Tennis Elbow: Blending Basic Science with Clinical Practice

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Abstract
Tennis elbow defines a condition of varying degrees of pain or point tenderness on or near the lateral epicondyle. It is prevalent in individuals that perform a combination of forceful and repetitive activities including athletes and wheelchair users. It is the most common work-related disorder at the elbow. Histopathological findings indicate that tennis elbow is a degenerative condition, called tendinosis, of the common extensor tendon, with the ECRB tendon more commonly implicated as the primary location of tendinosis. Despite the absence of inflammation, patients with tennis elbow still present with pain. Neurochemicals including glutamate, substance P, and calcitonin gene-related peptide have been identified in patients with chronic tennis elbow and in animal models of tendinopathy. Their presence provides an alternative mechanism for pain mediation. Based on what is known about tissue changes within chronic tendinopathies, implications for therapy including examination and interventions are discussed.
Introduction

Various names including tendinitis, tendinosis, paratenonitis, and peritendinitis have been used to represent the clinical condition known as tennis elbow, depending on the status of the tendon tissue at different stages of healing. This terminology is described in Table 1. The common extensor tendon inserts on to the lateral epicondyle which explains the use of terms such as lateral epicondylitis, lateral epicondyllosis, lateral epicondylalgia to describe what the lay person calls “tennis elbow”. The use of the suffix “itis” may be misleading since it assumes that there is an inflammatory state within the injured tendon. The suffixes “osis” and “algia” represent a degenerative condition or pain respectively. Waugh provides a strong argument for the use of the term lateral epicondylalgia since it may encompass all potential causes of lateral elbow pain without making an assumption about the underlying histopathology.

Although the term tennis elbow does not reveal the state of the tendon tissue, it is also not an appropriate term because most patients do not get the condition from playing tennis. This author prefers to use the term lateral elbow tendinopathy, but the reality of this academic discussion is that our patients, most clinicians, and the general public refer to the condition as tennis elbow. As a search phrase for electronic literature databases, tennis elbow is the “cash cow” revealing significantly more citations than any of the other preferred or appropriate terms. For this reason, the phrase tennis elbow will be used consistently in this article.

Clinical Presentation

Tennis elbow defines a condition of varying degrees of pain or point tenderness at the origin of the wrist extensor muscles near the lateral epicondyle of the humerus. Grip strength of the involved hand and the ability of the extremity to tolerate load especially with the elbow extended are limited. Any of the wrist or digit extensor muscles that share the common extensor
tendon may be involved, but the extensor carpi radialis brevis (ECRB) is more frequently implicated.5-9 This condition is more likely to occur in persons over the age of 35 years, particularly if they have a high activity level that includes forceful repetitive upper extremity motion.8-10 However, a single event, such as lifting a heavy object or performing an awkward grasping movement can develop into an elbow tendinopathy. The dominant upper extremity is most commonly affected.8

The pain complaints associated with tennis elbow are usually centered near the lateral epicondyle, but may radiate proximally or distally depending on the severity of the condition.6, 8, 9 Palpation may reveal point tenderness directly on the lateral epicondyle or up to 5 mm anterior and distal to it. Point tenderness along the lateral supracondylar ridge may implicate involvement of the extensor carpi radialis longus (ECRL).9 Patients frequently report an increase in aching in the evening and elbow stiffness in the morning.6 Functional use of the involved upper extremity, especially gripping activities usually exacerbates pain symptoms. Resisted wrist extension, radial deviation, finger extension, and forearm supination can elicit pain.6, 9, 11, 12 All or some of these movements may be painful depending on the irritability of the tissues. Resisted range of motion testing may be more painful with the elbow extended.9, 12 Active extension of the wrist is usually limited secondary to pain.6

