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Review

Achilles Tendon Rupture

A Review of Etiology, Population, Anatomy, Risk Factors, and Injury Prevention

Gregory William Hess, MS, ATC/L, CSCS

Abstract: Sports participation has undergone an increase in recent decades. Injury due to sporting activity has also recently risen. The Achilles tendon has been one of the most common sports-related injuries. A 2 in 100 000 individual Achilles tendon injury rate increased to a 12 in 100 000 individual injury rate in less than 10 years. The injury is typically observed in men in the fourth to fifth decades of life. Male to female injury ratios range from 2:1 to 12:1. Running, jumping, and agility activities involving eccentric loading and explosive plyometric contractions are usual mechanisms. Natural aging allows predisposing chronic degeneration of the tendon. Blood flow decreases and stiffness increases with aging to decrease the ability to withstand stress. Noninflammatory tendinosis and chronic tendinopathy are 2 separate processes proposed for tendon degeneration and subsequent rupture. Rupture typically occurs 2 to 6 cm proximal to the calcaneal insertion. Predisposing factors are grouped into 2 categories: intrinsic and extrinsic risk factors. Avoidance of degenerative changes within the tendon is the primary method to prevent rupture.

Regular physical activity as athletes age also promotes tendon hypertrophy, increases nutrient delivery, and reduces collagen fiber fatigue.

Keywords: Achilles tendon; rupture; population; risk factors; prevention

Etiology and Population

Popularity with recreational and competitive sport has undergone a marked increase in recent decades.^{1,2} With this increase, injury has also risen since the first documented Achilles tendon rupture in 1575 by Ambrose Paré. This is particularly true with the Achilles tendon, one of the most common sports-related injuries.^{2,3} Schepesis et al⁴ reported a 2 in 100 000 individual Achilles tendon injury rate in 1986 that increased to a 12 in 100 000 person injury rate in 1994. Typically, this injury is observed in men in the fourth to fifth decades of life.⁴ In fact, male to female injury ratios range from 2:1 to 12:1.³ Running, sprinting, jumping, and agility activities involving explosive plyometric contractions are usual mechanisms.^{3,5-7}

Initially, the injured individual experiences a sudden popping event that might be described as a “kick from behind” or a sudden snap in the calf.^{4,5} A rapid eccentric loading or an explosive plyometric-based activity will occur with 1 of 3 common mechanisms. In addition, this excessive loading of the Achilles tendon with inverting and evverting of the subtalar joint furthers the likelihood of injury.⁸ Pushing off the weight-bearing foot with the knee extended, unexpected dorsiflexion of the ankle, and violent

“Avoidance of the degenerative changes within the tendon is the primary means to prevent Achilles tendon rupture.”

dorsiflexion of a plantar flexed ankle are the usually reported mechanisms for Achilles tendon rupture.^{8,9} Maffulli et al⁹ specifically reported 53% of Achilles tendon ruptures to occur with a push-off mechanism, 17% with sudden and unexpected dorsiflexion of the ankle, and 10% with violent dorsiflexion of a plantar flexed foot.

Peak stress during these contractions can reach upward of 2233 Newtons, or

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6 to 12 times body weight.^{4,10,11} These values approach the physiological strength limit of a healthy Achilles tendon.⁴ If symptomatic or asymptomatic degeneration and overloading of the tendon occur over an extended period, macroscopic failure of the entire tendinous structure is likely.¹² The 3- to 6-cm region proximal to the Achilles tendon's calcaneal insertion is the most common rupture location because of the small cross-sectional area, large eccentric loads, and hypovascularity.¹²⁻¹⁴

Natural aging during a person's lifetime allows chronic degeneration of the tendon and failure of normal inhibitory mechanisms.¹⁵ Blood flow decreases to the susceptible region with age, as does the tensile strength of collagen.⁹ Stiffness of the tissue also increases with aging to decrease the ability to withstand repetitive stress.⁸ These in turn permit forceful and sudden contractions to tear the tendon.¹⁵

