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The painful nonruptured tendon: clinical aspects

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Tendon injuries account for a substantial proportion of overuse injuries in competitive and recreational sports participants, as well as in individuals whose jobs require repetitive activity. Overuse type injuries account for 30% to 50% of all sports injuries, and in many endurance and power sports tendon injuries are clearly the most frequent reason for interruptions to training and competition [1]. Because tendon problems are so common, and not easily managed, this issue of Clinics is devoted to them. This article aims to provide an understanding of the pathology underlying the conditions before outlining current evidence for clinical assessment and treatment of tendinopathies.

Tendon pathology: the myth of "tendinitis"

So the reader can better understand the abnormalities found in symptomatic tendons, we first review the macroscopic and light microscopic appearance of normal tendon. More detailed descriptions of tendon anatomy exist elsewhere [2,3].

Normal tendon

Tendons are anatomic structures interposed between muscles and bones that transmit the force created in muscle to bone and make joint movement possible.

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The basic elements of tendon are collagen bundles, cells, and ground substance or extracellular matrix, a viscous substance rich in proteoglycans. Collagen provides tendon with tensile strength; ground substance provides structural support for the collagen fibers and regulates the extracellular assembly of procollagen into mature collagen [4]. Tenocytes are flat, tapered cells sparingly distributed among the collagen fibrils that synthesize both the ground substance and the procollagen building blocks of protein.

Collagen is arranged in hierarchical levels of increasing complexity, beginning with tropocollagen, a triple-helix polypeptide chain, which unites into fibrils, fibers (primary bundles), fascicles (secondary bundles), tertiary bundles, and finally the tendon itself [2,3].

The entire tendon is covered by the epitenon, a fine, loose connective tissue sheath containing the vascular, lymphatic and nerve supply. More superficially, the epitenon is surrounded by paratenon, a loose areolar connective tissue consisting essentially of type I and type III collagen fibrils, some elastic fibrils, and an inner lining of synovial cells. Together, the paratenon and epitenon are sometimes called the peritendon [2].

The classic two-layered synovial tendon sheath is only present in certain tendons as they pass areas of increased mechanical stress. The outer layer is the fibrotic (ligamentous) sheath and the inner layer is the synovial sheath, which consists of thin visceral and parietal sheets [2]

The myotendinous junction is a highly specialized anatomic region in the muscle-tendon unit where tension generated by muscle fibers is transmitted from intracellular contractile proteins to extracellular connective tissue proteins (collagen fibrils) [2]. As this region is rarely affected by tendinopathy, its complex ultrastructure is not discussed further here, but the interested reader is referred elsewhere [2].

The osteotendinous junction is a specialized region in the muscle-tendon unit where the tendon inserts into a bone. In the osteotendinous junction, the viscoelastic tendon transmits the force into a rigid bone. The region has been described as containing four light-microscopic zones: (1) tendon, (2) fibrocartilage, (3) mineralized fibrocartilage, and (4) bone [5].

Under the light microscope, normal tendon consists of dense, clearly defined, parallel and slightly wavy collagen bundles. Collagen has a characteristic reflective appearance under polarized light. Between the collagen bundles there is a fairly even sparse distribution of cells with thin wavy nuclei. There is an absence of stainable ground substance and no evidence of fibroblastic or myofibroblastic proliferation. Tendon is supplied by a network of small arteries oriented parallel to the collagen fibers in the endotenon [2,3].

The myth of "tendinitis"

Until recently, if a patient presented with a history of exercise-related pain and tenderness at one of the common sites of tendinopathy (the Achilles, patellar, rotator cuff, or elbow tendons), and if history and examination features suggested

that pain was emanating from the tendon, the patient would most likely have been diagnosed as having "tendinitis," an inflammatory condition of the tendon. This was proven not to be the case as long ago as 1976, when Giancarlo Puddu of Rome examined the Achilles tendon of symptomatic runners and showed that inflammatory cells were absent. This research finding has not been widely incorporated into clinical practice, however, until more recent years [6–10]. Here we summarize the findings of histopathological examination at commonly reported sites of tendon pain.

