Noninsertional Tendinopathy of the Achilles

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KEYWORDS

• Achilles tendinopathy • Achilles tendinosis • Noninsertional tendinopathy
• Noninsertional Achilles tendinosis

KEY POINTS

- The clinical triad of pain, swelling, and decreased performance ability indicates the diagnosis of Achilles tendinopathy.
- The current understanding of the etiology likely involves a combination of overuse leading to repetitive microtrauma, poor vascularity of the tissue, mechanical imbalances of the extremity, genetic predisposition, and a variety of metabolic factors.
- Conservative measures are generally recommended as the initial treatment strategy with an attempt to address and correct some of the underlying etiologic factors, followed by activity modification, medications, and stretching/strengthening programs.
- Surgery is considered an acceptable option for patients who do not respond to conservative measures.

INTRODUCTION

Noninsertional Achilles tendinosis is a condition that causes a predictable triad of symptoms, including pain, swelling over the affected site, and decreased performance ability. Achilles tendinosis must first be differentiated based on anatomic location—it can be subdivided into insertional (arising at the tendo-Achilles junction) or noninsertional (located 2–6 cm proximal to this Achilles insertion).[1,2] It is also important to differentiate between tendonitis and tendinosis with regards to Achilles pathology. Tendonitis refers to primarily an inflammatory response within the tendon after mechanical overloading, typically in an acute setting. Tendinosis on the other hand is primarily a degenerative process referring to a chronic overuse phenomenon whereby repetitive strains cause repeated tears and degeneration. Thus, the tissue's inability to adequately heal perpetuates pain and functional limitations and the role of inflammation although controversial is thought to be limited.[3,4] This has important implications in treatment approaches, which are discussed.

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EPIDEMIOLOGY

Achilles tendinosis is frequently seen in the athletic population and is associated with activities that require running and jumping. Achilles tendinopathy can also be found among patients who do not participate in sports.[5,6] High prevalence in runners has been reported in studies with prevalence ranging between 8% and 9%.[7–12] Similar to degenerative changes seen elsewhere in the musculoskeletal system, it is likely that many individuals have histologic findings of tendinosis, but are asymptomatic throughout their lifetime.

ETIOLOGY

The exact etiology of noninsertional Achilles tendinosis is unclear. Several explanations have been proposed, and it is likely a multifactorial process. In the future, the relative impact of the different contributing factors will need to be elucidated. Broadly, these factors can be categorized into the framework of intrinsic (ie, age, gender, body mass index, biomechanical abnormalities, foot malalignment, gastrocnemius-soleus dysfunction, ankle instability, and so forth) and extrinsic (ie, steroid use, fluoroquinolones, improper training, environmental factors, footwear, and so forth) factors.[13] Several pathophysiologic processes thought to be associated with tendinosis are discussed.

Mechanical Injury

Mechanical injury to the tendon is the inciting stimulus toward pathology. It may arise from physiologic overloading of the tendon or by accumulation of multiple physiologic loads without adequate healing. Healing in the setting of the Achilles tendon is likely mediated by tenocytes that detect alterations in the extracellular matrix. It has been suggested that failure to restore this extracellular matrix may result in release of cytokines that further modulate tenocyte activity and inhibit proper healing creating a degenerative spiral.[14]

Activities that require strong repetitive toe push-off forces, such as running and jumping, generate both tensile and torsional forces and predispose an individual to overuse and increased mechanical loads up to 9000 N.[15,16] Although an acute overload may result in an inflammation of the synovial sheath of the Achilles (as in tendinitis), repetitive physiologic loads without adequate healing predispose to degenerative changes within the tendon substance itself.[17,18]

Vascular Anatomy

Proponents of a vascular etiology for Achilles tendinosis suggest that pathology stems from the unique pattern of flow to the Achilles tendon. Although this topic has been investigated many times, there is no uniform consensus regarding either the Achilles' exact vascular topography or the uniformity of its flow.[19–23]

Chen and colleagues[24] performed an anatomic, histologic, and angiographic cadaveric study and concluded that the Achilles tendon is supplied by 2 arteries, the posterior tibial and peroneal arteries. They designated 3 vascular areas, with the midsection supplied by the peroneal artery and the proximal and distal sections supplied by the posterior tibial artery. They noted that the midsection of the Achilles tendon was markedly more hypovascular that the rest of the tendon in all specimens. From this they suggested the midsection of the Achilles was at highest risk for rupture as well as complications after surgery.

Astrom and Westlin[25] conducted a study looking at blood flow specifically in patients with chronic Achilles tendinopathy compared with normal controls. They used laser Doppler flowmetry at rest and during physical provocation by passive