Therapists should perform a thorough physical examination of a patient referred with lateral elbow pain. Since tennis elbow is such as common condition, many non-specialized clinicians are quick to diagnose non-traumatic elbow pain as “tennis elbow” despite the many potential sources of lateral elbow pain. Differential diagnosis includes cervical radiculopathy, proximal neurovascular entrapment, and radial tunnel syndrome. It is often difficult to differentiate between tennis elbow and radial tunnel syndrome in an acutely painful elbow.
Clinical examination techniques used to provoke symptoms of each condition usually stress the same tissues. A key clinical feature for differential assessment is the location of point tenderness. It is expected that if a patient has pain due to inflammation or degeneration of the common extensor tendon, the point tenderness would be located on or near the lateral epicondyle.\textsuperscript{3, 8, 9} Point tenderness is most commonly found at the leading edge of the supinator muscle with radial tunnel syndrome, approximately 3 cm distal and posterior to the lateral epicondyle.\textsuperscript{13} Additional information on the physical examination of tennis elbow including differential diagnosis is available elsewhere.\textsuperscript{5, 10, 14}

**Epidemiology**

Upper extremity tendinopathies are associated with repetitive movement, including active muscle contractions and stretching over bony surfaces, with and without force. The incidence of upper extremity tendinopathies increases with age and the amount of exposure to forceful repetitive movement.\textsuperscript{15} Tennis elbow is considered the most prevalent work-related musculoskeletal disorder (WMSD) of the elbow and sufficient evidence exists for a strong association between its prevalence and a combination of physical risk factors including force, repetition, and posture.\textsuperscript{16, 17} Job classifications with high force demands and manually intensive work have a high prevalence of tennis elbow including construction workers, mechanics, butchers, and others.\textsuperscript{16, 18-20} The prevalence of tennis elbow ranges from 6-15\% in specific jobs identified in the meat and fish processing industries.\textsuperscript{18, 21, 22} Unskilled or untrained workers appear more like to develop tennis elbow.\textsuperscript{18} Physical workplace demands such as force, repetition, and awkward upper extremity postures are not only risk factors for developing tennis elbow, but are also indicators of poor prognosis for medical intervention.\textsuperscript{23, 24} High costs have been associated with tennis elbow in terms of health care costs, indemnity costs, and workdays
lost. Analysis of work-related claims in the federal workforce identified therapy as the primary expenditure for tennis elbow and the number of lost work-days was 79 days.\textsuperscript{25}

Forceful repetitive activity does not need to be work-related to cause tennis elbow. For example, wheelchair users are also at risk for developing tennis elbow, although shoulder tendinopathies and carpal tunnel syndrome are more prevalent.\textsuperscript{26, 27} Functional activities such as pressure reliefs, transfers, and wheelchair propulsion are the commonly reported aggravating activities associated with elbow pain.\textsuperscript{26} Tennis elbow has also been reported in recreational rock climbers\textsuperscript{28} and tennis players.\textsuperscript{29} Factors that contributed to either the occurrence or recurrence of tennis elbow in tennis players were the player’s age, level of ability, and amount of playing time.\textsuperscript{29} (The article by Badia and Stennett in this issue further discusses tennis elbow in the athlete.) It is generally theorized that these tendon injuries occur because there is insufficient opportunity for musculotendinous tissue to adapt to new tension loads placed upon it due to inappropriate training methods, improper technique or equipment, poor flexibility, and lack of recovery.\textsuperscript{30-32}

**Tendon Structure and Function**

Tendons serve as the interface between bone and muscle to transmit muscle force to the bone to create joint movement. The composition of tendon is primarily collagen, ground substance, and tenocytes. An aggregate of collagen fibrils form a collagen fiber which is the basic unit of a tendon. A network of thin reticular connective tissue known as the *endotenon* binds collagen fibers together to form the primary (subfascicle), secondary (fascicle), and tertiary bundles that compose the tendon. (Figure 1) In addition to binding collagen fibrils together, the endotenon surrounds each of the collagen bundles. Tendons that are not enclosed in a tendon sheath are surrounded by two connective tissues layers called the *epitenon* and the *paratenon*. 