Achilles tendon

Histopathologic examination of symptomatic Achilles tendons reveals degeneration and disordered arrangement of collagen fibers. Collagen fibers are thinner than normal and separated by large mucoid patches and vacuoles. In addition to collagen fibers in symptomatic Achilles tendons being abnormal in themselves, the characteristic hierarchical structure is also lost [6]. Also, there is an increase in Alcian-blue-staining ground substance. Whether this precedes or follows the collagen separation is currently under investigation. Symptomatic Achilles tendons reveal increased vascularity [11]. It is important to note that inflammatory lesions [6,11], intracellular lipid aggregates, and acellular necrotic areas were "exceptional" and not regarded as normal elements of the degenerative process [6]. The authors concluded that "the absence of inflammation and the poor healing response demonstrate a state of degeneration that conforms to the histopathology described by previous authors in total ruptures and in chronic tendinopathy" [6].

With respect to the paratenon, Kvist et al [12,13] found evidence of mucoid degeneration, fibrosis, and vascular proliferation, with only a slight inflammatory infiltrate—similar to other series. Astrom and Rausing [6] found virtually no evidence of paratenonitis in their series of Achilles tendon specimens. These differences may be explained by the fact that Kvist et al did not report pathology of the tendon itself, and studied more active, younger patients. Thus, paratenonitis is not a prerequisite for Achilles tendon symptoms in a population of recreational sportspeople and office workers. The major lesion in chronic Achilles tendinopathy "is a degenerative process characterized by a curious absence of inflammatory cells and a poor healing response" [6].

Patellar tendon

Macroscopically, the patellar tendon of patients with patellar tendinopathy (also commonly called "jumper's knee") contain yellow-brown and disorganized tissue in the deep posterior portion of the patellar tendon adjacent to the lower pole of the patella [14–16]. Under the light microscope, the tendons of patients suffering jumper's knee do not consist of tight parallel collagen bundles, but instead are separated by increased mucoid ground substance that gives them a disorganized and discontinuous appearance. Clefts in collagen and occasional necrotic fibers may suggest microtearing. There is loss of the characteristic

reflective polarized light appearance [17]. A consistent feature across studies of the patients with chronic patellar tendinopathy was the finding of mucoid degeneration with variable fibrosis and neovascularization. The collagen producing tenocytes themselves lost their fine spindle shape and nuclei were more rounded and sometimes chondroid in appearance, amounting to fibrocartilaginous metaplasia. Small vessel ingrowth was also evident. As with Achilles tendinopathy, patellar tendinopathy occasionally revealed erythrocytes and positive staining for iron pigment, but the histopathology remained identical to those cases without rupture [17]. As with the Achilles tendon pathology described above, myofibroblasts are prominent [18,19] and board-certified pathologist authors have found inflammatory cells to be absent [17,18]. In addition, there is disruption of the bone tendon junction and a propensity to develop cystic lesions where active repair is not occurring.

Extensor carpi radialis brevis tendon

The term ''tennis elbow'' was used for 100 years before the pathology of the extensor carpi radialis brevis tendon was associated with lateral tennis elbow [20]. At surgery the symptomatic extensor carpi radialis brevis contained disrupted collagen fibers, increased cellularity, and neovascularisation [20]. Acute inflammatory cells were almost always absent from the tendon, but a mild sprinkling of chronic inflammatory cells were noted in supportive or adjacent tissues. When chronic inflammatory cells were present, they resulted from repair of partial tears [20]. A recent study in 20 cases of chronic (6–48 months) lateral ''epicondylitis'' confirmed that there was no histopathologic evidence of either acute or chronic inflammation in any of the specimens [21]. The histopathology reported in lateral tennis elbow also exists in medial tennis elbow [22,23]. As at the patellar tendon, there is disruption of the bone tendon junction, as well as a propensity to develop cystic lesions where active repair is not occurring.

Rotator cuff

Histopathology of symptomatic rotator cuff tendons reveals collagen disruption and fibrocartilaginous metaplasia [24,25], as well as cellular distortion and necrosis, calcium deposition, fibrinoid thickening, hyalinization, fibrillation, and microtears. There is loss of the characteristic crimped pattern of tendon, and parallel bundles of collagen separate and become disorganized [24–26]. Characteristically, the symptomatic rotator cuff reveals ''disruption of fascicles, formation of foci of granulation tissue, dystrophic calcification, thinning of fascicles, associated with cell and vessel proliferation'' [24]. Hypervascularity of the degenerative rotator cuff has also been reported by others [27,28].

