1992;11:533–578**
22. Waterston SW, Maffulli N, Ewen SWB. Subcutaneous rupture of the Achilles tendon: Basic science and aspects of clinical practice. **British Journal of Sports Medicine 1997;31:285–298**
23. Almekinders LC, Temple JD. Etiology, diagnosis, and treatment of tendonitis: An analysis of the literature. **Medicine and Science in Sports and Exercise 1998;30:1183–1190**
24. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. Update and implications for clinical management. **Sports Medicine 1999;27:393–408**
25. Khan KM, Cook JL, Maffulli N, Kannus P. Where is the pain coming from in tendinopathy? It may be biochemical, not only structural, in origin. **British Journal of Sports Medicine 2000;34:81–83**
26. Alfredson H. In situ microdialysis in tendon tissue: High levels of glutamate, but not prostaglandin E2 in chronic Achilles tendon pain. **Knee Surgery Sports Traumatology Arthroscopy 1999;7:378–381**
27. Galloway MT, Jokl P, Dayton OW. Achilles tendon overuse injuries. **Clinical Sports Medicine 1992;11:771–782**
28. Ohberg L, Lorentzon R, Alfredson H. Neovascularisation in Achilles tendons with painful tendinosis but not in normal tendons: An ultrasonographic investigation. **Knee Surgery, Sports Traumatology Arthroscopy 2001;994:233–238**
29. Alfredson H, Ohberg L, Forsgren S. Is vasculo–neural ingrowth the cause of pain in chronic Achilles tendinosis? An investigation using ultrasonography and colour Doppler, immunohistochemistry, and diagnostic injections. **Knee Surgery Sports Traumatology Arthroscopy 2003;11(5):334–338**
30. de Vos RJ, Weir A, Cobben LP, Tol JL. The value of power Doppler ultrasonography in Achilles tendinopathy: A prospective study. **American Journal of Sports Medicine 2007;35(10):1696–1701**
31. Fredericson M. Common injuries in runners. Diagnosis, rehabilitation and prevention. **Sports Medicine 1996;21:49–72**
32. Clement DB, Taunton JE, Smart GW. Achilles tendinitis and peritendinitis: Etiology and treatment. **American Journal of Sports Medicine 1984;12:179–184**
33. Gibbon WW, Cooper JR, Radcliffe GS. Distribution of sonographically detected tendon abnormalities in patients with a clinical diagnosis of chronic Achilles tendinosis. **Journal of Clinical Ultrasound 2001;28(2):61–66**
34. Kvist M. Achilles tendon injuries in athletes. **Annales Chirurgiae et Gynaecologiae 1991;80:188–201**
35. Robinson JM, Cook JL, Purdam C, Visentini PJ, Ross J, Maffulli N, Taunton JE, Khan KM. The VISA-A questionnaire: A valid and reliable index of the clinical severity of Achilles tendinopathy. **British Journal of Sports Medicine 2001;35(5):335–341**
36. Alfredson H, Pietila T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. **American Journal of Sports Medicine 1998;26:360–366**
37. Aström M, Gentz CF, Nilsson P, Rausing A, Sjoberg S, Westlin N. Imaging in chronic Achilles tendinopathy: A comparison of ultrasonography, magnetic resonance imaging and surgical findings in 27 histologically verified cases. **Skeletal Radiology 1996;25(7):615–620**
38. Khan KM, Forster BB, Robinson J, Cheong Y, Louis L, Maclean L, Taunton JE. Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two-year prospective study. **British Journal of Sports Medicine 2003;37(2):149–153**
39. Boesen MI, Koenig MJ, Torp-Pedersen S, Bliddal H, Langberg H. Tendinopathy and Doppler activity: The vascular response of the Achilles tendon to exercise. **Scandinavian Journal of Medicine and Science in Sports 2006;16(6):463–469**
40. Leung JL, Griffith JF. Sonography of chronic Achilles tendinopathy: A case–control study. **Journal of Clinical Ultrasound 2008;36(1):27–32**
41. Gibbon WW, Cooper JR, Radcliffe GS. Distribution of sonographically detected tendon abnormalities in patients with a clinical diagnosis of chronic Achilles tendinosis. **Journal of Clinical Ultrasound 2001;28(2):61–66**
42. Ohberg L, Alfredson H. Effects on neovascularisation behind the good results with eccentric training in chronic mid-portion Achilles tendinosis? **Knee Surgery Sports Traumatology Arthroscopy 2004;12(5):465–70**.

43. Peers KH, Brys PP, Lysens RJ. Correlation between power Doppler ultrasonography and clinical severity in Achilles tendinopathy. **International Orthopaedics 2003;27(3):180–183**
44. Richards PJ, Win T, Jones PW. The distribution of microvascular response in Achilles tendonopathy assessed by colour and power Doppler. **Skeletal Radiology 2005;34(6):336–342**
45. Sengkerij PM, de Vos RJ, Weir A, van Weelde BJ, Tol JL. Interobserver reliability of neovascularization score using power Doppler ultrasonography in midportion Achilles tendinopathy. **American Journal of Sports Medicine 2009;37(8):1627–1631**
46. Boesen MI, Boesen A, Koenig MJ. Ultrasonographic investigation of the Achilles tendon in elite badminton players using color Doppler. **American Journal of Sports Medicine 2006;34(12):2013–2021**.

