The Practical Management of Achilles Tendinopathy

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The Achilles tendon, named after the legendary warrior and hero of Homer's *Iliad,* is the strongest and thickest tendon in the human body. Despite this fact, Achilles tendinopathy is a common overuse injury, particularly in runners and other athletes. Kujala et al[1] showed a 10-fold increase in Achilles injuries in runners compared with age-matched controls. Another study reported the incidence of Achilles tendinopathy in top-level runners as 7% to 9%.[2] The specific factors linking this injury with running include excessive mileage, sudden increase in training intensity, decrease in recovery time, change of running surface, and poor footwear.[3,4] This injury also is common in athletes who compete in racquet sports, track and field, volleyball, and soccer.[5] Other factors that have been associated with Achilles tendinopathy include various biomechanical deficits, older age, male gender, increased body weight and height, and fluoroquinolone exposure.[6,7]

TERMINOLOGY

The terminology commonly employed to describe Achilles tendon injury can be confusing and misleading. Although the term *tendinitis* is often used in clinical practice to depict tendon injury, inflammatory cells are seen infrequently in biopsy specimens of injured tendons except in association with tendon rupture.[8] No increase in the amount of biochemical mediators of inflammation, such as prostaglandin E_2, has been found in patients with Achilles pathology compared with controls.[9] In reality, there seem to be various histopathologic entities that can cause Achilles tendon pain. The most common of these pathologies is *tendinosis,* tendon degeneration without histologic or clinical signs of intratendinous inflammation. Many clinicians use the word *tendinitis* to describe a condition that is actually a *tendinosis*; this misnomer can lead to an underestimate of the chronicity of Achilles tendon injury. *Paratenonitis* is the state of acute edema and hyperemia of the paratenon, accompanied by the infiltration of inflammatory cells and the possible presence of a fibrinous exudate filling the tendon sheath. Clinically, one can appreciate this phenomenon by feeling crepitus along the Achilles tendon sheath. A *partial tear* of the Achilles tendon refers to a macroscopically evident discontinuity of the tendon, an uncommon acute lesion. Finally, Mufalli et al[10] proposed that the combination of pain, swelling, and impaired performance be labeled *tendinopathy*.

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OVERVIEW

In considering a musculoskeletal injury, five components affect either anatomy or function and contribute to the production or continuation of symptoms: (1) *tissue injury*—the anatomic distribution of pathology; (2) *clinical symptoms*—the complaints of acute pain, swelling, and dysfunction; (3) *functional biomechanical deficits*—the combination of muscle inflexibilities, weakness, and imbalances causing inefficient mechanics; (4) *suboptimal functional adaptations*—the inadequate adjustments made in an attempt to maintain performance and minimize symptoms; and (5) *tissue overload*—the anatomy subject to excess tensile or eccentric stress. These elements are connected in a negative feedback loop called *Kibler's vicious overload cycle*[11] that is operative in every musculoskeletal injury (Fig. 1).

Method of Presentation

The presentation of Achilles tendinopathy usually involves the gradual onset of pain and dysfunction secondary to a chronic overuse injury. As described earlier, changes in training, running surface, and footwear are frequent precipitants of Achilles tendinopathy in runners. Uncommonly, patients can present with the acute onset of symptoms due to partial or even complete tears of the Achilles tendon.

Tissue Injury

The Achilles tendon is the combined tendon of the gastrocnemius and soleus muscles. The tendon is surrounded by a paratenon, as opposed to a synovial sheath, which is continuous with the fascia of the gastrocnemius and soleus muscles and the periosteum of the calcaneus. Achilles tendinopathy

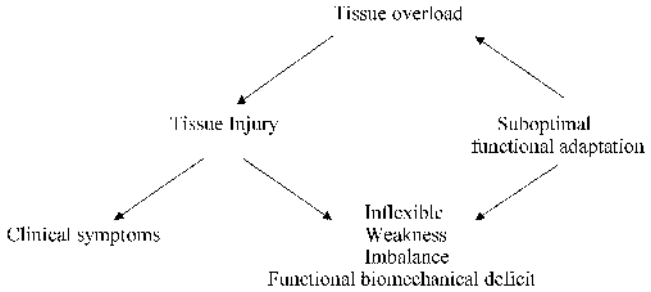

FIGURE 1. Kibler's vicious overload cycle

usually occurs 2–6 cm proximal to the calcaneal insertion at a site of decreased vascularization.[12]

