

Achilles Tendon Rupture

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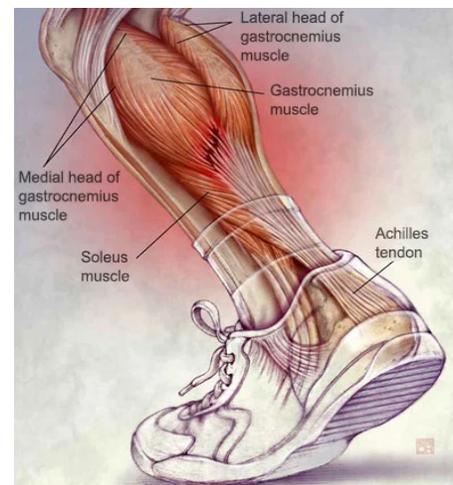
Introduction:

The Achilles tendon is the largest and strongest tendon in the human body and is located along the posterior aspect of the lower leg (calf). Measuring approximately 12-15 cm in length, it is formed from the muscles of the gastrocnemius and soleus, which merge and insert on the calcaneus (heel). “It arises near the middle of the calf and rotates approximately 90 degrees laterally during its course to insert on the posterior aspect of the calcaneal tuberosity...Despite its size and tensile strength, the Achilles tendon is the most commonly injured tendon in the human body” (3). Acute Achilles tendon rupture (AATR) is a relatively common injury, with an estimated annual incidence of 5 to 50 events per 100,000 individuals, and may result in severe disability and prolonged absence from work or physical activity (5).

Tendons transmit force from muscle to bone, as well as store elastic energy for movement. The Achilles tendon complex, including the gastrocnemius and soleus muscles and their tendinous attachments, is distinctive because it spans three joints: the knee, the ankle (tibiotalar), and the subtalar joints. Its primary functions are to transmit force between the calf and foot, and to store energy in order to release energy to support high-speed locomotion and overall movement efficiency. It plays a key role in foot plantar flexion, hindfoot inversion, and aids in knee flexion, all of which are crucial in walking, running and jumping (10).

“Acute rupture of the AT (Achilles tendon) can be caused by sudden excessive loading, cumulative degeneration, or less commonly as a manifestation of systemic disease” (13). Classification of Achilles tendon ruptures is commonly done using the Kuwada classification, which was established in 1990. This classification categorizes ruptures into four types based on the extent of the tear and size of the gap (“defect”) between the tendon ends (6).

The Kuwada classification has also been used to help guide management options according to the severity of injury (13).



(27) (Illustration)

Kuwada System

- 1) Partial tear of <50% of tendon fibers. Conservative treatment is often utilized.
- 2) Complete rupture, with less than or equal to 3 cm gap. Corrected with primary surgical repair and end-to-end anastomosis.
- 3) Complete rupture, with 3-6 cm gap. Secondary repair needed, often requiring a tendon or synthetic graft.
- 4) Complete rupture, with a greater than 6 cm gap. Complex reconstruction needed, with grafts and gastrocnemius recession.



(28) (Illustration)

Early classification is clinically important because it supports timely treatment decisions and may reduce the risk of substantial morbidity, impaired mobility, and prolonged loss of work time. In addition to acute rupture, other forms of Achilles tendon pathology may occur. “Achilles tendon disorders are now grouped together into what is known as Achilles tendinopathy. Achilles tendinopathy includes tendinosis and peritendinitis. Tendinosis is differentiated from tendonitis in that there is degeneration of the tendon without inflammation or evidence of intratendinous inflammatory cells” (6).

According to Pean et. al, “Achilles tendon rupture (ATR) is a fairly common injury that results in substantial morbidity with functional deficits persisting more than two years after the initial injury regardless of treatment” (26). “Pathology of the Achilles tendon can be acute or chronic, ranging from tendinosis to frank tears, and can broadly affect athletes and non-athletes alike” (23).

Forces Causing Injury:

The precise etiology of Achilles tendon ruptures is unknown; however two major theories have been proposed: (23)

1. Degenerative “low energy” theory: this theory assumes that chronic degeneration of the tendon may lead to a spontaneous rupture without the need for excessive force loads to be applied. Generally, these types of injuries occur later in life, after 60 years.
2. Mechanical “high-energy” theory assumes that different movements and forces exerted on the tendon will lead to failure. These types of ruptures are often seen in earlier ages, between 25-40 years.

