Evaluation and Management of Tendinopathy

Last month, *AAOS Now* editorial board member Letha Y. Griffin, MD, conducted a focused round-table on the use of platelet-rich plasma in the treatment of tendinopathy (chronic inflammation of the tendon). That discussion continues this month with a broader look at treatments, ranging from physical modalities to oral and topical anti-inflammatory medications, high- or low-intensity pulsed ultrasound, extracorporeal shock wave therapy (ESWT), and injected platelet-rich plasma (PRP).

Joining Dr. Griffin are James R. Andrews, MD; Brian J. Cole, MD, MBA; Nicola Maffulli, MD, PhD; Bert Mandelbaum, MD; Scott A. Rodeo, MD; and Holly J. Silvers, MPT.

						
Letha Y. Griffin, MD	*James R. Andrews, MD*	*Brian J. Cole, MD, MBA*	*Nicola Maffulli, MD, PhD*	*Bert Mandelbaum, MD*	*Scott A. Rodeo, MD*	*Holly J. Silvers, MPT*

Dr. Griffin: How would you define tendinopathy? What distinguishing characteristics are needed to make the diagnosis of tendinopathy vs. tendinitis?

Dr. Rodeo: The clinical features of tendinopathy include chronic activity-related pain of the involved tendon. Depending on severity, the pain will occur some time after onset of activity of the involved extremity. In more severe cases, the pain begins immediately with initiation of activity and may persist after the activity stops.

Symptoms typically must be long-standing for me to make the diagnosis of tendinopathy. The physical exam will find tenderness at the involved tendon, pain with resistive testing of the involved muscle-tendon unit, sometimes pain with passive stretch, and occasionally palpable thickening or nodularity of the involved tendon.

I would make the diagnosis of acute tendinitis if a new and sudden onset of tendon-related pain occurs after excessive activity. My sense is that acute pain in this setting is due to an inflammatory response in the paratendon (peritendinitis); however, we have essentially no confirmatory biopsy data to prove this.

An unanswered question is the mechanism by which this acute injury response leads to chronic tendinosis, with the associated structural changes in the tendon (Fig. 1). At the histologic level, tendinosis refers to the structural and compositional changes in tendon due to cumulative overload. It is characterized by increased cellularity, increased vascularity, changes in cell morphology (cells

become more rounded in contrast to normal elongated fibroblasts), myxoid changes in the matrix with increased glycosaminoglycan deposition, and occasionally dystrophic changes such as calcification and lipid deposition.

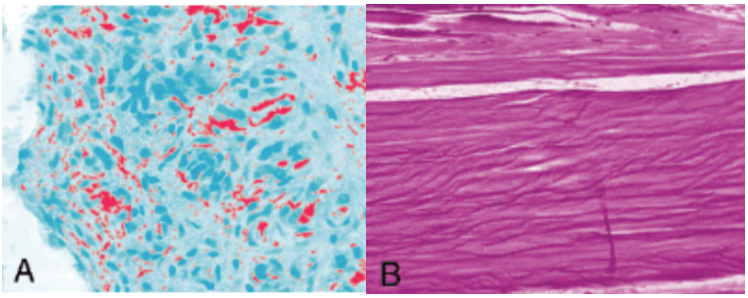

Fig. 1 A, *The histologic appearance of angiofibroblastic tendinosis and* ***B,*** *normal tendon. (Reprinted from Nirschl RP, Ashman ES: Tennis elbow tendinosis (epicondylitis) in Helfet DL, Greene WB, eds: Instructional Course Lectures 53, Rosemont, Ill. American Academy of Orthopaedic Surgeons, 2004, pp 587-589)*

Dr. Andrews: Tendinitis, tendinosis, and tendinopathy are often used interchangeably by patients and physicians alike, but they do indeed have different pathologies. Tendinitis is an inflammatory acute process, whereas tendinosis or tendinopathy is more of a chronic injury to the tendon.

I believe some of the confusion arises from the fact that both conditions can exist simultaneously in the same patient and persistent tendinitis can cause chronic changes in the tendon itself. Also, the initial treatment is often the same for both.

In addition to the chronicity of the symptoms, I am more likely to diagnose an athlete with tendinosis if he or she has enlargement of the tendon, waxing and waning of symptoms, and pain that occurs more with tendon use than with palpation.

Dr. Griffin: Dr. Maffulli, you have been actively involved in defining the pathophysiology of tendinopathy. How close are we to understanding how an acute inflammatory condition transitions to a chronic degenerative state?