RELATED SPORTEX ARTICLES

■ Speed C. Tendon injuries: an overview. **sportEX medicine 2009;39:7-11**
■ Cook J. Blood vessels: their role in tendon pain and pathology. **sportEX medicine 2006;29:18-20**
■ Mafulli N and Sharma P. Understanding Achilles tendionpathy. **sportEX medicine 2006;27:10-13**

OTHER ARTICLES BY THIS AUTHOR

■ Padhiar N. Chronic compartment syndrome of the leg. **sportEX medicine 2009;40:16-22.**

CONTRIBUTORS TO THIS REVIEW

The following authors contributed equally to this review, however because of space constraints only four of them are featured here. You can read about the others online at

Ms Namita Acharya iBSc (Hons) SEM, medical student
Dr Otto Chan MBBS FRCS FRCR, radiologist
Mr Royce Chan iBSc (Hons) SEM, medical student
Dr Tom Crisp MBBS FFSEM, sports physician
Mr Kiran Divanii iBSc (Hons) SEM, medical student
Mr David Goodier MBBS FRCS, orthopaedic surgeon
Dr Rosy Jalan MBBS FRCR, radiologist
Mr John B King MBBS FFSEM FRCS, orthopaedic surgeon
Dr Dylan Morrisey MSc PhD MCSP, physiotherapist
Professor Nicola Maffulli MD PhD FRCS, orthopaedic surgeon
Dr Nick Webborn MBBS FFSEM, sports physician

■ Does the position of the patient and the position of the foot matter when scanning for Achilles tendinopathy?
■ What is the reliable grading system for neovascularisation in Achilles tendinopathy?

THE AUTHORS

Dr Nat Padhiar MSc PhD FCPodS of London SportsCare graduated in podiatry from Chelsea School and pursued a career in podiatric sports medicine and podiatric surgery. In 1999 he was awarded a doctor of philosophy degree in the field of orthopaedics and sports medicine from St Bartholomew's and the Royal London School of Medicine and Dentistry, Queen Mary at London University. He is also an honorary senior lecturer in the Centre for Sports and Exercise Medicine (CSEM), Queen Mary at London University. He is a keen sportsman who enjoys playing cricket and tennis and taking part in endurance sport (32 marathons to date). In a professional capacity he has been part of the medical team at various sporting venues. He is appointed by LOCOG as an expert medical volunteer and lead clinician for podiatry for the London 2012 Olympic and Paralympic Games.

Dr Tom Crisp MBBS FFSEM of London SportsCare is a full time specialist in sport and exercise medicine, being the second doctor in the UK to be placed on the specialist Register of General Medical Council as a sports physician. Tom started working in sports medicine in 1982 when he opened a sports injury clinic in Chelmsford in the local physiotherapy clinic and acted as team doctor to Essex Cricket Club. In 1986 he gained a diploma in sports medicine at Royal London Hospital (now Queen Mary College at University of London) and travelled to Moscow with the england hockey team soon after. He has attended many sporting venues and is now a member of the International Medical Commission of FISU (International University Sports Federation) supervising World Games and championships around the world in many different sports.

Mr John King MBBS FFSEM FRCS of London SportsCare is at present Emeritus consultant orthopaedic surgeon and senior lecturer at the Centre for Sports and Exercise Medicine (CSEM) at the The Royal London Hospital. He founded (in 1981) and developed the current CSEM and remained the director of the academic department of sports medicine until 2003. He is also an examiner in Sports Medicine of The Worshipful Society of Apothecaries of London, and an academic associate at The University of London Interdisciplinary Research Centre in Biomedical Materials, Queen Mary College. He is the past chairman of the British Association of Sports and Medicine (BASM) and an external adviser to the Bath Distance Learning Course for Doctors in Sports Medicine. He has over 100 peer-reviewed publications to his name including books and research papers and is also a recipient of the prestigious Sir Roger Mannister Medal for lifetime services to sports and exercise medicine.

Dr Otto Chan MBBS FRCS FRCR of London SportsCare was appointed at The Royal London Hospital in 1993 and dedicated his career to teaching and advancing emergency, trauma, musculoskeletal and intervention radiology. He now works solely in the private sector and is based at BMI London Independent Hospital with his main interest in musculoskeletal and sports radiology. He is one of the leading radiologists in this field and has lectured extensively across the globe. He is very much in favour of a multidisciplinary team approach to patient management, providing excellent opportunities for teaching, research and cutting-edge innovation. He is also the author and co-author of many peer-reviewed papers and books. He has a simple motto – Treat every patient as if they were a member of your family!

Correspondence to: Dr Nat Padhiar at nat.padhiar@virgin.net or to London SportsCare, London Independent Hospital, 1 Beaumont Square, London E1 4NL.