Anatomy Related to Injury

The Achilles tendon is the extension of 2 separate muscles.³ The gastrocnemius muscle originates from the supracondylar ridge and adductor tubercle of the posterior femur medially and the lateral condyle of the posterior femur and proximal and posterior lateral epicondyle laterally. The soleus originates from the posterior surface of the upper tibia and the posterior aspect of the proximal third fibula. These independently functioning muscles have movement ability of their own, depending on knee positioning.⁴ In all, the gastrocnemius and soleus muscles are the chief plantar flexors of the ankle, allowing for locomotion and force production at the ankle.⁴ Both muscles merge at the mid-calf region of the lower leg, where the Achilles tendon begins and become a single tendon near 5 to 6 cm proximal to the calcaneal insertion.^{3,4} Tendon fibers begin to exhibit rotation or spiraling at approximately 12 to 15 cm proximal to the insertion.⁴ This spiraling causes near 90 degrees of rotation, with the medial fibers rotating posteriorly and the lateral fibers rotating laterally.⁴ Greater elastic and recoil properties

are enabled by the spiraling of the tendon fibers.^{4,9}

The Achilles tendon is not covered by a synovial sheath but by a peritenon.³ This peritenon is a single-cell layer that enhances gliding function during dynamic activity and provides vascular support to the tissue within.^{4,15} Other vascular supply originates from the musculotendinous junction and the osteotendinous junction.⁴

Fibers within the extracellular matrix of the Achilles tendon are primarily parallel-oriented collagen, composing approximately 70% of the matrix.⁷ Collagen is responsible for the resistance to tensile stresses, resulting from active movement and explosive contractions of the plantar flexors.⁷ At rest, the collagen appears wavy and relaxed.⁹ The wavy appearance disappears when loads are placed on the tendon and failure of the collagen cross-links begin to occur near a 4% stretch.⁹ Beyond an 8% stretch, macroscopic failure of the collagen fibers and complete rupture of the tendon are likely.⁹ Normally, the Achilles tendon is able to withstand peak stresses of greater than 70 mPa; however, failure has been noted at the 100-mPa loading rate.¹² Even the slightest defect to the tendon structure can initiate large-scale tendon failure.

Two separate processes for Achilles tendon degeneration and subsequent rupture have been proposed.¹⁶ Tendinosis, a noninflammatory and degenerative process, may occur to predispose the tendon without symptoms prior to rupturing the tendon.¹⁶ Chronic tendinopathy may also occur, where long-term paratenonitis manifests via pain with activity and marked edema.^{4,16}

Achilles tendons experiencing noninflammatory tendinosis lack clinical symptoms to forewarn the clinician and the athlete.^{4,10} Collagen fibers become disorganized, with type I collagen production being replaced by weaker type III collagen from tenocytes.^{9,16} Increased separation of the smaller diameter collagen fibers occurs with disappearance of the normal parallel fiber arrangement.^{12,16} The tenocytes also begin to round and assume the resemblance of chondrocytes.¹⁶ Furthermore, blood supply to the

tendonotic tendon has been observed to be random.¹⁶ These changes may equate to spontaneous tendon failure with specific acceleration and deceleration due to the imbalance between tendon degradation and repair.^{4,16}

Chronic tendinopathy typically results when overuse occurs.¹³ Inflammation of the tendon results in impaired gliding within the paratenon due to tendon thickening.¹³ Hypoxic mucoid degeneration with noduling and calcific formations may initiate problematic fissuring within the tendon, decreasing tensile strength.^{3,10} In addition, chronic overuse increases avascularity at the tendon's insertion, preventing repair. Overall, prolonged paratenonitis increases the likelihood of microscopic collagen failure and further degeneration, comparable to non-inflammatory tendinosis.¹³

The location of Achilles tendon rupture is within a poorly vascularized zone, 2 to 6 cm proximal to the calcaneal insertion.¹⁵ In fact, 80% of Achilles tendon ruptures occur within this region.¹² At this location, the tendon is narrowest with the smallest cross-sectional area of the entire structure.^{3,12} With compounded degeneration and insufficient nutrients to repair the damaged tissue within this naturally avascular zone, tendon rupture is common.¹⁵

Risk Factors for Injury

Factors that predispose athletes for Achilles tendon rupture are grouped into 2 categories: intrinsic and extrinsic risk factors.^{7,17} Combined, the presence of these characteristics furthers the likelihood of an acute Achilles rupture. Intrinsic risk factors include any anatomical predispositions or inability of the body's biomechanics to naturally absorb force, whereas extrinsic risk factors are composed of errors in training techniques or environmental factors.^{4,7,17}

Biomechanically, subtalar hyperpronation of the foot causes a whipping-like action to occur at the Achilles tendon.^{3,4,9,13} Upon repetition at heel strike, shear forces will occur across the Achilles tendon, causing high eccentric stresses at the medial aspect of the

tendon.⁴ Microtearing occurs with overuse, which then predisposes the tendon to unequal distribution of tensile loads during other activity.⁹ To further the anatomically intrinsic factor at the foot, excessive rearfoot and forefoot varus and valgus cause unequal distribution of tensile forces on the Achilles tendon.¹⁰ It has been also noted that tibial varum and calcaneovalgus existence alter normal tensile stress placed on the Achilles tendon.¹⁵ In all, the shock absorption capacity of the tendon is decreased when these distal kinetic chain disturbances persist.

