

Overuse Tendinosis, Not Tendinitis

Part 1: A New Paradigm for a Difficult Clinical Problem

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This is the first of two articles on tendinopathies by Dr Khan and colleagues; [the second](#), on patellar tendinopathy, will appear in a subsequent issue.

In Brief: Overuse tendinopathies are common in primary care. Numerous investigators worldwide have shown that the pathology underlying these conditions is tendinosis or collagen degeneration. This applies equally in the Achilles, patellar, medial and lateral elbow, and rotator cuff tendons. If physicians acknowledge that overuse tendinopathies are due to tendinosis, as distinct from tendinitis, they must modify patient management in at least eight areas. These include adaptation of advice given when counseling, interaction with the physical therapist and athletic trainer, interpretation of imaging, choice of conservative management, and consideration of whether surgery is an option.

Physicians in primary care, sports medicine, and orthopedics see patients with overuse tendon conditions nearly every day. Tendon conditions are not restricted to competitive athletes but affect recreational sports participants and many working people, particularly those doing manual labor (1). Unfortunately, these disorders are not only common, they can also be recalcitrant to treatment.

One factor that may interfere with optimal treatment is that common tendinopathies may be mislabeled as tendinitis. Advances in the understanding of tendon pathology indicate that conditions that have been traditionally labeled as Achilles tendinitis, patellar tendinitis, lateral epicondylitis, and rotator cuff tendinitis are in fact tendinosis. An increasing body of evidence supports the notion that these overuse tendon conditions do not involve inflammation.

If this is correct, then the traditional approach to treating tendinopathies as an inflammatory "tendinitis" is likely flawed. The recommendations presented here reflect the current scientific data and will help physicians avoid common misconceptions about tendinopathies and their management (table 1) (2-11).

TABLE 1. Common Misconceptions About Tendinopathies and Their Management

Misconception	Evidence-Based Finding
Tendinopathies are self-limiting conditions that take only a few weeks to resolve (2)	Tendinopathies often prove recalcitrant to treatment and may take months to resolve

Imaging appearance can predict prognosis

Imaging does not predict prognosis; it adds to the likelihood of a diagnosis of tendinopathy but does not prove it (3-5)

Cyst-like ultrasonographic abnormalities in tendons are indications for surgery

Surgery should be based on clinical grounds; cyst-like ultrasonographic findings can be found in asymptomatic athletes (5)

Surgery provides rapid relief of symptoms in almost all subjects

After surgery, return to sport takes a minimum of 4-6 mo (6-11); not all patients do well (8,9)

Tendinosis: A Noninflammatory Disorder

Pathology. The pathology of the major tendinopathies has been well described and is based on examination of surgical specimens. In studies of the Achilles (11), patellar (12), lateral elbow (9,13), medial elbow (14), and rotator cuff tendons (15), tissue appeared remarkably consistent. Macroscopically, abnormal tissue examined at surgery shows the tendon to be dull-appearing, slightly brown, and soft. Normal tendon tissue is white, glistening, and firm.

When examined under a light microscope, abnormal tendon from patients with chronic tendinopathies differs from normal tendon in several key ways. It has a loss of collagen continuity (figure 1) and an increase in ground substance, vascularity, and cellularity (figure 2: not shown). Cellularity results from the presence of fibroblasts and myofibroblasts (figure 3: not shown), not inflammatory cells. Thus, in patients who have chronic overuse tendinopathies, inflammatory cells are absent.