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Together these two layers are known as the peritendon. The paratenon, a layer of loose areolar connective tissue, is the outermost layer and serves as an elastic sleeve to allow gliding of the tendon within the surrounding tissues. It is composed of type I and type III collagen fibrils, elastic fibrils, and synovial cells that line the inner surface of the paratenon that interfaces with the endotenon. The epitenon is sandwiched between the paratenon and the tendon and consists of a dense network of collagen fibrils. The orientation of these fibrils is varied including longitudinal, oblique, and transverse to withstand loads applied from various directions. 1,3

Tendons receive innervation, primarily sensory, from surrounding nerve fibers in the muscle or skin. The peritendinous tissues (paratenon and epitenon) are richly innervated with free nerve endings that function as pain receptors. Other nerve fibers penetrate through the connective tissue sheaths to the surface of the tendon and terminate on sensory nerve endings. The sensory end organs are thought to play a role in coordination, motor control and pain mediation.1 Neurokinin 1 – receptors (NK1-r), a primary receptor for substance P has been observed in the proximal ECRB tendon.33 Substance P, a neuropeptide, is a recognized pain modulator.34

The vascularity of tendons arises from three distinct locations including the myotendinous junction, osseotendinous junction, and the paratenon. Tendons enclosed in a sheath have a more distinct vascular supply that arises from the vincula and mesotenon. In general, the vascularity of a mature tendon is poor and even absent in some regions of the tendon. This may contribute to the poor healing potential of some tendon injuries.1, 35 Neovascularization is present with tendon grafts and following acute tendon injuries.35 Increased angiogenesis or hypervascularization is frequently associated with chronic tendinopathies.36-38 Although the increased capillary infiltration at the level of the chronic tendon
lesion is not associated with tissue repair, it is not clear what role vascularity may play in the degenerative process of tendinosis. Abnormal vascularity may contribute to pain mediation in chronic tendinopathies.  

**Histopathology of Tendinopathies: Tendinosis**

The etiology associated with degenerative tendon changes is not well understood, but is described extensively in the literature for the Achilles tendon, patellar tendon, and the ECRB tendon. The term tendinosis has been used to describe the histopathologic findings identified in an overuse injury to a tendon. The findings include: absence of inflammatory infiltrates; tenocyte hyperplasia and morphology; endothelial cell hyperplasia; microvascular thrombosis; hyaline, fatty, mucoid, calcified, fibrous infiltrates within the tendon substance; and cell necrosis.

Kraushaar and Nirschl described tendinosis as a tennis elbow condition characterized by degenerative changes of the common extensor tendon tissue. Tissue studies conducted via immunohistochemical analysis have revealed degenerative changes involving fibroblasts, blood vessels, and collagen. Tendinosis is confirmed with the presence of angiofibroblastic hyperplasia and the absence of cell types involved in inflammation. Nirschl described four stages of tendinosis that may assist the therapist in determining what type of intervention to provide the patient. Stage 1 is described as a peritendinous inflammation. This stage is actually what most clinicians refer to as tendinitis. Crepitus is usually palpable over the common extensor tendon. Stages 2, 3 and 4 refer to the presence of angiofibroblastic degeneration, with stage 4 being the most severe. Due to fibrosis, stage 3 may lead to tendon rupture and stage 4 to calcification.
Despite the absence of inflammation, patients with tennis elbow still present with pain, particularly with abusive activity. The reason for distinct pain in patients with tendinosis is not well understood. Also, tendinosis has been observed via tissue analysis following excision of the involved tendon. Staging patients’ tendinosis via clinical examination remains a challenge.

**Tendon Changes: Animal Models**

Most observations that have been published on tendinosis involve tendon tissue that has been excised from patients with chronic tendinopathies. This raises the question as to whether degenerative changes are a chronic phenomenon or if they occur in the acute stage of tendon injury as well. Several studies using animal models have been conducted to reproduce tendon injury, using either volitional or passive exercise loading methods.  

Backman reported that the Achilles tendon of rabbit hind limbs exposed to repetitive controlled kicking induced by electrically elicited contractions had irregular thickening and palpable nodules within 1 cm of the tendon insertion into the calcaneus after 4 weeks. While the tendons of the control limb demonstrated a normal histological appearance, after 5 or 6 weeks of controlled kicking the tendons of the experimental limbs displayed varied degenerative changes primarily within the central portion of the tendon. The most significant histopathological changes occurred in the paratenon with evidence of fibrosis and inflammation.  