Dr. Maffulli: A major issue is that athletes with overuse injuries may have acute tendon pain, but from a biologic viewpoint, the condition is already a chronic failure of healing of the tendon. Although scientific evidence is lacking, it appears that the tendon sustains a lesion that, for some yet unclear reason, cannot be repaired. This may be due to a variety of conditions, of which overuse could be one (but underuse should not be discounted).

There is no evidence that tendinitis, in the context of overuse athletic injuries, exists, and there is good evidence that tendinitis does not progress to tendinosis, which is at best a misnomer, because the affected tendon is not really degenerated; it exhibits features of failed healing response. Hence, the use of the term tendinopathy.

Dr. Cole: Clinically, it is important to differentiate acute from chronic tendon pain because treatment and prognosis may differ greatly. An athlete with acute onset of tendon pain, once recognized, has

possibly the best chance of achieving expedited resolution of his or her symptoms. At the onset of acute symptoms, relative rest, local treatment such as ice, topical nonsteroidal anti-inflammatory drugs (NSAIDs), load protection, maximizing flexibility, and even systemic NSAIDs may be effective.

Depending on the tendon involved, the acute onset of symptoms may be associated with tenosynovitis. Alternatively, acute microscopic tendon tearing may be the initial pathologic entity. Finally, the clinical presentation may be the first manifestation of a pre-existing chronic asymptomatic tendinosis.

Clearly, some patients with an acute presentation will be recalcitrant to treatment, often when relative rest, for example, is not a practical option. Once the condition becomes chronic, we may see more prominent findings on the physical examination and magnetic resonance imaging (MRI) compared to the acute scenario.

I have performed serial MRIs on jumping athletes and can recall several examples in which the initial findings supported the conclusion of a chronically “diseased” tendon at symptom onset. The progression of findings over time, however, appears commensurate with the patient’s symptoms if the initial treatment is unsuccessful.

We don’t have enough information to tell us how aggressive to be initially, and typically, we don’t employ more aggressive options (such as ESWT or PRP) until we have been initially unsuccessful at resolving the patient’s symptoms. Perhaps we should be more aggressive earlier, especially if we can identify risk factors for patients for whom standard first-line treatment options will fail to resolve the condition quickly.

Dr. Mandelbaum: The theoretical explanation for tendon injuries suggests a continuum of events—including hypovascularity and repetitive microtrauma—that results in localized tendon degeneration and weakness. Ultimately, the tendon may rupture if it is continually subjected to loads that exceed its physiologic capacity.

Defining the type of tendinopathy present is important, because each may have different clinical manifestations and require different treatments. For example, in Achilles tendinosis, a mobile nodule may be present. Histopathologic changes such as mucoid degeneration, lipomatous infiltration, and calcifying tendinopathy have been noted at the time of surgical repair.

Patellar tendinosis, on the other hand, most commonly involves the proximal bone-tendon junction, and the intrasubstance changes within the tendon histologically represent areas of pseudocystic cavities filled with necrotic tissue.

Dr. Griffin: Given our present knowledge, what treatment initiatives are reasonable (effective and clinically and financially efficient) in symptomatic athletes to permit them to return to sport safely? For example, is physical therapy, including specific exercises or modalities such as ultrasound or electrical stimulation, helpful?

Dr. Andrews: The treatment of tendinosis initially should be multimodal. Physical therapy, anti-inflammatory medications, PRP, rest, ice, and even a short period of immobilization can be used together to help relieve symptoms. The poor vascularity of tendons makes this a difficult and sometimes lengthy problem to treat.

In my practice, we routinely use physical therapy modalities, such as ultrasound and electrical stimulation. We trust our well-trained therapists with implementing the program.

Dr. Mandelbaum: Early on, simple steps of relative rest, splinting with controlled mobilization, stretching, icing, topical anti-inflammatories, therapeutic exercises, neuromuscular training, and modalities such as ultrasound, electrical stimulation, or ESWT can be beneficial in managing symptoms. I have been impressed how successful ice baths and ice massage can be in many situations.

The degree of success or failure in symptom alleviation will drive the next steps in the algorithm. Determinants of next step treatment include timing of the problem with respect to the timing in the season and the severity of symptoms.

Dr. Griffin: Ms. Silvers, as a physical therapist, what do you recommend as a treatment protocol for chronic tendinopathy?

Ms. Silvers: Limited research supports the use of modalities such as ultrasound, low-level laser therapy, ESWT, nitric oxide patches, and electrical stimulation to treat tendon pathology. Rest, NSAIDs, stretching, and cryotherapy can have some effect on acute effusion and pain, but these treatments seldom exhibit long-standing benefits.

