Acute Achilles Tendon Rupture

Clinical Evaluation, Conservative Management, and Early Active Rehabilitation

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KEYWORDS

• Functional rehabilitation • Nonsurgical treatment • Postsurgical rehabilitation

KEY POINTS

- History and physical examination is the cornerstone for acute Achilles tendon rupture. Imaging is rarely needed for diagnosis, as physical examination is highly sensitive and specific.
- Conservative treatment provides the same outcome as surgical treatment without the risk of surgery, but only when functional rehabilitation protocols are used.
- There remains variability in reported functional rehabilitation protocols, although it is clear that early weight bearing with controlled early range of motion improve patient satisfaction and outcome.
- Regardless of conservative or surgical treatment, functional rehabilitation should be used.

INTRODUCTION

The Achilles tendon (AT) is the strongest and largest tendon in the human body. It is also the most frequently ruptured. The incidence of rupture has reportedly risen over time.[1–4] Increase in incidence is often attributed to increasing elderly and obese populations as well as increase in recreation sporting activities by middle-aged individuals.[5] Investigations into etiology have observed multiple factors that may be contributory to primary AT rupture.

Treatment of AT rupture remains controversial as risk and benefits of conservative versus surgical repair continue to be debated.[6] The conclusions of most early studies observing surgical versus conservative treatment were that surgery provides lower risk of re-rupture with higher complication rates.[7–13] Conservative treatment has gained increasing support with the advent of functional rehabilitation protocols, with

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more recent studies concluding that nonoperative treatment with functional rehabilitation is preferred. It leads to similar re-rupture rates and functional outcomes as surgery, with decreased complications.[14–18] Functional rehabilitation in the postoperative setting has also increased over the past few decades with studies suggesting it can reduce time to return to normal activities, increase patient satisfaction, and improve functional outcome.

EPIDEMIOLOGY AND ETIOLOGY OF RUPTURE

Incidence rates of AT rupture range from 11 to 37 per 100,00 population.[19–23] Gender distribution is predominately male with a reported male-female ratio ranging from 2:1 to 12:1.[24] Epidemiologic studies show an increase peak in incidence in middle-aged (30–39 years) individuals with AT rupture related to sports injuries and a second smaller peak due to other causes in later in life. Moller and colleagues[25] and Nillius and colleagues[26] showed the second peak to occur in the eighth decade of life, whereas Leppilahti and colleagues[21] reported the second peak distribution in the fifth decade.

Rupture of the AT most commonly occurs 3 to 6 cm proximal to the calcaneal insertion, with most injuries occurring during athletic activity.[27] The mechanism of injury is classified into 3 categories. First, weight bearing with the forefoot pushing off and the knee extended; second, an unanticipated dorsiflexion of the ankle; and third, a violent dorsiflexion of a plantarflexed foot.[28] Although the mechanism of rupture is observable, it is still unclear why the tendon itself ruptures and several theories have been advocated for. The location of rupture is partially explained by very high peak stresses experienced in this midportion area.[29] These stresses can lead to damage in tendons that are otherwise free of degenerative changes.[30]

It has been suggested that the failure of inhibitory mechanisms that would normally protect against excessive and/or uncoordinated muscle contractions could cause rupture at the site of maximum stress, and that athletes returning following inactivity may be particularly susceptible to this.[31]

Degenerative changes have been observed in AT ruptures. These changes can result from multiple factors, including chronic overloading and microtrauma, pharmacologic treatments, and reduced vascularity with associated heat necrosis.[32–39] Ischemia of the AT at the watershed zone has long been described as a factor in tendon rupture. This is despite recent experimental evidence showing uniform hemodynamic flow throughout the tendon at rest and at work. This is discussed in detail in this issue (See Paul Dayton's article, "Anatomic, Vascular, and Mechanical Overview of the Achilles Tendon," in this issue). It has been observed that corticosteroid use can cause degenerative tendon changes due to necrosis and delay in healing[40] and that the anti-inflammatory effect of steroids may mask painful symptoms of a damaged tendon, increasing the risk of rupture.[41] A study in 2004 by Gill and colleagues,[42] however, failed to show any causality between steroid injection for Achilles tendinopathy and AT rupture. Fluoroquinolones have been associated with AT degeneration and rupture as well. Multiple studies, including a cohort study of more than 6 million people, show increased risk for AT rupture with use.[43–47] Degenerative changes from fluoroquinolones likely follow from the drug's side effect of decreased decorin transcription.[33] Decorin is a small proteoglycan molecule that plays an important part in maintaining the molecular integrity of a tendon. Its decreased transcription is hypothesized to alter the viscoelastic properties of tendons and increase fragility.[39]

Claessen and colleagues,[48] in 2014, performed a systematic review to assess the many reported factors contributing to Achilles rupture. They identified nonmodifiable