Archambault et al. using the same animal model with a similar training paradigm as the Backman model with lower repetition rates demonstrated less severe histopathological changes in the Achilles tendon. No degenerative changes were exhibited in the tendon substance, but signs of inflammation and fibrosis within the paratenon were evident after 6 weeks of training. Substance P was not investigated in these models, but cytokines, IL-1β and TNFα that initiate the inflammatory response were expressed in the experimental tendons.
The use of the controlled kicking model provides some insight into the cellular response and tissue changes in tendon that occur with repetitive movement, but there are some limitations. First, the studies trained the experimental limb without volitional muscle activity. Electrically elicited contractions use a different physiological mechanism to create a muscle contraction than a volitional contraction. Second, the repetition rates exceed the rates of occupational tasks in humans making comparison to human tendinopathy conditions difficult. Finally, there was no bioquantification of the tissue changes.

Animal models that employ volitional muscle activity to examine the histopathological changes associated with forceful repetitive movement may help clinicians understand the pathogenesis of tendinopathies such as tennis elbow. A downhill treadmill training rat model has been developed to examine the effects of injury and overuse on the rotator cuff tendon. Acute tendon injury, external compression mimicking subacromial impingement, and overuse have all produced tendinosis separately. A study that combined, extrinsic injury or subacromial impingement with overuse injury created by downhill treadmill training produced greater evidence of tendinosis than either of the factors in isolation. The conclusion that the development of tendinosis is multifactorial is consistent with systematic reviews of the risk factors associated with work-related musculoskeletal disorders.

Barbe and Barr have developed a rat training model using volitional movement at occupational rates of repetition common in human workers. Similar to the Backman model, the tendons of rats that were exposed to a high repetition-high force task for 6 and 12 weeks demonstrated histopathological changes within the paratenon and epitenon with evidence of tissue hypertrophy, fibrosis, and cellular infiltration.

**Neurochemical Response**
Despite the absence of inflammation, patients with tennis elbow still present with pain, particularly with abusive or aggravating activity. Two tissue studies have identified the presence of neurochemicals within the tendon of the ECRB.\textsuperscript{47,48} Significant levels of substance P and calcitonin gene-related peptide (CGRP) were reported within the ECRB tendon in patients with chronic tennis elbow with an average duration of symptoms of 22.7 months.\textsuperscript{47} Alfredson, Ljung, Thorsen, and Lorentzon investigated the use of a microdialysis technique also used on the Achilles tendon\textsuperscript{49} and patellar tendon\textsuperscript{50} to determine the local concentrations of glutamate, an excitatory neurotransmitter for pain, and prostaglandin E\textsubscript{2} (PE\textsubscript{2}) an inflammatory mediator in the ECRB tendon\textsuperscript{48} of patients with tennis elbow for at least 6 months. The results of the study yielded statistically significant differences in mean concentration levels of glutamate in the tennis elbow patients compared to the control subjects. No significant differences were noted in prostaglandin levels between groups.\textsuperscript{48} Glutamate via NMDAr1, a glutamate receptor, immunoreactivity has been observed within neural structures of excised Achilles tendons and patellar tendons in patients with respective chronic tendinopathies.\textsuperscript{51,52} The presence of significant levels of glutamate, substance P, and CGRP in tendinosis may provide an alternative mechanism for pain mediation in tennis elbow as well as other chronic tendinopathies.

We do not know if the neurochemical response is present in a tendinopathy with duration of symptoms of six months or less and if there are concurrent inflammation or degenerative changes within the tendon since the human subject studies were only performed on tendons of patients with chronic tendinopathies at the time of surgery. Animal models must be employed to determine if there is an early neurochemical response associated with tennis elbow and other tendinopathies. A chemically induced experimental model of tennis elbow in Sprague-Dawley rats was used to investigate the involvement of sensory and sympathetic nerve fibers in pain.
mediation of tennis elbow. Following irritation using Carrageenan and Freund adjuvans, chemicals used to induce inflammatory injury, samples of ECRB muscle perfusates taken 2, 6, and 24 hours following injection of these irritants indicate that substance P is abundant during an acute inflammatory response compared to similar tissue samples in the control group. Messner et al. examined immunoreactivity for substance P in the endotenon and paratenon tissues in the hindlimb triceps muscle following repetitive eccentric muscle contractions in a controlled kicking rat model. Neurofilament labeling was evident within the epitenon and paratenon of the trained tendons, but only sparsely apparent in the control tendons. Immunoreactivity for substance P was intensive in the experimental limbs of the trained animals and sparse in the contralateral limbs of the trained animals and control animal limbs. Bioquantification techniques to measure the immunoreactivity were not performed. Substance P immunoreactivity was determined using bioquantification techniques in a volitional rat model of repetitive forceful motion. Substance P increases in peritendon tissue in forelimb tendons that have been exposed to highly repetitive and forceful tasks. The response is also dependent on task exposure, with the greatest response at 12 weeks. The observations in these animal studies suggest that at least substance P is present in acute overuse tendon conditions such as tennis elbow.