Tendinosis

These data clearly indicate that painful, overuse tendon injury is due to tendinosis—the histologic entity of collagen disarray, increased ground sub-

Table 1
Bonar's classification of tendinopathies [7]

Pathological diagnosis	Concept (macroscopic pathology)	Histologic finding
Tendinosis	Intratendinous degeneration (commonly due to aging, microtrauma, vascular compromise)	Collagen disorientation, disorganization and fiber separation by an increase in mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularization and focal necrosis or calcification
Tendinitis/Partial rupture	Symptomatic degeneration of the tendon with vascular disruption and inflammatory repair response	Degenerative changes as noted above with superimposed evidence of tear, including fibroblastic and myofibroblastic proliferation, hemorrhage and organizing granulation tissue.
Paratenonitis	"Inflammation" of the outer layer of the tendon (paratenon) alone, whether or not the paratenon is lined by synovium	Mucoid degeneration in the areolar tissue is seen. A scattered mild mononuclear infiltrate with or without focal fibrin deposition and fibrinous exudate
Paratenonitiswith tendinosis	Paratenonitis associated with intratendinous degeneration	Degenerative changes as noted in tendinosis with mucoid degeneration with or without fibrosis and scattered inflammatory cells in the paratenon alveolar tissue

From Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common overuse tendon conditions: update and implications for clinical management. Sports Med 1999;27:393–408; with permission.

stance, neovascularization, and increased prominence of myofibroblasts. Bonar's classification is tabled (Table 1).

Although the term "tendinosis" was first used by German workers in the 1940s, its recent usage results from the work of Puddu et al [29]. Tendinosis is tendon degeneration without clinical or histological signs of an inflammatory response. It may result from the pathological process of apoptosis [30] or other mechanisms of failed healing. It appears that tendinosis is the major, and perhaps

Table 2
Implications of the diagnosis of tendinosis compared with tendinitis

Trait	Overuse tendinosis	Overuse tendinitis
Prevalence	Common	Rare
Time for full recovery (initial presentation)	2–3 mos	Several days to 2 wks
Time for full recovery (chronic)	3–6 mos	4–6 wks
Likelihood of full recovery to sport from chronic symptoms	~80%	99%
Focus of conservative therapy	Encourage collagen synthesis, maturation, and strength	Anti-inflammatory modalities and drugs
Role of surgery	Excise abnormal tissue	Not known
Prognosis for surgery	70%–85%	95%
Time to recover from surgery	4–6 mos	3–4 wks

the only clinically relevant chronic tendon lesion [7], although minor histopathologic variations may exist in different anatomical sites.

The finding that the clinical tendon conditions in sportspeople are due to tendinosis is not new. Writing about the tendinopathies in 1986, Perugia et al noted the "remarkable discrepancy between the terminology generally adopted for these conditions (which are obviously inflammatory because the ending "-itis" is used) and their histopathologic substratum, which is largely degenerative" [31]. Table 2 summarizes the main differences that a presumptive diagnosis of tendinosis implies, compared with a diagnosis of "tendinitis."

Clinical assessment of the painful tendon

As in all branches of medicine, the keys to diagnosis remain history and physical examination. Imaging has an important, but secondary role [32,33].

History

The diagnosis of tendon pain is generally straightforward, but exceptions exist. The classic presentation is one of increasing pain at the site of the offending tendon, often with recognition that there has been an increase inactivity. The pain is usually load-related. In very early tendinopathy, pain may be present at the beginning of an activity and then "warm-up" (disappear) during activity itself, only to reappear when cooling down if the activity is prolonged, or to be more severe on subsequent attempts to be active. The patient can usually localize the pain rather clearly and the pain is described as "severe" or "sharp" during the early stages and sometimes as a "dull ache" once it has been present for some weeks. Pain that is vague in nature and distribution or referred to as "tingling" or as a "numb feeling" should alert the physician that the problem may not primarily emanate from tendon. Tendon pain itself usually does not radiate, although referred pain can contribute to the development of a secondary tendon problem (classically, a neck problem contributing to elbow pain; see below). Aggravating activities generally are those that increase load on the tendon in question, relieving factors include relative rest, ice, and nonsteroidal anti-inflammatory drugs and other analgesic medications. In any case of suspected tendinopathy, the history should include questions about general health, not only because this is a good clinical practice, but also because tendinopathy can mark the presence of an underlying spondyloarthropathy (eg, psoriasis) [34]. Symptoms such as previous gastrointestinal infection or sexually transmitted diseases should alert the clinician to the possibility of a secondary tendinopathy. Many patients who have well-established psoriasis are not aware that tendinopathy is an extradermal manifestation of this condition.

