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# Achilles Tendon and Athletes

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## Abstract

Achilles tendon (AT) is the strongest human tendon. AT disorders are common among athletes. AT pathologies vary from tendinopathy to frank rupture. Diagnosis is made clinically. Imaging modalities are used adjunctively. Management of AT rupture in athletes is challenging to surgeons due to worldwide growing popularity of sports and potential social and financial impact of AT injury to an athlete. Hence, new surgical techniques aim at attaining quick recovery with good outcome, finding similar results with both open and percutaneous techniques when accompanying these with functional rehabilitation protocols. Non-operative strategies include shoe wear modification, physiotherapy and extracorporeal shock wave therapy. Surgical interventions vary based on the AT pathology nature and extent. Direct repair can work for small-sized defects. V-Y gastrocnemius advancement could approximate the tendon edges for repair within 2–8 cm original gap. Gastrocnemius turndown can bridge tendon loss > 8 cm. Autogenous, allogeneous or synthetic tendon grafts were used for AT reconstruction purposes. In AT tendinopathies with no tendon tissue loss, surgical procedures revolve around induction of tissue repair through lesion incision or debridement to full detachment followed by reattachment. Extra-precautions are exercised for prevention of AT disorders especially among susceptible athletes participating in sports involving excessive AT strain.

**Keywords:** Achilles tendon, tendinopathy, AT, Achilles rupture, Achilles tendinosis, Haglund's exostosis, athletic injury, sports injury, percutaneous repair

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## 1. Introduction

Injuries to the Achilles tendon (AT) are usually related to sports, especially those that involve jumping, running and sudden accelerations, such as soccer, tennis or basketball [1]. A fraction

of patients with AT injuries tend not to present for medical care as they feel better following the event. On the other hand, healthcare workers fail to identify a quarter of AT injuries on initial presentation [2–6]. Yet, the precise pathogenesis of AT rupture remains unclear in a great deal of cases, partly because many of those patients never had prodromal symptoms [1, 7].

A patient with AT sport-related injury could present with a clinical picture ranging from mild inflammation to permanent injury [8]. These overuse injuries are increasingly reported; this is attributed not only to the rising number of individuals who participate in recreational sporting activities, but also to the greater intensity and duration of training in professional athletes.

In the following sections, reviews on common AT injuries among athletes are addressed namely: acute tears, chronic tears and different types of tendinopathies.

### 1.1. Epidemiology

AT injuries represent up to 50% of all injuries related to sports [1]. AT injury represents 20% of all tendon pathology in the lower limb [9]. It is interesting that AT is the most frequently injured tendon despite the fact of being the strongest and the thickest among human tendons [8, 10]. Sports-related AT conditions are commonly encountered. Runners are often affected with AT tendinopathy. AT overuse is believed to result in insertional tendinopathy [11, 12]. According to Cassel et al. report, 1.8% of adolescent athletes had AT tendinopathy [13]. Incidence of AT injuries is estimated between 10 and 20 per 100,000 population [14, 15]. Raikin et al. studied a group of patients with AT injuries stating that around a quarter of them were chronic cases [6]. Complete spontaneous AT ruptures have a strong association with sports activities [1]. It has been found that 60–75% of all AT ruptures are related to sports [16–18].

In the last decades, it has observed an increase of AT ruptures in the Western countries [1, 7, 19]. Although the epidemiology varies in the athletic population comparing to those who do not practice sports to a competitive level (the risk of sports act) [6]. About 8–20% of all AT ruptures in the general population are diagnosed in competitive athletes and 75% in recreational athletes [7]. Such injuries are common in individuals who are involved in athletic activities which implicate maximal exertion or explosive acceleration [1, 20, 21]. The incidence in each sport is shown differently depending on the country. For instance, basketball is the predominant sport in AT ruptures in the USA and football in Germany; badminton accounts for most in Denmark and Sweden and skiing is found more in Austrian and Swiss reports [7].

Different reports claim predominance AT ruptures in male population, with confusing data being published about this matter. It has been said there is a male predominance in AT ruptures with ratio men to women varying from 3 to 1 and 17 to 1 [7, 22]. However, some reports deny such a relation referring to AT ruptures in professional athletes [23, 24]. This is consistent with the dramatic increase of sports injury in females observed in the last decades, probably due the greater involvement of women in sports [25].