Increased femoral anteversion similarly initiates compensatory internal rotation of the lower limb to correct body positioning.³ This potentially compounds the pronation risk factor, so that the center of gravity is located correctly.³

Musculotendinous flexibility has also been identified as a potential risk factor. Tight triceps surae or hamstring muscle groups may cause compensatory motion at the ankle, producing heel varus and pronation.^{3,15} Once again, tensile force will not be absorbed or distributed appropriately. With repetition of this faulty biomechanical state, collagen microfailure can evolve to tendon macrofailure. In addition, inadequate warm-up and stretching prior to dynamic activity have been proposed as preventing needed creep and stress relaxation responses of the tendon tissue.³ Failure to properly prepare the active tissue for dynamic and usually explosive loads can lead to acute failure.

Other intrinsic risks are related to comparable alterations in the distribution of forces and the absorption of these forces. Leg length discrepancy alters normal gait and changes directional loading of stresses on the tendon.¹⁷ Muscle weakness and resulting muscular imbalance prevent successful force distribution and may require excessive force to be dissipated by the Achilles tendon.¹⁷ Also, overweight individuals naturally will overload the musculotendinous structure because they possess excessive body mass, which the tendon cannot withstand during movements with elevated rates and magnitudes.^{7,17} Finally, aging of the

individual and tendon alters normal and younger tendon properties.³ Increasingly stiffer musculotendinous junctions, decreasing tensile strengths, and decreasingly less strain to cause collagen failure all occur as one ages.³ Therefore, levels of activity intensity and volume must be progressed to allow structural adaptation and repair.

Extrinsic factors are composed of primarily errors in training. Running durations and intensities above individualized levels, plyometric activity that is novel and excessive, and environmental surface that is unfamiliar all lead to degeneration of the extracellular matrix.^{7,10,15,17} Excessive running mileage composes the overuse aspect that, when applied to intrinsic factors, may result in failure of the tissue. Plyometrics, such as jumping, are forceful concentric contractions that occur following a forceful eccentric moment and amorphization period.^{7,15} Inability of the elastic tendon components, such as the Golgi tendon organ, or simply extreme loading of the tendon results in rupture. Poor or altered environmental contact surfaces add new stresses to the tendinous structures.¹⁰ An athlete adapted to a paved running trail or basketball court may not experience the same detrimental incident as an athlete unaccustomed to the same surface. These factors alone may not cause acute tendon rupture; however, without adequate recovery and cellular repair time, a compounding effect occurs. This is especially true for the aging athlete.

Two drugs that have been associated with delayed healing and tendon necrosis are fluoroquinolone antibiotics and corticosteroids.^{4,7,9,11,15} Fluoroquinolone antibiotics have been observed to weaken the Achilles tendon extracellular matrix, resulting in less tensile tendon strength.¹¹ Corticosteroids, used to decrease tissue inflammation, also cause collagen to weaken and decrease blood supply to an already avascular structure.^{3,11} Although corticosteroids are common in the use of tendinopathy, athletes must rest for a period to allow normal collagen strength and alignment to return.

Prevention of Injury

Avoidance of the degenerative changes within the tendon is the primary means to prevent Achilles tendon rupture.¹² Collagen must retain necessary tensile strength to resist the applied forces.⁷ Furthermore, the production of weaker type III collagen must be avoided.¹¹ Maintained blood supply via regular physical activity is advised.⁸ Maintenance of physical activity levels as athletes' age increases will also promote tendon hypertrophy, increase nutrient delivery, and reduce fiber fatigue accumulation.⁸

In addition to physical activity, warm-up periods prior to exercises can increase temperature of tissue and general preparation of the tendon for loading.⁸ Extensibility of the fibers will be increased as warm-up periods are instituted.¹⁸ Adding stretching to the preactivity routine, although not proven to prevent rupture, may enhance this preparatory extensibility.³

Strengthening of the ankle plantar flexors has also been linked to the prevention of Achilles tendon rupture.³ Eccentric plantar flexor strengthening is specifically recommended because of the force generated during eccentric contraction and with functional activity.³

The use of corrective orthotics may also be used to correct foot malalignments.³ Hindfoot and forefoot varus and valgus can be specifically and individually targeted with these relatively inexpensive shoe inserts.³ Typically, the materials will provide a resilient posting of the arch or a cushioning effect, depending on specific needs.¹⁸

Finally, avoidance of fluoroquinolone antibiotics and prevention of corticosteroid abuse must be ensured. This will prevent weakening of the tendon's extracellular matrix, preserving tensile strength.¹¹

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