Specific differential diagnoses to consider at various anatomical regions are listed in Table 3. The specifics of these conditions are outside the scope of this article but can be found elsewhere [35].

Table 3
Specific differential diagnoses to consider when patients present with 'tendinopathy' at various anatomical regions

Region	Differential diagnoses to consider	Keys to correct diagnosis
Achilles	Posterior impingement, bursitis, referred pain (less common)	Careful palpation, passive plantarflexion test for posterior impingement
Patellar	Patellofemoral pain	Careful palpation
Lateral elbow	Referred pain from the cervical spine (common), nerve entrapments in the forearm	Careful examination of the cervical spine, awareness of forearm nerve entrapments
Rotator cuff	AC joint pain and osteolysis of the distal clavicle, shoulder instability, and glenoid labral tears	Examination of the AC joint, assessment for instability, and labral tests
Tibialis posterior – medial ankle	Flexor hallucis longus tendinopathy	Careful palpation – FHL tendinopathy is generally at the tunnel; tibialis posterior tendinopathy is generally at the navicular insertion

Physical examination

Physical examination must include tests that load the tendon to reproduce pain and other loading tests that load alternative structures. After consideration of the history and the behavior of the tendon pain under load, careful palpation together with knowledge of surface anatomy allows a confident clinical primary diagnosis of tendinopathy. Palpation should reveal focal tenderness that essentially reproduces the patient's pain. At the Achilles this may be 3 cm to 5 cm above the calcaneal insertion (classic midportion tendinopathy), or less commonly at the insertion (insertional tendinopathy) itself. Many experienced clinicians find the latter condition responds less well to treatment, and thus distinguish these two types of Achilles tendinopathy because of their very different prognoses. Insertional tendinopathies at less commonly affected sites (eg, Achilles insertion, patellar tendon distal insertion) should increase the clinician's index of suspicion to the possibility that a spondyloarthopathy is involved (see history, above) [34]. The tenderness of patellar tendinopathy is generally found on the deep surface of the proximal attachment of the patellar tendon. This can be palpated when the knee is in about 30° of flexion and the quadriceps muscle is totally relaxed. Note that some tenderness is usual at this location, but moderate or severe tenderness is associated with pathology [36]. Similarly, the cardinal signs of lateral and medial elbow tendinopathy is tenderness at the origins of the elbow extensors and flexors, respectively. As mentioned above (history), the prevalence of cervical pathologies (such as joint hypomobility) means that the neck must be examined carefully in all cases of shoulder and elbow tendinopathy. A skilled physical therapist or clinician aware of upper limb tension tests [37] should examine

difficult cases of elbow tendinopathy, as referred pain and cervical contribution is often not in the classic radicular pattern. The clinician must understand the role of somatic pain in both the upper and lower limbs so that somatic pain is not overlooked inadvertently. Once this diagnosis has been made, however, further examination should aim to identify why the tendinopathy has arisen, and this generally requires expertise in understanding sporting biomechanics.

Imaging assessment

In apparently straightforward cases of tendinopathy, many expert clinicians feel that the diagnosis can be made confidently clinically, thus obviating the need for any investigation. In cases where the history and examination may not be typical, or the clinical recovery is not as expected, both ultrasound and magnetic resonance (MR) imaging provide a great deal of morphological information about tendons. Although there is an association between tendon morphology and symptoms, there are many cases where structure does not parallel pain, so the clinician must interpret imaging findings bearing symptoms in mind. Detailed discussion of this topic is beyond the scope of this section, but the reader is referred elsewhere for further discussion of lack of clinical correlation between tendon imaging and symptoms [38–43].