Classic ATRs “are produced by a single high-load impact (for example, an ATR associated with sudden or violent dorsiflexion of the ankle or lunge). Moreover, an acceleration-deceleration mechanism has been reported in up to 90% of sports-related ATRs” (3). Patients typically report

no preceding “warning” signs or symptoms, and the rupture most often occurs during a single, discrete traumatic event involving the ankle. Because rupture commonly occurs during rapid, excessive dorsiflexion or during acceleration-deceleration movements associated with sudden changes in direction, it is frequently encountered in sports such as basketball, soccer, and tennis. In a study of NBA players with Achilles tendon rupture, all 13 athletes were in a dorsiflexed position at the time of injury and demonstrated a false-step mechanism, initiating movement with a rearward step of the injured limb posterior to the center of mass before translating forward (1). These same force patterns may also occur in non-sport settings, including motor vehicle collisions or falls from height.

Injury to the Achilles tendon is often multifactorial, with both intrinsic and extrinsic forces play a role in the rupture. Several factors have been identified in the literature as increasing risk of ATR, these include, but are not limited to: (3, 4, 5, 6)

- Gender (males > females).
- Degenerative changes.
- Age (30-50 yrs).
- Gastrocnemius-soleus dysfunction.
- Changes in training patterns, or poor technique with activity.
- Previous injuries.
- Various pathologic conditions (inflammatory conditions, autoimmune, etc.).
- Antibiotics (especially fluoroquinolones).
- Systemic or injectable steroid use.

Patient Presentation (Symptoms and Physical Findings):

“Patients with ATR report sudden and severe pain in the acute phase, and, if left untreated, the injury results in worsened physical function” (3). The clinical presentation is often characteristic, and a diagnosis can frequently be made based on the patient’s history and physical examination findings. Patients typically present with acute pain and swelling in the posterior ankle and calf and often describe a traumatic event or a sensation of being “kicked” in the back of the heel. An audible “pop” may be reported, followed by difficulty with ambulation, particularly with walking uphill or climbing stairs.

Clinical examination includes a thorough assessment of the lower extremities. The physician typically evaluates both the injured and non-injured leg to compare the degree of edema and bruising, calf circumference and atrophy; continuity of the Achilles tendon, including the presence of a palpable gap (usually found 2-6 cm proximal to the insertion of the tendon). Standard clinical tests include the Thompson test (formerly the “Simmonds” test), the Matles test, the O’Brien needle test, and Copeland sphygmomanometer test (6, 7, 14). Treatment outcomes have been evaluated using objective and patient-reported measures such as the Victorian Institute of Sports

Assessment-Achilles (VISA-A), the single-leg heel-rise endurance test, and assessment of movement-evoked pain with tendon-loading activities. These measures may be used alongside supine passive range of motion (PROM), manual muscle testing (MMT), weight-bearing (WB) functional testing, targeted special tests, and palpation to characterize impairment and track recovery.

Clinical Practice Guidelines issued by the American Academy of Orthopaedic Surgeons note that a diagnosis of acute Achilles tendon rupture may be made when two or more of the following examination findings are present (25):

1. Positive Thompson test (calf squeeze test).
2. Decreased plantar flexion strength (with heel lift test).
3. Palpable defect (gap) distal to the insertion site.
4. Increased passive ankle dorsiflexion (Matles test).

Diagnostic Testing:

Diagnosis of acute Achilles tendon rupture relies primarily on clinical examination, and a standard radiography may not be needed in straight forward cases. Ultrasound (US) and magnetic resonance imaging (MRI) may be utilized when needed in order to rule out differential diagnosis, obtain additional clinical detail, or to monitor the healing process. “Imaging, in the form of ultrasound and radiographs, or MRI, for Achilles tendinopathy is recommended when there is uncertainty in the diagnosis, if there is a delayed recovery, if there is negative change in symptoms over the course of care, or if a procedure is being considered” (2).

Surgical and Non-Surgical Treatment:

Tendon healing generally occurs in three phases: inflammation, proliferation and remodeling. “Tendon injuries are often treated with a variety of non-surgical (eg. immobilization, ice, physical therapy) and surgical interventions, along with reduction of physical activities to allow for healing” (12). Treatment options for ATR typically grouped into two broad categories: surgical repair and conservative (nonoperative) treatment.