We need to understand why the pathology developed in the tendon in the first place and address that core issue. If it is an underuse issue, which is certainly less frequently identified in the active population, but may be seen in the postoperative population, the underlying pathokinematics leading to this scenario need to be addressed, as does any muscle weakness or imbalance that may exist between the agonist and antagonist muscle groups. Finally, we need to check the proximal joint for any concomitant pathology, such as patellar tendinopathy related to an ipsilateral deficiency in the proximal iliopsoas and/or lateral and posterior hip. An ergonomic assessment is recommended, particularly for patients with patellofemoral tendon symptoms.

We effectively use massage on the involved tendon, acupressure, exercise techniques using a foam roller, joint mobilization, taping, orthotics and/or bracing to address faulty joint positioning or soft-tissue edema, core conditioning (such as Pilates), neuromuscular balance training, and muscle strengthening (eccentrically and concentrically). A weight-bearing reduction treadmill or aquatic therapy may be helpful in chronic cases in which the patient cannot tolerate full weight-bearing.

When the patient resumes sports-specific activities, integrating cross-training and activity diversification is crucial to prevent tissue overload. By addressing the underlying faulty biomechanical movement patterns, an effective treatment plan may provide extensive relief and long-standing therapeutic results.

Dr. Griffin: Are there other considerations in treatment protocols?

Dr. Maffulli: Ultrasound and electrical stimulation may alleviate symptoms in some athletes for awhile. Although these modalities are widely used, scientific studies on their effects have not found very impressive results.

Dr. Griffin: Is there a place for immobilization in treatment protocols?

Dr. Rodeo: We need more information about the effect of mechanical load on basic tendon biology. Specifically, we need further information about the type of load, timing of load onset, and the role of load magnitude, frequency, and duration on the healing process.

That said, I do believe a brief period of immobilization in the acutely injured tendon is appropriate to allow resolution of acute inflammation. However, my own opinion is that this should be brief, with early resumption of controlled loading of the tendon. I believe that the type of loading applied to the tendon (eccentric versus concentric) and the magnitude and frequency of load are more important than the simple question of when we begin to load the injured tendon.

Dr. Griffin: How frequently do you use oral, injectable, or topical anti-inflammatory drugs to treat tendinopathy? What do you perceive as the benefit of these drugs?

Dr. Mandelbaum: My use of oral, topical, and injectable anti-inflammatory drugs depends on the location, the severity of symptoms, and timing. Most tendinoses are not inflammatory problems. Oral NSAIDs, for example, appear to alleviate symptoms in the short term.

We have also used topical Voltaren® (diclofenac) and compounded combinations of ketoprofen, piroxicam, and 1 percent lidocaine applied transcutaneously three or four times a day. I never use oral steroids in the athlete, because of both the potential of tendon avulsion and the issues of doping control. In selected circumstances of lateral or medial epicondylitis and plantar fasciitis, I have used steroid injections. In other tendinopathies it is contraindicated.

One last point, it is always important to understand the overall health status of the patient and athlete. Quinolones, thyroid disorders, and inflammatory arthritis can influence symptoms.

Dr. Cole: I frequently use oral and topical NSAIDs. I only use injectable steroids for lateral epicondylitis or rotator cuff pain (with or without a tear). I have been reluctant to treat conditions such as patellar tendinitis, quadriceps tendinitis, or Achilles' tendinitis with anything more than NSAIDs (oral or topical) and have no experience in injecting these areas with corticosteroids.

Some patients who get minimal benefit from oral NSAIDs will show an additional clinical benefit as soon as they switch to a topical NSAID. Because most of these conditions are diseased tendons, and inflammation is not the hallmark of the associated pathophysiology, it is unclear why corticosteroid injections actually are successful. Perhaps the injection stimulates a healing response through creating inflammation rather than treating inflammation as in lateral epicondylitis.

I have been rather surprised at the success of topical agents used to treat tendinopathy. I do believe that, in addition to these agents, appropriate stretching modalities, relative rest, and load sharing devices when applicable (ie, patella compression or Chopart strap) will effectively round out the treatment program.

Dr. Griffin: Pulsed ultrasound (low and high frequency) has been used as a treatment modality, but is it helpful?

Dr. Maffulli: Pulsed ultrasound seemed to work in experimental models, but randomized controlled trials have shown it has no effect.

Dr. Griffin: Thank you all for an interesting discussion of a very complex, albeit very common, problem. Readers who are often frustrated in treating these injuries should refer to the online version of this article for additional treatment pearls and sage advice from our participants.

Disclosure information: Dr. Griffin—Piedmont Hospital Board of Directors; AOSSM; AAOS Now; Dr. Andrews—Biomet Sports Medicine; Biomet; Bauerfiend; MiMedx; Theralase; Physiotherapy

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