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Case Report

Update for the Evaluation and Treatment of Achilles Tendon Injuries

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Abstract

Achilles tendinopathy is a common medical complaint. Achilles tendon injuries include acute tendinitis from overuse in two distinct anatomic areas. Acute tendinitis may turn chronic and may require surgical intervention. Achilles tendon rupture, although usually a result of trauma, may occur spontaneously. Medication including fluoroquinolone and corticosteroids can play a significant role in these spontaneous ruptures. As a result of this discovery, care must be taken when prescribing these medications. Treatment for Achilles tendon rupture is either by surgical or non-surgical intervention. Outcomes are the same for either treatment modality.

Keywords:

Tendinitis, Tendinopathy, Achilles tendon rupture, Bone marrow aspirate concentrate (BMAC), Fluroquinolones, Corticosteroids

Content

A 37-year old father of three is first up to bat at the annual company picnic softball game. On the first pitch he connects with a line-shot just on the fair side of first base. He sees the ball skid past the first baseman heading into the right field corner. He rounds first and knows he will make second easy. Ten feet off second base, he hears a "pop" in right lower leg and he falls tumbling head-over-heels to the infield. As he rolls over, he wonders "how did the right fielder get to the ball so fast and throw it in just so it hit him in the back of his right lower leg?" He stands up and tries for second but the right leg won't support him. He falls again and now the pain comes. As he is lying in the sun, he sees the right fielder is just now getting to the ball. The batter is a physician assistant and knows instantly he will be going to surgery within the next three days to repair his torn right Achilles tendon. Thank goodness he knows a good orthopedic surgeon.

Introduction

The above is a classic description of the events surrounding an acute traumatic rupture of an Achilles tendon. A male who participates in strenuous activities only on an occasional basis, typically between ages of 30 and 50, who is involved in a vigorous running activity, which requires sudden acceleration, is the typical scenario for such an injury [1,2]. Although this history is typical for the traumatic tendon rupture, the much less common spontaneous non-traumatic Achilles tendon rupture has also been reported. In those instances, the history is very different. The patients tend to be older, have no history of trauma to the lower extremities whatsoever, but when specifically asked, the patient reveals a history of corticosteroids and/or fluroquinolones usage [3,4]. The patient also may be predisposed to acute non-traumatic Achilles tendon rupture if they have systemic diseases such as lupus erythematous, rheumatoid arthritis, or chronic renal failure [4]. Rare occurrences of bilateral non-traumatic ruptures of the patella tendons have even been noted in the literature [4].

Achilles tendon ruptures account for 20% of all large tendon ruptures with the annual incidence rate being approximately 2 out of 10,000 in the general population [2-4]. The incidence of

acute Achilles tendon rupture is steadily rising probably due to increasingly sedentary lifestyles with only occasional participation in "weekend" sports [5]. Males are five times more likely to rupture their Achilles tendon than females [6].

Anatomy

The Achilles tendon is the thickest and strongest tendon of the body and is the tendinous extension of the gastrocnemius, soleus and the plantaris muscles of the lower leg [3]. These muscles coalesce into the Achilles tendon approximately 15cm above its insertion site at the heel [2]. The tendon descends and inserts into the mid-portion of the superior-posterior surface of the calcaneus [3]. The tendon does not have a true synovial sheath but instead glides over the visceral and parietal layers of the posterior lower leg which allows approximately for 1.5cm of travel [7]. There are three sources of the tendon's blood supply: (1) musculo tendinous junction, (2) osseous insertion, (3) and multiple mesosternal vessels on the tendon's anterior surface [7]. Even with these three supply sites, the vascular supply of the tendon is the weakest in the area 2-6cm above the calcaneus. This area is the region where the tendon is most often seen to rupture [2].

Acute and Chronic Achilles Tendinosis

The Achilles tendon, like any tendon, can suffer from overuse and the Achilles tendon is a common injury site [8]. The pain from acute Achilles tendinosis typically occurs in one of two discreet areas; either at the insertion point of the tendon at the calcaneus, or 3-4cm proximal/superior to the insertion [8]. Insertional tendinosis is noted by pain, warmth, and swelling in the area, while non-insertional tendinosis is associated with thickening of the tendon at its midway point and severe tenderness to palpation in that area [8]. Because these conditions are more degenerative rather than inflammatory, the correct descriptive term is tendinosis rather than tendonitis [8]. Any persistent pain in the Achilles tendon, which lasts more than six weeks and is not due to rupture, is said to be a chronic tendinosis [9].