Figure 1: Courtesy of S. F. Bonar, MD

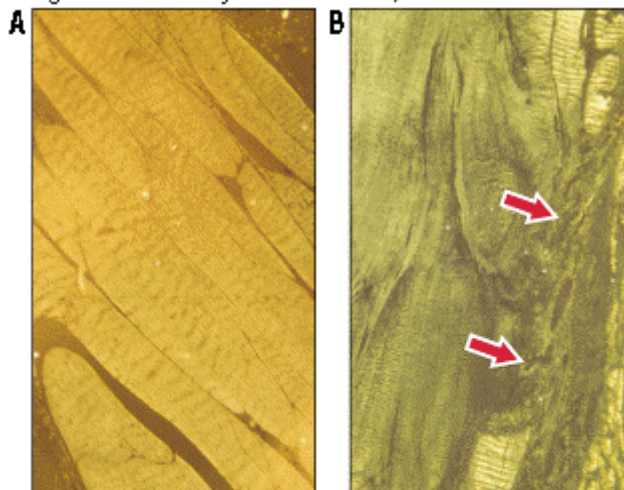


FIGURE 1. Histopathologic comparison of normal tendon and abnormal tendon as seen under a polarized light microscope (x 100). Normal patellar tendon (A) consists of tightly bundled collagen fibers with a characteristic golden reflectivity. In an abnormal patellar tendon from a postsurgical patient with chronic tendinopathy (B), loss of collagen continuity, loss of reflectivity, and a frank collagen defect (arrows), are easily seen.

Tendinosis vs tendinitis. Key features of tendinosis are described in table 2. Tendinitis is a rather rare condition but may occur occasionally in the Achilles tendon in conjunction with a primary tendinosis. Paratenonitis, one of the disorders in the differential diagnosis, is a condition of inflammation of the outer layer of the tendon (paratenon) alone, whether or not the paratenon is lined by synovium. It is commonly associated with intratendinous degeneration (16) and produces the "crepitus" that is easily felt in some cases of Achilles paratenonitis. Unfortunately, distinguishing tendinosis from the rare tendinitis is difficult clinically. But because tendinosis is far more likely, our advice is to treat patients initially as if tendinosis were the diagnosis.

TABLE 2. Bonar's Classification of Overuse Tendon Conditions (17)

Pathologic Diagnosis	Macroscopic Pathology	Histologic Finding
Tendinosis	Intratendinous degeneration commonly due to aging, microtrauma, or vascular compromise	Collagen disorientation, disorganization, and fiber separation by increased mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularization, and focal necrosis or calcification
Partial rupture or tendinitis	Symptomatic degeneration of the tendon with vascular disruption, 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 is seen in the areolar tissue: a scattered mild mononuclear infiltrate with or without focal fibrin deposition and fibrinous exudate
Paratenonitis with 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

Tendinosis as a concept. Although recent research has clearly demonstrated the presence of tendinosis in chronically injured tendon tissue, this is not a new discovery (17). The term tendinosis was first used by German researchers in the 1940s; the term's recent usage results from Puddu et al (18) and Nirschl and Pettrone (9). Writing about tendinopathies in 1986, Perugia et al (19) noted the "remarkable discrepancy between the terminology generally adopted for these conditions (which are obviously inflammatory since the ending 'itis' is used) and their histopathologic substratum, which is largely degenerative."

Thus, physicians must shift their perspective and acknowledge that tendinosis is the pathology being treated in most cases and that treatment needs to combat collagen breakdown rather than inflammation. Tendinosis may require a reasonable period of relative rest and attention

to strengthening with the aim of first breaking the tendinosis cycle. Once this is done, the patient uses modalities that optimize collagen production and maturation so that the tendon achieves the necessary tensile strength for normal function.

Clinical Significance of the Diagnosis of Tendinosis

If we accept that a patient with overuse tendinopathy has an injury that is due to collagen degeneration, then the diagnosis has repercussions (table 3) on at least eight aspects of our practice.

TABLE 3. Implications of the Diagnosis of Tendinosis Compared With Tendinitis

Trait	Overuse Tendinosis	Overuse Tendinitis
Prevalence	Common	Rare
Time for recovery, early presentation	6-10 wk	Several days to 2 wk
Time for full recovery, chronic presentation	3-6 mo	4-6 wk
Likelihood of full recovery to sport from chronic symptoms	~80%	99%
Focus of conservative therapy	Encouragement of 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 mo	3-4 wk

1. Imaging. Tendinopathies are very well visualized with both magnetic resonance imaging (MRI) and diagnostic ultrasound (2). Histopathologic studies prove that the characteristic imaging appearances (see figure 1) are due to tendinosis (12,20,21). We use MRI to confirm our clinical diagnosis and to demonstrate to the patient that in tendinosis, tendon collagen has lost continuity.

