ELSEVIER

Operative Techniques in

Sports Medicine

Achilles Injuries in the Athlete: Noninsertional

Nicola Maffulli, MD, PhD, FRCP, FRCS,[*,†] Alessio Giai Via, MD,[‡] and Francesco Oliva, MD, PhD[‡]

Achilles tendinopathy is common in both athletic and nonathletic individuals, and the incidence has risen in the last few decades. Although Achilles tendinopathy has been extensively studied, there is a clear lack of properly conducted scientific research to clarify its cause, pathology, natural history, and optimal management. The treatment of Achilles tendinopathy lacks evidence-based support, and individuals having tendinopathy are at risk of long-term morbidity with unpredictable clinical outcome. Most patients respond to conservative treatments. When conservative management is unsuccessful, surgery is recommended. Similar results have been reported with both open and minimally invasive techniques. However, minimally invasive techniques appear to reduce the risks of infection and provide better cosmetic results. An Achilles tendon rupture is frequent in young athletes and middle-aged people who practice recreational activities, and it is a serious injury. The management should take into account the age, occupation, and level of sporting activity. Open surgery provides good functional results and a lower rerupture rate, but it is frequently associated with a higher risk of superficial skin breakdown and wound problems. Percutaneous repair aims to provide good functional outcome while decreasing the problems associated with open surgery for wound healing and skin breakdown. Percutaneous repair followed by early functional rehabilitation is becoming increasingly common and should be considered in selected patients.

Oper Tech Sports Med 22:321-330

KEYWORDS Achilles tendon, tendinopathy, rupture, repair, minimally invasive surgery

Introduction

Achilles tendinopathy and rupture can occur in athletes. Achilles tendinopathy is a clinical condition characterized by both pain and pathologic changes in and around the tendons. In the past 3 decades, the incidence of Achilles tendinopathy has risen because of greater participation in sporting activities. It is estimated to occur in approximately 7%-9% of top-level runners. A 10-fold increase in Achilles tendon (AT) injuries has been reported in runners when compared with that in age-matched controls.[1] Achilles tendinopathy is also common among people participating in racquet sports, track and field, volleyball, and soccer. However, the condition is by no means confined to athletes. In a recent study, 31% of 58 patients with Achilles tendinopathy did not participate in sports activities.[2]

Acute AT ruptures are frequent in young athletes and middle-aged people who practice recreational activities.[3] Most of these injuries occur in soccer, tennis, badminton, and squash players, but 25% of ruptures occur in sedentary patients as well. The incidence rate ranges from 6-18 per 100,000 per year.[3] Although the rupture seems to occur because of a traumatic injury on a healthy tendon, in reality, it is caused by an eccentric contraction on a pathologic asymptomatic tendon.[4]

Etiology and Pathophysiology

The etiology of Achilles tendinopathy remains unclear, and many predisposing factors have been proposed. AT injury can be acute or chronic, and it is caused by either intrinsic or

*Department of Musculoskeletal Disorders, School of Medicine and Surgery, University of Salerno, Salerno, Italy.
†Mary University of London, Barts and the London School of Medicine and Dentistry, Centre for Sports and Exercise Medicine, Mile End Hospital, London, UK.
‡Department of Orthopaedic and Traumatology, University of Rome "Tor Vergata," School of Medicine, Rome, Italy.
Address reprint requests to Nicola Maffulli, MD, PhD, FRCP, FRCS, Mary University of London, Barts and the London School of Medicine and Dentistry, Centre for Sports and Exercise Medicine, Mile End Hospital, 275 Bancroft Rd, London E1 4DG, UK. E-mail: n.maffulli@qmul.ac.uk

http://dx.doi.org/10.1053/j.otsm.2014.09.001
1060-1872/

extrinsic factors. In acute trauma, extrinsic loads exceed the tensile strength of the tendon. Extrinsic factors such as increased frequency and duration of training have been associated with Achilles tendinopathy. Changes in training pattern; poor technique; previous injuries; footwear; and environmental factors, such as training on hard, slippery, or slanting surfaces, may predispose the athlete to this condition.[5] However, a retrospective study showed that tendinopathy was not necessarily associated with the level of physical activity.[6] Fluoroquinolones and corticosteroids have been implicated as risk factors in tendinopathy. Ciprofloxacin causes enhanced interleukin-1β–mediated matrix metalloproteinase 3 (MMP3) release, inhibits tenocytes proliferation, and, as does corticosteroids, reduces the collagen and matrix synthesis.[7]