**Implications for Treatment: Are There Any?**

Management for tennis elbow usually involves patient education about activity modification, referral to therapy and/or a trial of non-steroidal anti-inflammatory drugs or a corticosteroid injection. Systematic reviews of the literature related to conservative management of tennis elbow usually indicate that there is a lack of evidence to support a particular plan of care or interventions frequently used by therapists. The reviews identified problems in the available literature including the lack of randomized controlled clinical trials, poor quality
studies, and vague inclusion criteria. After reviewing the literature related to histopathological changes associated with tennis elbow and other tendinopathies, it is clear that the status of the tendon tissue needs to be considered before a plan of care is established. Of course, this will also require an adequate physical examination. The following discussion is likely to raise more questions than answers.

*Physical Examination*

First and foremost, how can therapists determine the histological status of the ECRB tendon in a patient with tennis elbow? The use of the clinical examination alone presents a challenge, but is unlikely that all patients with tennis elbow, if any, are likely to have a biopsy of their common extensor tendon. It seems reasonable that the patient history may provide the most useful data. The duration of symptoms and the number of recurrences may suggest either an acute injury or condition consistent with a peritendinous inflammation or early stage tendinosis. A more longstanding or chronic condition would increase the likelihood of advanced stage tendinosis. Generally, if the duration of symptoms is 3 months or less this is considered an acute condition\(^6\)\(^0\) and a chronic condition would be consistent with duration of symptoms greater than 3 months\(^6\)\(^1\). A history of previous occurrences of tennis elbow also suggests tendinosis.

Imaging techniques such as MRI or diagnostic ultrasound are useful to identify calcification, tears or ruptures of the ECRB.\(^6\)\(^2,\)\(^6\)\(^3\) Therapists would not likely be able to determine the presence of these histopathological changes without imaging studies, keeping in mind that an intact ECRL tendon would certainly mask a complete rupture of the ECRB tendon. Although imaging studies are described in the tennis elbow literature, they are typically not performed unless the patient fails conservative management and surgery is being considered.\(^6,\)\(^10,\)\(^11,\)\(^5\)\(^5,\)\(^5\)\(^6\) Imaging studies are the only non-invasive manner to provide some evidence of tissue changes.
Palpation may also be useful during the clinical examination. Special tests such as the tennis elbow test or Cozen’s test, Mill’s test, and variations in grip strength measures are commonly used during the physical examination.\textsuperscript{5, 7, 14} The validity, specifically sensitivity and specificity, of these tests has not been determined. Tissue observations noted by imaging may serve as the standard for determining the validity of the special tests; this certainly is an area wide open for research. Pain free grip strength\textsuperscript{64} and the Patient-rated tennis elbow\textsuperscript{61,65} are commonly used to study the effects of clinical intervention. Further investigation of these measures is needed to determine if there are differences in initial scores that may correlate with histopathological findings such as peritendinous inflammation, tendinosis, or ECRB tendon rupture.