The treatments for acute and chronic tendinosis are different. Treatment for acute Achilles tendinosis includes stretching exercises, activity modifications, possible usage of a heel lift, and non-steroidal anti-inflammatory drugs (NSAIDs), if there are no contraindications to the medication usage [8]. There is little evidence to support the typical usage of NSAIDs, ice, and stretching in the treatment of chronic Achilles tendinosis [9]. The preferred treatment for insertional chronic Achilles pain is eccentric strengthening followed by four to six weeks of immobilization in a walking boot because cure rates are similar between immobilization/walking boot and shock-wave therapy [9]. The recommended first line treatment for mid-substance chronic Achilles tendinosis is an intense eccentric strengthening program of the gastrocnemius and soleus muscles [9]. There is also good evidence to show that treatment of Achilles tendinosis with steroid injections predisposes the patient to rupturing the tendon [9].

A surgical option is open for the patient who has failed six to nine months of conservative treatment [8]. Chronic insertional Achilles tendinosis pain is treated with surgical debridement of the degenerated tendon and removal of bone spurs and repair of the insertion site. If a significant superior calcaneal bone spur is note, a so-called Haglund deformity, it is removed at the same time. Mid-tendon, or non-insertional, chronic Achilles pain is treated with debridement of the degenerated tendon. If the majority of the tendon is nonviable, a tendon graft using the flexor hallucis longus tendon is used for the repair [8].

Acute Achilles Tendon Rupture

Achilles tendon rupture is the most common tendon rupture in the lower extremity [6]. With the type of injury described above, it is important to note that the patient may suffer a partial rupture of the tendon as opposed to a complete disruption. It has been noted that patients who experience an incomplete tear have more pain than those patient with complete disruption [6]. The reasoning for experiencing more pain is that partial function of the foot and ankle is maintained with an incomplete tear and therefore tendon loads across the injury site are partially intact and this loading causes the increased pain [6].

Most often patients who experience an acute Achilles rupture are unable to plantar flex their ankle, but some patients are able to walk on tiptoes and have some plantar flexion against resistance because other ankle plantar flexor mechanisms are intact [5,6]. Up to one third of all patients with complete tendon rupture do not complain of pain [5]. Because of these nuances, partial tear versus complete tear, no plantar flexion function versus some function on plantar flexion after injury, severe pain versus minimal pain, non-specialist fail to diagnose Achilles tendon rupture 20% of the time [5]. The problem with delaying treatment for an acute Achilles tendon rupture is that delay leads to poorer patient outcomes [5]. Once the tendon is torn, the free ends of the tendon begin to retract. This intern lengthens the distance between the ends, which must be pulled together for re-anastomosis. Meaning more complicated surgery, longer surgical scars, higher risk of complications and possible delay in return to normal activities including sports [5].

Physical Exam and Diagnosis

The time-honored test used to diagnosis an Achilles tendon rupture is the Thompson test; in fact, some authors believe this is the most reliable test if done within 48hrs of rupture [10]. The Thompson test is performed with the patient lying prone (face down), with both feet and ankles hanging over the end of the exam table [1]. The examiner squeezes the gastrocnemius-soleus complex on the injured side and looks for ankle plantar flexion. The examiner then compares the movement of the injured side to the uninjured ankle [6]. The patient with an Achilles rupture will typically show little to no movement of the ankle with the "calf squeeze".

Some authors have suggested using the Simmonds Triad of tests to better diagnose an Achilles tendon rupture [5]. The three test include (1) assessing for an altered angle of declination of the injured ankle as compared to the uninjured side, (2) assessment for any palpable gap in the mid-portion of the Achilles tendon, and (3) lack of plantar flexion on the calf squeeze-Thompson test [5]. Performing the Simmonds Triad as opposed to the Thompson Test alone, shows that the Simmonds Triad is 100% sensitive for rupture [5].

If the diagnosis remains unclear, an ultrasound or magnet resonate image of the area may be ordered [2]. The American College of Orthopedic Surgeons note that most often diagnostic test are not usually needed [10].