Achilles tendon degeneration is evident as increased signal on MR imaging [44] and hypoechoic regions on ultrasound [45–47]. Regions of tendinosis produce increased signal on MR imaging [17,18] and hypoechoic regions on ultrasound [17,47]. At the shoulder, elbow, and patellar tendons, MR delineates tendon pathology [21], but ultrasound can be technically challenging, as the tendon insertion is adjacent to bone, which limits image quality [48]. At the shoulder, however, ultrasound comes into its own, as the skilful operator can use the dynamic capacity of the investigation to assess the rotator cuff in great detail. Regions of tendon degeneration produce high-intensity signal on MR imaging [49]. A high proportion of asymptomatic volunteers (89%–100%) have regions of high signal in the rotator cuff tendon [43,50]. This suggests, but does not prove, that subclinical tendon degeneration may be a relatively common phenomenon amongst asymptomatic individuals. The take-home message for physicians is that patients must be managed on clinical grounds, not according to a predetermined, imaging-driven algorithm. If imaging reveals normal morphology but symptoms have been present for some time and are moderately severe, however, the diagnosis of tendinopathy is less likely and other pathologies should be considered.

Conservative management of tendinopathies

Although many conservative treatments have been used to treat tendinopathies, there have been few randomized controlled trials. In this section we review the evidence that supports common methods used to treat tendinopathies in clinical practice.

Relative rest

Relative rest may be an important component of treatment of tendinopathy, given that there is frank structural damage to the tendon. Collagen healing may require longer than has traditionally been afforded a patient with tendinopathy. In sports, tendons must often sustain more than 10 times body weight [51], yet the tissue has a slow metabolic rate, as evidenced by its having only 13% of the oxygen uptake of muscle and requiring more than 100 days to synthesize collagen [52]. Thus, symptom relief in tendinosis may take months rather than weeks.

There are no objective guidelines as to how much rest is optimal. Traditionally, pain has been used as a guide. Thus, the patient is generally advised to undertake relative rest until he or she can perform activities of daily living essentially free of pain. After that time, strengthening (see below) can begin. Continued rest allows a decrease in musculotendinous strength and less ability for the unit to function optimally. Alfredson has postulated that pain is a normal part of the healing process, however, and encourages patients to perform certain strengthening exercises unless they have "debilitating" pain [53].

Strengthening

Strengthening, particularly eccentric strengthening, has been advocated as a treatment of tendon overuse conditions since the early 1980s [54–56]. Clinical studies point to the efficacy of eccentric strengthening regimens [54,56–59]. Mechanical loading accelerates tenocytes metabolism and may speed repair [60]. These data provide rationale for judicious, progressive strengthening in the treatment of tendinosis [9,61,62].

Nonsteroidal anti-inflammatory drugs (NSAIDs)

On theoretical grounds one would predict that the anti-inflammatory action of NSAIDs would have no therapeutic effect in tendinosis, a noninflammatory condition. Furthermore, the analgesic effect of NSAIDs [63] may permit patients to ignore early symptoms, further damage tendon, and thus delay definitive healing. In practice, there is little evidence that NSAIDs should play a role in management of tendinopathy [64], and Astrom et al found no beneficial effect of NSAIDs in patients with Achilles tendinopathy [65]. Until there is evidence to suggest otherwise, it seems that NSAIDs are inappropriate in the management of uncomplicated tendinopathy.

Corticosteroids

The role of corticosteroids in treatment of tendon conditions has been the subject of considerable debate [66,67] but very few well-designed studies [68]. Further studies are desperately needed. It is clear that corticosteroid injection that inadvertently enters into tendon tissue leads to cell death and tendon atrophy [69]. As tendinosis is not an inflammatory condition, the rationale for using cortico-

steroids needs reassessment, particularly as corticosteroids inhibit collagen synthesis [70,71] and decrease load to failure [72]. Corticosteroids, however, provide short-term pain reduction by mechanisms that are poorly understood [40].