Surgical repair can be achieved in a variety of methods, including:

1. Open repair.
2. Percutaneous repair.
3. Minimally invasive surgery/repair.
4. Augmentative repair: including fibrin sealant, platelet-rich fibrin matrix (PRF), and platelet-rich plasma (PRP).

The goal of any surgical intervention is to restore anatomic length and physiologic tension, while providing adequate strength to aid in returning to activity with normal function, strength, and ROM. Surgical repair is normally performed within 2 weeks (4). In general, operative management has been associated with earlier return to sport and lower re-rupture rates compared with traditional conservative treatment, although it carries inherent surgical risks. Percutaneous and minimally invasive techniques have been increasingly utilized in an effort to reduce wound complications, scar adhesions, and sural nerve injury while preserving the potential benefits of operative repair, including faster recovery and reduced re-rupture risk relative to nonoperative options (5, 9, 17).

Conservative treatment has evolved to include two main options for non-operative management.

1. Cast immobilization: Non-weight-bearing (NWB) up 4 weeks as directed, then transitioning to walking cast for an additional 4 weeks.
2. Functional bracing with early rehabilitation: patient placed in a boot with heel wedges to provide a gradual reduction in plantar flexion to neutral over 6 weeks, then beginning formal physical therapy. This group of patients is allowed to be WBAT immediately in the boot. In studying this group of patients, it has been found that there is an increased dorsiflexion ROM, earlier return to activities, lower re-rupture rates (similar to those seen in surgical groups), all without the surgical risk factors.

Current literature remains mixed regarding the optimal management strategy, and treatment selection is often individualized. Historically, operative repair was considered the standard approach. More recently, there has been a growing trend toward nonoperative management using early functional rehabilitation protocols, driven by evidence of favorable functional outcomes and low re-rupture rates while avoiding operative risks (21). One comparative analysis evaluating open versus endoscopic flexor hallucis longus (FHL) transfer reported comparable long-term functional outcomes (9). In addition, a 2022 randomized trial of 554 participants by Myhrvold et al. reported no meaningful differences in patient-reported outcomes between operative treatment (open or minimally invasive repair) and nonoperative treatment. Re-rupture rates were higher in the nonoperative group (6.2% vs 0.6%), whereas nerve injury rates were highest following minimally invasive repair (5.2%), followed by open repair (2.8%) and nonoperative treatment (0.6%) (19).

Rehabilitation Required:

Early functional rehabilitation is now frequently encouraged as science has proven mechanical stimulation and use plays a key role in tendon healing. “Tendon development and healing is a well-orchestrated process requiring highly ordered molecular signaling events influenced by mechanical stimulation and surrounding environmental factors”(15). Regardless of initial treatment strategy, emerging evidence is proving that early functional rehabilitation is crucial in any successful plan. “Early weightbearing and early functional rehabilitation after operative and non-operative treatment of ATRs has been advocated since they lead to new tendon formation and

better ultimate functional outcomes (such as return to work)” (3). “Protocols using early weight-bearing have two major goals. First, the mechanical loading enhances collagen maturation and consequently tendon healing. Second, the muscle atrophy associated with prolonged immobilization is prevented” (18).

Physical therapy plays a central role in recovery following acute Achilles tendon rupture and generally follows a staged approach, with specific interventions tailored to the severity of injury, treatment method, and clinical discretion. Rehabilitation is commonly organized into the following phases:

- Early phase/Protection: Immobilization, pain control, edema control (elevation, ice, compression), and gentle ROM (if allowed by physician) (generally weeks 0-2).
- Intermediate phase/Return to walking: Increasing ROM, beginning strengthening exercise, balance and proprioception, as well as gait training (generally weeks 2-6).
- Advanced phase/Strength progression: Functional exercise, plyometrics, and sport specific training if indicated (generally weeks 6-12).

Return to sport following Achilles tendon rupture is typically gradual and depends on the demands of the sport, the treatment approach, and the individual’s recovery. Available evidence suggests that return to regular sports participation should not occur earlier than 12 weeks after surgery and return to strenuous sport is often deferred for at least 12 months. In a meta-analysis including 15 studies, the overall return-to-play rate among professional athletes was 76%, with an average time to return of 11 months (31). These findings are consistent with Zellers and colleagues’ systematic review and meta-analysis of 85 studies, which reported a return-to-sport rate of 80% (32). Despite successful return to participation, many athletes continue to demonstrate performance limitations, and return to preinjury performance levels may remain difficult for up to 2 to 3 years postoperatively (9).