Treatment

An Achilles tendon rupture may be treated non-operatively or with surgery and either management option provides similar outcomes [11]. Further, there is no clear evidence-based criteria to decide which patients should undergo surgery or not. When controlled early mobilization is used in non-operative management, re-rupture rates seem to be the same as in surgical management [11]. Meta-analysis research shows that there is no significant difference in rates of deep infections, deep venous thrombosis, calf circumference, strength of extremity, functional outcomes, or the number of patients who successfully return to sports after either treatment method [11]. The re-rupture rate is less than 5% with either treatment method [2].

When making the decision between surgical and non-surgical treatment of Achilles tendon rupture, the degree of tendon retraction, the patient's age, activity level, general medical condition, and general surgical risk must all be considered. That being said, surgery is usually reserved for the young, athletic patient, who is looking to return to playing sports with non-surgical treatment being generally best for older, less active patients with comorbidities [5-7]. Non-surgical treatment usually entails two weeks of immobilization in plantar flexion. This is followed by six to eight weeks of cast or flexible splint immobilization with progressive dorsiflexion to neutral and progressive weight bearing. The next phase of rehabilitation is placement of a heel-lift in the shoe and progression to normal shoes. Resistive exercises of the ankle are started by 8-10 weeks post injury and return to sports is projected at four to six months. Maximum recovery can take up to one year [7]. The surgical option rehabilitation course is very similar except that the course overall, is shortened with range of motion and strengthening beginning earlier.

Another option is available to the trained athlete with an Achilles tendon rupture; surgical repair with placement of bone marrow aspirate concentrate (BMAC) in the wound at the time of surgery. BMAC contains mesenchymal stem cells and hematopoietic stem cells. A single study of 27 patients beginning in 2009, where BMAC was used at the time of surgery, showed patients were able to return to work early, returned to their particular sport, had normal calf circumference after completion of rehabilitation, and all with no re-ruptures of the tendon in the patients who participated in the study [12].

Tendinopathies, Fluroquinolones, and Corticosteroid Usage

No modern discussion of spontaneous tendon ruptures and tendinopathies could be complete without discussing the ramifications of taking any antibiotics in the fluroquinolone group or corticosteroid usage and the associated increase of tendinopathies. The first reported case of fluroquinolone associated Achilles tendinopathy was described in 1983 [4]. By the mid-1990s, fluroquinolone antibiotics were being implicated in the etiology of Achilles tendinopathy and subsequent tendon ruptures including the highly unusual bilateral partial Achilles tendon ruptures [13]. Bilateral patella and Achilles tendon ruptures continue to be reported in the current literature [3,4]. Fluroquinolone associated tendinopathies remain a rare side effect with an estimated incidence of between 0.14-0.4% [4]. This tendinopathy usually occurs within the first 2 weeks of treatment but cases have been described up to six months after therapy termination [4]. The Achilles tendon is the most effected tendon, with the mean affected age being 64, male to female ration 2:1, and with a 27% bilateral involvement [4]. It is therefore imperative that any patient with a tendinopathy be questioned as to antibiotic use and if on fluroquinolone, discontinue its usage immediately [4,13].

Tendinopathy is now considered a fluoroquinolone class-wide toxicity [14]. On May 12th, 2016, the Food and Drug Administration stiffened an already significant "Black Box" warning from 2008 on all fluoroquinolone antibiotics concerning their usage and possibility of resultant tendinopathy. Other co-morbidities include higher doses and longer durations of taking the medication being most commonly associated with tendinopathy, patients who are over 60 years old, concomitant therapy with corticosteroids, the presence of renal dysfunction and history of solid organ transplantation [14]. Research has shown that cells treated with both levofloxacin and dexamethasone exhibit synergistic toxicity more pronounced than drug exposure to either drug alone [14].

Conclusion

Treatment of Achilles tendinopathies is complex. To have the best patient outcomes, the clinician must accurately diagnosis the level of injury as near to the time of injury as possible. Whether the tendon is inflamed, degenerated, partially torn, or completely ruptured must discovered and appropriate treatment instituted as each condition has different treatment protocols. It is imperative that the clinician be knowledgeable as to the anatomy of the injury area, the different treatment criteria, the available options to the patient,

and the probably outcomes of those treatments. Even though injuries to the Achilles tendon are common, care for the patient with this type of tendon injury is not.

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