2. Patient education. The physician should take the time to explain and illustrate the pathology of tendinosis, especially since textbooks and Web sites have yet to embrace this pathology and its clinical implications. We find that patients with chronic tendinopathy need no

convincing of the veracity of tendinosis. They are generally relieved to find a scientific rationale behind their prolonged symptoms. Patients who have symptoms of short duration but are still able to "warm up" the injury and engage in sports are the ones who need the most education. They are likely to try to continue playing without undergoing appropriate treatment, and thus worsen the tendinosis.

3. Biomechanical deloading. Because tendinosis results from collagen degeneration and, generally, mechanical overload, it is vital that the physician establish *why* this occurred. Training errors are a common cause (22), but in some instances a more subtle mechanism underlies tendinosis. Thus, it is important to assess any equipment being used (eg, running shoes, tennis racket), examine movement biomechanics (eg, running, throwing motion, stroke pattern), and diagnose and treat any muscle imbalances (23). The details of biomechanical correction are outside the scope of this paper, but their importance cannot be overemphasized in treatment.

4. Anti-inflammatory strategies. Common strategies to reduce inflammation include cryotherapy, electrotherapeutic modalities, nonsteroidal anti-inflammatory drugs (NSAIDs), and corticosteroid injections.

Patients with tendinosis may benefit from cryotherapy because ice has a vasoconstrictive role, and abnormal neovascularization is a feature of the pathology (see figure 2A: not shown). Because a strong clinical impression exists that icing is helpful in tendinopathies, this modality should not be discarded.

Physical therapies that purport to reduce inflammation would not help treat tendinosis if that were their only role. Enwemeka and others (24,25), however, have shown that electrotherapy (eg, laser, high-voltage galvanic stimulation) can stimulate collagen synthesis in the laboratory setting. Clearly this effect would be favorable to the treatment of tendinosis.

Almekinders and Temple (26) have published a thorough review of the role of NSAIDs and corticosteroids in treating tendinopathy and found little evidence that they were helpful. It is not known whether the antiprostaglandin action of NSAIDs and cyclooxygenase-2 inhibitors helps or hinders tendon repair through mechanisms other than the pathways associated with inflammation.

The role of corticosteroid injection or iontophoresis in tendinosis remains controversial (27). In brief, because tendinosis is not an inflammatory condition, the same arguments as those for NSAIDs apply. Further, given the known deleterious effect of corticosteroid injection into tendon and inhibition of collagen repair when the drug is injected around tendon, this treatment has lost favor. Astrom (28) found that corticosteroid injection was the only predictor of partial tendon tears in a large series of cases of chronic Achilles tendinosis that eventually required surgery. This being stated, it is clear that in some cases, corticosteroid injection provides short-term but rapid symptom relief.

5. Load-decreasing devices. Because tendinosis results from excessive load on collagen, braces and supports that attenuate load through the tendon may benefit the patient. Braces have gained clinical support to unload the tendon at both the elbow and the knee. Heel lifts unload the Achilles tendon, and orthoses serve to control pronation and whip at both Achilles and patellar tendons. Further research into the efficacy of these devices is required because they have not yet undergone randomized, controlled trials. They do, however, have a solid biomechanical rationale for therapy.

6. Interaction with the physical therapist. It is important that the physical therapist and physician have a similar understanding of the pathology of tendinopathies and a parallel approach to management. When referring a patient for physical therapy, it is essential that both therapist and patient have a realistic time frame for rehabilitation. A randomized, controlled trial of strengthening treatment in Achilles tendinopathy (29) and data from other cases (1) suggest that for patients who first present with short-duration tendon symptoms, return to full sporting competition takes, on average, 2 to 3 months. A patient with chronic

symptoms may require 4 to 6 months to achieve a similar outcome. Clearly, these guidelines will vary for individual patients, but they are supported strongly by the literature (1,8).

7. Appropriate strengthening. Eccentric strengthening programs have a long track record of clinical effectiveness (22,29,30), and recent research adds further scientific support (31,32). It is likely that specific eccentric training drills result in tendon strengthening by stimulating mechanoreceptors in tenocytes to produce collagen, and thus help reverse the tendinosis cycle (figure 4: not shown) (33). Collagen production is probably the key cellular phenomenon that determines recovery from tendinosis. Animal experiments have revealed that loading the tendon improves collagen alignment and stimulates collagen cross-linkage formation, both of which improve tensile strength (34). Therefore, the clinical success of tendon strengthening programs has a demonstrable biological basis that is supported by clinical (32) and laboratory evidence (35).