Clinical Symptoms

The pain associated with Achilles tendinopathy ranges from mild to severe. Resisted plantar flexion and passive dorsiflexion at the ankle typically worsen the pain, making it difficult for a patient to stand on tiptoe or to traverse stairs. Initially, pain may be felt at the beginning and end of a training session; however, as the pathologic process progresses, pain may be felt throughout exercise. Eventually, pain may affect functional mobility adversely. On physical examination, tenderness may be pinpoint or more diffuse, even extending several centimeters along the tendon. Associated swelling, crepitus, and nodules also may be present.

Functional Biomechanical Deficits

Biomechanical deficits eventually lead to the development of Achilles tendinopathy by placing excess stress on the tendon. These inflexibilities, weaknesses, and imbalances can be seen throughout the kinetic chain. Clement et al[13] studied 109 runners and reported that in addition to overtraining, the most prevalent factors contributing to Achilles tendinopathy were functional overpronation (61 cases) and gastrocnemius/soleus insufficiency (41 cases). Clement et al[13] speculated that runners may be susceptible to Achilles tendinopathy due to microtrauma of the tendon caused by eccentric loading of fatigued muscle, excessive pronation necessitating whipping action of the Achilles tendon, and simultaneous pronation and knee extension that impart conflicting internal and external rotatory forces on the tibia. A study by Kaufman et al[14] showed that hindfoot varus deformity and decreased ankle dorsiflexion with the knee in extension was associated with Achilles tendinopathy. Finally, in a study of 455 athletes with Achilles tendon overuse injuries, Kvist[15] discovered biomechanical deficits in 60% of the athletes, including forefoot varus deformity and markedly limited passive subtalar joint mobility and ankle joint dorsiflexion with knee extension.

Other studies highlight the importance of coordinating proximal kinetic chain musculature, specifically the gluteus medius and maximus, to control ankle motion. In a study of individuals with previous severe unilateral ankle sprains, Bullock Saxton[16] found significant delays in the recruitment of the gluteus maximus during hip extension compared with controls. Beckman and Buchanan[17] reported altered firing patterns in the gluteus medius of subjects with hypermobile ankles. By affecting the control of ankle motion, proximal hip musculature and its associated biomechanical deficits can be assumed to play an important role in the development of Achilles tendinopathy.

Suboptimal Functional Adaptations

Over time, the above-outlined functional biomechanical deficits cause excessive repetitive overload of the Achilles tendon and worsen the tendinopathy. The patient makes inadequate adjustments in mechanics in an attempt to maintain function and minimize pain. Because the pain associated with Achilles tendinopathy typically is exacerbated by resisted plantar flexion and passive dorsiflexion at the ankle, these adaptations may include weakened gastrocnemius and soleus muscles, decreased ankle dorsiflexion, limited ankle and foot mobility, and increased flexion at the hip and knee.

Tissue Overload Complex

These suboptimal functional adaptations produce excess stress on the gastrocnemius-soleus complex and the common Achilles tendon, contributing to the vicious overload cycle.

IMAGING

If the diagnosis of Achilles tendinopathy is still in question after a careful history and physical examination, imaging may be warranted. Plain x-rays are not useful in the diagnosis of Achilles tendinopathy, although they may reveal associated or, more likely, incidental bony abnormalities. MRI and ultrasound are sensitive in detecting abnormalities associated with Achilles tendon pathology. Achilles tendinopathy is associated with areas of increased signal on MRI and regions of hypoechogenicity on ultrasound. These radiologic abnormalities of the Achilles tendon generally correspond to histopathologic findings of tendinosis, specifically mucoid degeneration characterized by increased interfibrillar glycosaminoglycans disrupting collagen fiber structure. A study by Astrom et al[18] comparing ultrasound and MRI with histologic biopsy and operative findings of injured Achilles tendons found that both of these radiologic modalities provide accurate and similar information. Ultrasound seems to have numerous advantages over MRI, however, including speed, relative inexpense, and safety for patients with contraindications to MRI. Ultrasound also has the advantage of interactive facility whereby transducer compression reproduces symptoms leading to improved focus on the abnormal area. The primary disadvantage of ultrasound is its operator dependence.