Postoperative Modifications:

Short-term management focuses on accommodating reduced weight-bearing, maintaining participation in activities of daily living (ADLs), and minimizing the risk of reinjury. Bathroom safety may be supported with a shower chair, handheld shower head, raised toilet seat with arm supports, and appropriate wound protection. Additional adaptive equipment may include a bedside commode, item carriers, and mobility aids such as crutches, a walker, or a wheelchair, as clinically indicated. Immobilization and stabilization devices, including the Air Cam Walker (United Ortho), AirSelect Walker (Aircast), and VACOPed (OPED Medical Inc.), and Breg boot are commonly used during the early phases of recovery (33). Tendon loading is then progressed under clinician guidance and individualized to the patient. Client-centered home modifications may include portable ramps or threshold adjustments, installation of grab bars and handrails, and, when needed, temporary relocation of living space to a single level or use of a stair lift.

Long-Term Modifications

If a patient experiences long-term complications such as tendonitis, re-rupture, or weakness, permanent changes should be considered to promote safety, independence, and mobility. These may include fixed grab bars in high-use areas, raised laundry baskets, anti-fatigue mats for prolonged standing, and supportive footwear to decrease pain and improve mobility. Community mobility devices, such as a power scooter with lift may be necessary as one ages. Adjustable beds can aid rest and assist with leg elevation, while recliners with or without lift functions support edema management, transfers, and independence as mobility declines with age.

Early Complications:

Potential complications of operative management for acute Achilles tendon rupture should be considered. Reported complications include deep vein thrombosis (DVT), re-rupture requiring reoperation, wound dehiscence, surgical site infection, sural nerve injury, tendocutaneous adhesions, and pneumonia (24). A meta-analysis by Kilkenny, et. al. reviewed long term outcomes following surgical interventions starting at five years post operative and found good long term outcome rates of total of 806 patients demonstrating re-rupture rates of 4.2%, revision rates of 3.7%, DVTs at 6.3% and wound infections observed in 3.6% (17).

Conservative treatment with prolonged cast immobilization has historically been associated with higher re-rupture rates. In contrast, when nonoperative management is combined with early functional rehabilitation, re-rupture rates have been reported to decrease to levels comparable with operative treatment. As noted in the literature, recent studies have demonstrated favorable outcomes of conservative treatment using accelerated functional rehabilitation. In such studies, functional rehabilitation was more effective in reducing re-rupture rates than long-term cast immobilization, and functional improvement after nonoperative treatment was comparable to that after operative treatment” (8, 20). Other authors similarly report that “patients treated nonoperatively show an increased rate of re-rupture only if functional rehabilitation employing early range-of-motion protocols are not used” (14).

Both operative and nonoperative approaches carry risks and potential complications, including joint stiffness, re-rupture, and deep vein thrombosis (24, 25).

Future Complications:

Achilles tendon rupture may result in persistent functional limitations in both athletes and non-athletes. According to a study by Hoeffner et. al, “in conclusion, this narrative review highlights

the fact that patient related outcome measures data currently available in the literature suggest that recovery following total Achilles tendon rupture takes longer than 6 months (Achilles Tendon Total Rupture Score, ATRS 70), that one year post injury, the ATRS only reaches 82, and that this remaining deficit does not appear to noticeably improve thereafter” (11).

When discussing chronic ATR, “In the absence of treatment for more than four weeks, the tendon ends retract and the gap is filled with fibrous tissue, resulting in the rupture being defined as a “chronic” Achilles tendon rupture (CATR). Patients will eventually seek medical care complaining of pain and functional deficits such as weakness at push-off and poor balance, which creates significant alterations in the gait pattern” (16). In neglected tendon ruptures, or those that become chronic, the tendon will heal, but often at an elongated length leading to long-term weakness (7). In these instances, the tendons also tend to develop a solid mass of scar tissue, making surgical correction a more difficult, and complicated, procedure.

Long term tendinopathy may be a lasting issue for these patients, and “several studies have suggested the prospect of stem cell therapies in the treatment of tendinopathy. (15)

“Recovery from midportion achilles tendinopathy is likely influenced by a combination of biological factors (tendon structure, BMI), motor function (lower extremity muscle strength and endurance), psychosocial factors (fear of movement), and severity of disability (VISA-A)” (2). Overall, ATR tends to demand a long recovery process. But early diagnosis and proper treatment will provide the patient with the best chance at minimizing effects on long term ADL performance, while also improving their ability to return to sports or return to work in a timelier manner.