8. Surgery as a last resort. Surgery has been considered the treatment of last resort for tendinopathies, and this certainly applies, if not more so, for a confirmed case of tendinosis. Surgery can be used to excise tissue affected by tendinosis, but surgery has not been proven to stimulate collagen synthesis or maturation. Thus, the tendon that has had surgery requires time for repair and strengthening. Reviews suggest that surgery in tendinosis has a 75% to 85% success rate, and for some tendons this figure may well be a very-best-case estimate (7,8).

Therefore, an important implication of tendinopathy's underlying pathology being tendinosis is that conservative management must progress slowly. If the initial prognosis the patient receives is realistic, it is less likely that the patient will attempt to return to sport prematurely, suffer re-injury, and thus, "fail" conservative management. Because surgical treatment of tendinosis is not without failure, and recovery takes a minimum of 4 to 6 months, this treatment should be reserved for failure of a high-quality program of conservative management.

The New Paradigm

Tendinopathies have provided long-lasting frustration for both patients and physicians, and attributing the pathology to tendinitis rather than tendinosis may have contributed to this problem. Physicians must acknowledge that the cause is most often due to tendinosis, rather than tendinitis, and treat the problem using a fundamentally different paradigm. Advice and suggestions for patients along these lines of clinical thought can help them recover more quickly and avoid surgery.

References

1. Cook JL, Khan KM, Harcourt PR, et al: A cross-sectional study of 100 athletes with jumper's knee managed conservatively and surgically: The Victorian Institute of Sport Tendon Study Group. *Br J Sports Med* 1997;31(4):332-336
2. Lian O, Holen KJ, Engebretsen L, et al: Relationship between symptoms of jumper's knee and the ultrasound characteristics of the patellar tendon among high level male volleyball players. *Scand J Med Sci Sports* 1996;6(5):291-296
3. Shalaby M, Almekinders LC: Patellar tendinitis: the significance of magnetic resonance imaging findings. *Am J Sports Med* 1999;27(3):345-349
4. Cook JL, Khan KM, Harcourt PR, et al: Patellar tendon ultrasonography in asymptomatic active athletes reveals hypoechoic regions: a study of 320 tendons: Victorian Institute of Sport Tendon Study Group. *Clin J Sport Med* 1998;8(2):73-77
5. Anderson DL, Taunton JE, Davidson RG: Surgical management of chronic Achilles tendinitis. *Clin J Sport Med* 1992;2(1):38-42