Other pharmaceutical agents

The protease inhibitor aprotinin and other drugs such as low-dose heparin have been used in the management of peri- and intratendinous pathology [73]. More recently, researchers have used a sclerosing agent, poldicanol, better known for its role in treatment of varicose veins and esophageal varices, to ablate the neovessels associated with tendinopathy [74,75]. Although preliminary results are encouraging, these results must be considered preliminary at the time this journal goes to press.

Modalities

Physiotherapists employ a wide variety of modalities, including ultrasound, laser, and heat, to treat tendinopathies [76]. Modalities are proposed to decrease inflammation and promote healing, but there is very limited evidence to support these claims. Studies of severed tendons have shown that ultrasound increases collagen synthesis in fibroblasts [77] and increases tensile strength of healing tendon [78,79] and has little effect on inflammation [80,81]. After rabbit Achilles' tenotomy there was a 26% greater collagen concentration in tendons that had received laser photostimulation compared with those that received sham laser [82]. Biomechanical and biochemical measures of tendon healing were improved by a combination of ultrasound, laser, and electrical stimulation of rabbit Achilles tendons after tenotomy and suture repair [83]. All of these effects would help to reverse the pathology of tendinosis by stimulating fibrosis and repair; however, relevant human data are required.

Cryotherapy

Cryotherapy may decrease the extravasation of blood and protein from the neovessels found in painful tendons [74,76]. It would also be expected to decrease the metabolic rate of tissue and decrease tendon temperature after exercise. Thus, ice may play a role in treating tendinosis. Ice would be particularly effective in treating tendinopathies where paratenonitis is associated with tendinosis (eg, some Achilles tendinopathies). As ice may mask pain in tendinosis and increases tissue stiffness, it ought not be used before sports participation.

Braces and supports

Braces and supports are used as an adjunct to treatment of elbow and knee tendinopathies. Braces may act to keep the tendon warm during sporting performance but would not be expected to protect the tendon, except by aiding proprioception.

Orthotics

Orthotics are commonly prescribed in the treatment of Achilles tendinopathy, and less commonly for jumper's knee. Ankle plantarflexion peak torque differs between patients with Achilles tendinopathy and controls [84], and this would provide a rational mechanism of action for orthotics in this condition. Further studies of biomechanics in athletes with tendinopathies and the role of orthotics are required.

Technique correction

As technique correction aims to decrease the load that is placed through a tendon, it clearly has a place in managing the tendinosis associated with overuse conditions [9,85]. For example, attention to a tennis player's backhand drive technique can play a major role in treating tendinosis at the lateral elbow [86], and adjusting jumping technique in volleyballers may contribute to treatment of patellar tendinosis [87].

Surgery

The aims of surgery have been outlined elsewhere [88], and clinical research suggests it can be effective in a proportion of cases [89,90]. Why surgery promotes healing of tendon is still not understood. It has been argued that perhaps the postoperative healing response and the careful progression of rehabilitation after surgery rather than the surgery itself causes improvement in the patient's condition, but the time course of healing after surgery in Coleman's study [91] argues against this being the only cause. An important point for clinicians to remember and to emphasize to patients is that surgery, when successful, does not permit immediate return to sport. Prospective outcome studies (likely to be more accurate on this than retrospective studies or clinical impression) indicate that elbow surgery may permit patients' return to sport in 4 to 6 months [20], Achilles surgery may do so in 6 to 9 months [92], and patellar tendon surgery may do so in 9 to 12 months [93]. Rotator cuff surgery is difficult to categorize, as the time to recover depends on the type of surgery, and there are many different procedures for shoulder tendinonpathy. Nevertheless, an elite throwing or racquet sports player is unlikely to return to full competition at the same level after a tendinopathy procedure (eg, repair of partial tear) for a minimum of 6 to 9 months.

Summary

Tendon conditions cause a great deal of morbidity in both elite and recreational athletes, and outcome of treatment is often unsatisfactory. Evidence that the common clinical conditions (eg, Achilles, patellar, elbow and rotator cuff tendinopathies) are due to tendinosis has been present for many years, yet the misnomer "tendinitis" is still widely used for these conditions in clinical

practice. Clinical practice remains very different from evidence-based recommendations [8], but this is a common challenge in medical practice. Thus, in addition to further research in an area of medicine rife for such endeavor, there must be attention to knowledge translation—ensuring that the patient benefits from what is already known.

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