**Interventions**

Since tennis elbow was perceived to be primarily an inflammatory condition, most interventions have been directed towards resolving inflammation. What is currently known about the histopathology of tennis elbow indicates that although tennis elbow may start out as an inflammatory condition it will progress to a degenerative state. Although both tissue conditions are painful, it is unclear which interventions may be helpful in pain modulation in chronic tennis elbow. We do not know if any of the commonly used physical agents may resolve the neurochemical response and its associated pain mediation in either the acute or chronic tennis elbow. In addition to pain modulation, physical agents such as ultrasound or electrotherapy may be used to facilitate tissue healing. Tendinosis, at least at initial onset, is similar to a stagnant state of fibroplasia. Theoretically, ultrasound and electrotherapy may be able to stimulate cellular responses to promote tissue healing. Detailed information on the physiological effects of ultrasound and electrotherapy may be found elsewhere.\textsuperscript{66}
Patient education regarding activity modification to reduce exposure to aggravating activity appears to play a crucial role in resolving the pain associated with tennis elbow.\textsuperscript{7, 67} There are a large variety of therapy interventions described for tennis elbow to address muscle strength and flexibility. However, there is a lack of evidence to support a particular exercise prescription to increase tolerance for loading the common extensor tendon, however there is general agreement that exercise is beneficial to treatment outcome.\textsuperscript{7, 14,68-70} This includes painful eccentric exercise which has been shown to be effective in the management of chronic mid-portion Achilles tendinopathy, but not insertional Achilles tendinopathy.\textsuperscript{71} Since tennis elbow is considered an insertional tendinopathy, this may be why studies using eccentric exercise have not been shown to be superior to other types of exercise programs such as stretching or concentric progressive strengthening programs. Further investigation is needed to identify the unique features of an exercise program that would be beneficial to improved physical tolerance and pain reduction in patients with tennis elbow.

**Closing Remarks**

Clinical research is needed to delineate treatment paradigms for improving patient outcomes based on the status of tendon tissue, either degenerative or inflammatory. The histopathological findings discussed present opportunities for therapists to evaluate and improve existing clinical examination skills. These findings may also generate new theories in regards to the management of pain either by revising currently used interventions or developing new techniques. New treatment guidelines need to be identified for exercise prescription to enhance the physical tolerance of the involved tendon tissue to carry out the physical activities performed by patients with tennis elbow.
REFERENCES


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Modified from Józsa and Kannus, 1997

**Figure 1 Tendon Structure with Connective Tissue.** The collagen fibril is the smallest unit of tendon that can be mechanically tested. An aggregate of collagen fibrils form a collagen fiber. Collagen fibers are bound together by the endotenon to form the primary, secondary, and tertiary bundles. The epitenon and paratenon are the outermost layers of connective tissue and together are known as the peritendon.
<table>
<thead>
<tr>
<th>Terminology</th>
<th>Definition</th>
<th>Histopathological Changes</th>
<th>Clinical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paratenonitis</td>
<td>Inflammation of the paratenon with or without a synovial sheath</td>
<td>Inflammatory cells found within paratenon or peritendinous areolar connective tissue</td>
<td>Cardinal signs of inflammation</td>
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<td>Crepitus with palpation</td>
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<td>Duration of symptoms &lt;12 weeks</td>
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<tr>
<td>Paratenonitis with</td>
<td>Paratenon inflammation with associated tendon degeneration</td>
<td>Paratenon same as above with evidence of collagen fiber disorganization, hypervascularity</td>
<td>Cardinal signs of inflammation</td>
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<tr>
<td>Tendinosis</td>
<td></td>
<td>within the tendon, but no intratendinous inflammation</td>
<td>Crepitus with palpation</td>
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<td>Palpable tendon nodule or edema</td>
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<td>Duration of symptoms 12 weeks or more</td>
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<tr>
<td>Tendinosis</td>
<td>Intratendinous degeneration associated with aging, repetitive movement,</td>
<td>Collagen fiber disorganization, hypervascularity, increased fibroblasts, evidence of necrosis</td>
<td>Palpable tendon nodule tender to palpation</td>
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<td></td>
<td>and vascular compromise</td>
<td>and/or calcification</td>
<td>No signs of inflammation</td>
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<td>Duration of symptoms 12 weeks or more</td>
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<td>Recurrent condition likely</td>
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<td>Imaging studies to confirm tears, calcification</td>
</tr>
<tr>
<td>Tendinitis</td>
<td>Symptomatic degeneration of the tendon with evidence of inflammation and vascular disruption</td>
<td>Various presentations including inflammation, tendinosis, evidence of tears, calcification, or complete or partial rupture</td>
<td>Cardinal signs of inflammation proportional to vascular disruption or hematoma Duration of symptoms 12 weeks or more Recurrent condition likely Imaging studies to confirm tears, ruptures, calcification</td>
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Tissue information modified from$^{1,3}$