6. Alfredson H, Pietila T, Lorentzon R: Chronic Achilles tendinitis and calf muscle strength. *Am J Sports Med* 1996;24(6):829-833
7. Coleman BD, Khan KM, Maffulli N, et al: Studies of surgical outcome after patellar tendinopathy: clinical significance of methodological deficiencies and guidelines for future studies: Victorian Institute of Sport Tendon Study Group. *Scand J Med Sci Sports* 2000;10(1):2-11
8. Coleman BD, Khan KM, Kiss ZS, et al: Outcomes of open and arthroscopic patellar tenotomy for chronic patellar tendinopathy: a retrospective study. *Am J Sports Med* 2000;28(2):183-190
9. Nirschl RP, Pettrone FA: Tennis elbow: the surgical treatment of lateral epicondylitis. *J Bone Joint Surg (Am)* 1979;61(6):832-839
10. Budoff JE, Nirschl RP, Guidi EJ: Debridement of partial thickness tears of the rotator cuff without acromioplasty: long-term follow-up and review of the literature. *J Bone Joint Surg (Am)* 1998;80(5):733-748
11. Astrom M, Rausing A: Chronic Achilles tendinopathy: a survey of surgical and histopathologic findings. *Clin Orthop* 1995;316(Jul):151-164
12. Khan KM, Bonar F, Desmond PM, et al: Patellar tendinosis (jumper's knee): findings at histopathologic examination, US and MR imaging: Victorian Institute of Sport Tendon Study Group. *Radiology* 1996;200(3):821-827
13. Kraushaar BS, Nirschl RP: Tendinosis of the elbow (tennis elbow): clinical features and findings of histological, immunohistochemical, and electron microscopy studies. *J Bone Joint Surg (Am)* 1999;81(2):269-278
14. Ollivierre CO, Nirschl RP, Pettrone FA: Resection and repair for medial tennis elbow: a prospective analysis. *Am J Sports Med* 1995;23(2):214-221
15. Uthoff HK, Sano H: Pathology of failure of the rotator cuff tendon. *Orthop Clin North Am* 1997;28(1):31-41
16. Järvinen M, Jozsa L, Kannus P, et al: Histopathological findings in chronic tendon disorders. *Scand J Med Sci Sports* 1997;7(2):86-95
17. Khan KM, Cook JL, Bonar F, et al: Histopathology of common tendinopathies: update and implications for clinical management. *Sports Med* 1999;27(6):393-408
18. Puddu G, Ippolito E, Postacchini F: A classification of Achilles tendon disease. *Am J Sports Med* 1976;4(4):145-150
19. Perugia L, Postacchini F, Ippolito E: *The Tendons: Biology, Pathology, Clinical Aspects*. Milan, Italy, Editrice Kurtis, 1986
20. Astrom M, Gentz CF, Nilsson P, et al: Imaging in chronic Achilles tendinopathy: a comparison of ultrasonography, magnetic resonance imaging and surgical findings in 27 histologically verified cases. *Skeletal Radiol* 1996;25(7):615-620
21. Potter HG, Hannafin JA, Morwessel RM, et al: Lateral epicondylitis: correlation of MR imaging, surgical, and histopathologic findings. *Radiology* 1995;196(1):43-46
22. Clement DB, Taunton JE, Smart GW: Achilles tendinitis and peritendinitis: etiology and treatment. *Am J Sports Med* 1984;12(3):179-184
23. Brukner P, Khan K: *Clinical Sports Medicine*. Sydney, Australia, McGraw-Hill, 1993

24. Enwemeka CS: The effects of therapeutic ultrasound on tendon healing: a biomechanical study. *Am J Phys Med Rehabil* 1989;68(6):283-287
25. Gum SL, Reddy GK, Stehno-Bittel L, et al: Combined ultrasound, electrical stimulation and laser promote collagen synthesis with moderate changes in tendon biomechanics. *Am J Phys Med Rehabil* 1997;76(4):288-296
26. Almekinders LC, Temple JD: Etiology, diagnosis, and treatment of tendonitis: an analysis of the literature. *Med Sci Sports Exerc* 1998;30(8):1183-1190
27. Shrier I, Matheson GO, Kohl HW III: Achilles tendonitis: are corticosteroid injections useful or harmful? *Clin J Sport Med* 1996;6(4):245-250
28. Astrom M: Partial rupture in chronic Achilles tendinopathy: a retrospective analysis of 342 cases. *Acta Orthop Scand* 1998;69(4):404-407
29. Niesen-Vertommen SL, Taunton JE, Clement DB, et al: The effect of eccentric versus concentric exercise in the management of Achilles tendonitis. *Clin J Sport Med* 1992;2(2):109-113
30. Curwin SL, Stanish WD: *Tendinitis: Its Etiology and Treatment*. Toronto, Collamore Press, 1984
31. Hölmich P, Uhrskou P, Ulnits L, et al: Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomised trial. *Lancet* 1999;353(9151):439-443
32. Alfredson H, Pietila T, Jonsson P, et al: Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med* 1998;26(3):360-366
33. Leadbetter WB: Cell-matrix response in tendon injury. *Clin Sports Med* 1992;11(3):533-578
34. Vilarta R, Vidal B de C: Anisotropic and biomechanical properties of tendons modified by exercise and denervation: aggregation and macromolecular order in collagen bundles. *Matrix* 1989;9(1):55-61
35. Woo SL, Gomez MA, Woo YK, et al: Mechanical properties of tendons and ligaments, II: the relationships of immobilization and exercise on tissue remodelling. *Biorheology* 1982;19(3):397-408

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