Intrinsic factors include alteration of lower limb function, biomechanics, sex, age, and genetics. Lower limb alignment and biomechanical faults are claimed to play a causative role in two-thirds of athletes with AT disorders. In particular, hyperpronation of the foot has been linked with an increased incidence of Achilles tendinopathy.[8] The incidence of tendinopathy is lower in women than that in men, and estrogen levels might play an important role in tendon homeostasis.[7] Women showed a lower risk of tendinopathy during premenopausal years than men did, but after menopause, this risk increases.[9] Postmenopausal estrogen deficiency seems to downregulate collagen turnover and decrease the elasticity in the tendons. Cook et al[10] reported that AT health of women using hormone replacement therapy was better than that of the controls. An animal study showed poorer AT healing in estrogen-deficient rats compared with that in controls.[11] Little is known about the effect of estrogen on tenocytes. A recent in vitro study showed that proliferation and tenocyte biosynthesis are negatively affected by age and estrogen deficiency.[12] Tenocytes from excised ovary and old rats showed a significantly lower proliferation rate, decreased collagen type I synthesis, and overexpression of MMP compared with that in young controls, showing that aging and, more significantly, estrogen levels may affect tendon metabolism and healing.

Age and some metabolic diseases, such as diabetes mellitus and obesity, are also predisposing factors. Connective tissue aging is associated with compromised tissue function, increased susceptibility to injury, and reduced healing capacity. This has been partly attributed to collagen cross-linking by advanced glycation end products (AGEs) that accumulate with both age and chronic diseases, particularly diabetes mellitus.[13] Protein glycation is a spontaneous reaction depending on the degree and duration of hyperglycemia, the half-life of the protein, and permeability of the tissue to free glucose. Glycated proteins can undergo further reactions giving rise to poorly characterized structures called AGEs.[13] AGEs are complex, heterogenous molecules that cause protein cross-linking, which alter physical characteristics of collagen fibers. In tendons, AGE formation has been shown to affect the interactions between collagen fibers, extracellular matrix (ECM) protein, and tenocytes.[14] These changes have been associated with both reduced healing capacity and altered mechanical properties of connective tissues. The effects of AGEs on the mechanical properties of tendon collagen fiber have been recently studied in a rat model.[15] The formation of AGEs would change the way a tendon reacts to loading, significantly reducing collagen fiber sliding in particular. Conversely, tendons try to compensate for this loss of function by increasing collagen fiber stretch, which may have potentially important implications for predisposing collagen fibrils to damage during everyday use. The tissue stiffness does not appear to be significantly affected. Therefore, physiological loads in the tendons of patients who are aged and diabetic could involve fiber "overstretching" that leads to accelerated accumulation of damage.

These findings may have important clinical consequences, because middle-aged people who are used to playing sports regularly do not change their habits, but changes in the physiology and function of connective tissues begin to occur, and this leads to a higher rate of tendon injury.

The physiopathology of tendinopathy in patients with obesity is yet to be understood, but some studies show that obesity may affect tendon health and reduce its healing ability.[16] Many authors consider obesity as a risk factor for tendon injury,[17] and poorer outcomes have been reported after arthroscopic rotator cuff repair in patients with obesity than that in controls.[16] Anatomical studies show that AT thickness is significantly higher in individuals with obesity than in control groups,[18] and ultrasound images showed thicker and hypoechogenic tendons in subjects with obesity compared with that in normal people.[19] Histologic changes have been observed in animal studies. Lipid drops accumulate in the ECM.[20] During ultrastructural analysis by transmission electron microscopy, disorganized collagen fibrils can be observed in the ECM of tendons in obese animals.[21] Low levels of glycosaminoglycans (chondroitin and dermatan sulfate), which play an important role in the regulation of the ECM and collagen fibrillogenesis, have been detected, which may be responsible for the inadequate deposition and organization of collagen fibrils.[22] Finally, obesity is frequently associated with other pathologies, such as diabetes mellitus and insulin resistance, which may also play a role in tendon pathology.

Changes in the expression of genes, regulating cell-cell and cell-matrix interactions in Achilles tendinopathy, have been reported, with downregulation of MMP3 and upregulation of MMP2 messenger RNA in tendinopathic AT samples.[23]

Clinical Presentation

Pain is the main symptom of Achilles tendinopathy. Pain occurs at the beginning and a short while after the end of a training session, with a period of diminished discomfort in between. As the pathologic process progresses, pain may occur during the entire exercise session, and in severe cases, it may interfere with activities of daily living. Although significant advances have been made for understanding the pathologic changes in both the ECM and the tenocytes, relatively little is known about pain. Traditionally it has been thought to arise through inflammation or via collagen fiber degeneration. However, chronically painful ATs have no evidence of inflammation, and many tendons with intratendinous pathology detected on magnetic resonance (MR) or ultrasound