Clinical Case Scenario

Patient Profile:

A 55-year-old male delivery driver presented to the emergency department following a low-speed motor vehicle collision in which his vehicle was struck from the rear while stopped at a traffic light. The patient reported forcefully pressing the brake pedal with his right foot at the moment of impact. He immediately experienced sudden pain in the posterior aspect of his right ankle, describing a sensation of “something snapping” followed by difficulty with walking. He denied loss of consciousness, direct trauma to the lower extremities, or other significant injuries. His medical history was significant for type II diabetes mellitus, obesity (BMI 34), and a sedentary lifestyle. He reported intermittent posterior ankle stiffness over the past year but denied prior Achilles tendon injury.

On examination, the patient ambulated with difficulty and was unable to fully bear weight on the right lower extremity. Inspection revealed moderate swelling and ecchymosis along the posterior ankle and distal calf. There was no open wound or deformity. Palpation revealed tenderness along

the Achilles tendon. Active plantarflexion strength was markedly decreased, and the patient was unable to perform a single-leg heel raise. Passive dorsiflexion was increased compared to the contralateral side. A positive Thompson test was noted. Distal sensation and pulses were intact, and no signs of neurovascular compromise were present.

Plain radiographs of the ankle were obtained to exclude associated fractures and were negative. Given the patient's comorbidities and to assist in treatment planning, a diagnostic ultrasound was performed. Ultrasound revealed a partial rupture of the Achilles tendon approximately 3 cm proximal to its calcaneal insertion.

The patient was placed in a posterior splint with the ankle in plantarflexion and instructed to remain non-weightbearing. Ice, elevation, and analgesics were initiated to manage pain and swelling. Non-operative management was pursued. The patient was transitioned into a VACOPed boot to maintain plantarflexion and was allowed weight-bearing as tolerated immediately in the boot. The ankle was brought to neutral over a 6-week period. Deep vein thrombosis prophylaxis was prescribed during the initial immobilization phase.

Physical therapy was initiated at two weeks post-injury, focusing on edema control, gentle active range of motion within allowed limits, and proximal lower extremity strengthening. At six weeks, progressive strengthening of the gastrocnemius-soleus complex was initiated, along with balance training and gait re-education. By ten weeks, the patient began controlled closed chain strengthening and endurance activities.

At three months post-injury, the patient demonstrated improved ankle range of motion and was ambulating independently in regular footwear with a slight limp. At six months, he reported mild weakness during prolonged walking and difficulty with stair negotiation but denied pain at rest. At the one-year follow-up, the patient demonstrated a stable, pain-free gait, with mild persistent calf weakness and an Achilles Tendon Total Rupture Score (ATRS) of 75. He had returned to full-time work with minor activity modifications.

The patient was counseled on the importance of adherence to rehabilitation protocols, continued calf strengthening, and gradual progression of activity to minimize the risk of re-rupture. He was educated on potential long-term complications, including tendon elongation, persistent weakness, and altered gait mechanics. Home modifications and supportive footwear were recommended to reduce fall risk and improve endurance during prolonged standing and walking.

Life Care Plan Considerations:

- 1) Medical Follow-up:
 - Orthopedic follow-up to monitor tendon and potential tendinopathy across the lifespan.

- 2) Modalities:
 - Physical therapy for flare-ups, focusing on ROM, pain control, and activity tolerance; 1 round, every 3-5 years.
- 3) Diagnostic:
 - X-rays to monitor potential development of bone abnormalities, every 3-5 years.
 - MRIs/CT scans to monitor tendons, cartilage and ligaments, every 3-5 years.
- 4) Pain Management:
 - NSAIDs or analgesics, to life expectancy.
- 5) Orthotics and Support:
 - Shoes with increased cushion support for stability and comfort during daily activities. Replace every 6 months - 1 year, to life expectancy.
 - Assistive devices: wheelchair, crutches, or walker to assist in maintaining weight bearing precautions throughout healing. Consider any additional assistive devices needed for possible increased pain in the tendon/joint as patient ages, such as a cane or walker.
- 6) Work and Functional Needs:
 - Adaptive task modifications at work to reduce prolonged load bearing during flare-ups.
- 7) Durable Medical Equipment with replacement until life expectancy:
 - See above sections.
- 8) Home Care Needs:
 - Consider adaptive equipment within the home based on individual needs.

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