Because MRI and ultrasound are sensitive in showing pathology of the Achilles tendon, it is imperative to correlate any radiologic findings with the patient's symptoms. Several studies have reported abnormal findings in asymptomatic Achilles tendons.[19–21] These abnormalities may be precursors to symptomatic Achilles tendinopathy and have prognostic relevance. In a study of elite soccer players, Freberg and Bolvig[22] showed that ultrasound of asymptomatic Achilles tendons can be used to help predict which athletes will develop tendon symptoms. Of asymptomatic Achilles tendons, 11% (11 of 96) showed abnormal sonographic findings at baseline; during the season, 5 of these abnormal tendons became symptomatic compared with only 1 of the 85 sonographically normal tendons.

TREATMENT CONSIDERATIONS

As evidenced by the previously cited study, Achilles tendon damage likely exists before the onset of symptoms, so even patients who present acutely with Achilles tendinopathy may already have significant tendon degeneration. Treatment should begin with relative rest to allow time for the tendon to heal. The use of NSAIDs also should be considered initially; however, their role in treatment of Achilles tendinopathy is unclear because many believe antiinflammatory medications do not benefit the condition of tendinosis, which is classically noninflammatory. A study by Anstom and Westlin[23] found no beneficial effects of NSAIDs in the treatment of patients with Achilles tendinopathy. The role of corticosteroid injections in the treatment of Achilles tendon injuries is also uncertain. According to a comprehensive literature review, there are insufficient published data to determine the comparative risks and benefits of corticosteroid injections in Achilles tendinopathy. This study emphasizes the findings in animal studies of decreased Achilles tendon strength associated with intratendinous injections and suggests that tendon rupture may be a potential complication for several weeks after Achilles tendon injection.[24] Conversely, ultrasound seems to be helpful in the promotion of local healing. In animal studies, ultrasound has been shown to stimulate collagen synthesis in injured Achilles tendons and increase tendon tensile strength.[25,26] Other measures hypothesized to support healing include manual mobilization of the Achilles tendon and short-term use of a 12–15-mm heel lift, which helps reduce tensile loading. The heel lift should be eliminated, however, after calf flexibility improves because it can perpetuate calf inflexibility. Finally, cryotherapy has been shown to be effective in minimizing pain, decreasing the extravasation of blood and protein from capillaries, and lowering the metabolic rate in tendinopathy.

REHABILITATION

The rehabilitation of Achilles tendinopathy can be divided into three, often overlapping phases: (1) the acute phase, (2) the recovery phase, and (3) the functional phase. During the acute phase, emphasis should be placed on controlling pain and preventing progression of tendon degeneration. As described earlier, relative rest, pain medications, ultrasound, manual mobilization, heel lift, and cryotherapy are commonly used measures in this stage. When the pain is controlled and the patient is able to tolerate an eccentric strengthening program for the gastrocnemius-soleus complex, the second phase of rehabilitation can begin.

In the recovery phase, the goal of rehabilitation is to correct the biomechanical deficits identified in the physical examination. Specifically, manual mobilization is employed to improve foot and ankle range of motion; this includes subtalar and midtarsal mobilization and medial and lateral stretching of the Achilles tendon. Strengthening of the proximal muscles, particularly the gluteals, is also important in this stage. Additionally, multiplanar stretching and eccentric strengthening exercises for the gastrocnemius and soleus muscles are important components of rehabilitation in this stage. Several studies have shown that eccentric training of the gastrocnemius-soleus complex is superior to concentric training with regard to decreasing pain and improving function.[27–29] At the core of physical therapy for Achilles tendinopathy is the heel drop, an exercise performed with the knee flexed (to strengthen the soleus) and extended (to strengthen the gastrocnemius) (Fig. 2) with progression of weight through the injured leg. Fast eccentric strengthening exercises for the soleus also are important. As pain subsides and flexibility and strength improve, the final rehabilitation phase can begin.

During the functional phase, exercises should progress to being performed in multiple planes and with single-leg stance to promote normal motor patterns and control of the entire lower extremity. Jogging also should be introduced

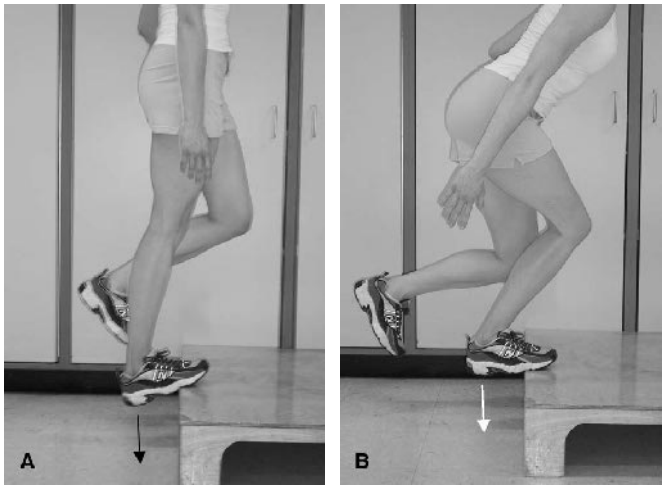

FIGURE 2. (A) Straight-leg heel drop: eccentric strengthening of the gastrocnemius. (B) Bent-knee heel drop: eccentric strengthening of the soleus.

gradually in this stage, increased only when there is no pain during or after exercise. Finally, footwear should be evaluated during the final rehabilitation phase because shoes with poor support or spikes with lowered heels can exacerbate the tendinopathy.

SURGERY

Conservative management has been shown to be effective in most cases of Achilles tendinopathy. Because this injury is usually secondary to chronic degenerative changes, however, a protracted period of time may be required before the clinical resolution of symptoms occurs. A trial of medical treatment and rehabilitation should continue for at least 6 months. If at this point such care has failed and the patient still is experiencing significant pain or decreased function, surgery may be considered (Table 1).

CONCLUSION

Achilles tendinopathy is a prevalent injury associated with significant morbidity in athletic and nonathletic populations. This injury is usually secondary to overuse; Achilles tendinopathy is characterized by the chronic degenerative changes of tendinosis rather than the acute inflammatory changes of tendinitis. This distinction has implications for the proper rehabilitation of this injury. Historically, conservative management has focused on minimizing acute inflammation; this approach is unlikely to resolve the issue of chronic degeneration. Tendinosis clinically manifests as inflexibility of the involved muscle-tendon group, weakness of the involved or surrounding muscle, and muscle strength imbalance between force generator and force regulator in the force couple.[30] The recognition of these biomechanical deficits focuses the goal of rehabilitation away from relief of the symptoms of the "itis" and toward the restoration of function that is lost with the "osis."[31]

TABLE 1. Achilles Tendinopathy Treatment Algorithm

Acute phase (pain control and promotion of local healing)
Relative test
Ice
Pain medication
Therapeutic ultrasound
Manual mobilization of the Achilles tendon
Heel lift
Recovery phase (correction of biomechanical deficits)
Mobilization of subtalar and midtarsal joints
Stretching of the gastrocnemius-soleus complex
Eccentric strengthening of the gastrocnemius-soleus complex
Strengthening of weak proximal musculature (ie, gluteal muscles)
Functional phase
Advance exercises to multiple planes and with single-leg stance
Evaluate footwear
Gradually introduce sport-specific training; increase only if no pain during or after exercise
Comprehensive home exercise program
Surgery
Consider surgery after 6 months if proper medical management and rehabilitation are unsuccessful

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