Additionally, it has been said that AT ruptures follow a bimodal distribution referring to age. Different studies describe different age range for the peak incidence, all coincide with the idea that AT ruptures are much more frequent in the aging athlete [1, 7, 21, 26]. Nevertheless, recent studies found no greater risk of developing tendinopathy or rupture in older athletes and aging has an uncertain meaning on tendon health [23, 24, 27].

There is a minor predominance in AT ruptures on the left limb and this could be explained by means of Hooker's hypothesis which states that the left limb is predominant in pushing off, with knee in extension that explaining the higher prevalence in this side among right-handed people [17, 28, 29].

It is been stated that runners show an incidence of AT tendinopathy between 6.5 and 18% representing the most common injury among them [30]. This is not only for the active young athletes, as former elite male runners have shown 52% risk of presenting AT tendinopathy during their entire life [31].

## 1.2. Biomechanics

Biomechanics related to injury of AT is a challenging subject. AT is a part of a complex myo-tendinous unit working across three joints, when it contracts; it flexes the knee, plantarflexes the ankle and supinates the subtalar joint. The subtalar joint becomes pronated during ambulation and hence forcing internal rotation to the tibia. This happens along with external tibial rotation imparted from an extended knee; AT is then submitted to this two contradictory forces together, causing a powerful stress [32].

AT functions as the major contributor to the plantarflexion of the foot during the gait cycle, contributing up to 93% to this movement [33]. It is subject to repetitive tensile stress and great loads in athletes. It has been estimated that when walking, AT tendon goes under a tension of 250% of the body weight, while the running load applies from 6 to 8 times the body weight, close to the maximum load tolerable by the tendon [26].

AT tendons have visco-elastic properties that allow the tendon itself to absorb energy during the stance phase of gait and to later release it when recoiling, contributing to an elastic movement. This simulates a spring function, especially important when running, as the time of ground contact decreases. The latter makes an important contribution of the tendon to the limb activity and helps to save muscular energy [34].

Tendons that stretch and recoil repeatedly, might ultimately suffer some variations. Due to its intrinsic properties and special material qualities, the AT becomes stiffer when put through rapid, forceful loads [35]. Different researches have investigated about this matter, stating that when applying a load to the entire lower limb modifies its stiffness trying to maintain the homeostasis in the athlete system [27, 36, 37]. Not only repetitive, long-term loading can cause a change in the tendon stiffness, but also single bouts of force applied to the AT tendon would be responsible for such adaptive changes [37]. These adjustments were thought to be reflected by means of increasing the cross-sectional area of the tendon [38, 39]. However, some researchers believe that in response to resistance training, the tendon cross-sectional area is not affected. They recognize the importance of changes in the material composition of the

tendon, which augments collagen synthesis and consequently, modifies the tendon properties and increases Young’s modulus and stiffness [37, 40, 41]. This is an adaption to repetitive forces which ultimately transforms the tissue composition and biomechanical behavior [37].

1.3. Risk factors and patho-etiology

Etiology of AT tendinopathy/rupture is a controversial dispute. Various hypotheses have been postulated in this context such as inflammatory, degenerative, infectious, drug-induced and neurological theories. Risk factors of AT tendinopathy/ruptures are summarized in **Table 1** [8, 42].

Certain drugs like fluoroquinolones and corticosteroids have been associated with higher potential of adverse effects on AT integrity [43]. Animal models have shown variable AT reactions in response to local steroid injections around the tendon compared to intra-substance infiltration [44, 45]. AT weakness with paratendinous injection was often reversible within 2 weeks during which strenuous activities should be avoided [46]. Clinically, AT ruptures have been reported with orally administered steroids [47, 48].

Excessive tendon hyperthermia, particularly during exercise, might lead to AT degenerative process resulting in tendinopathy or delayed disruption [42, 49].

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<i>Intrinsic factors:</i>
Aging (not fully proven)
Male gender (not fully proven)
High body mass index (BMI)
Tendon temperature
Systemic diseases
Muscle physiological and anatomical properties
Genetic predisposition
Blood supply
Malalignment: hindfoot hyperpronation, hindfoot varus
Leg length discrepancy
Stiff subtalar joint
Hindfoot hypermobility
Gastrocnemius-Soleus contracture
<i>Extrinsic factors:</i>
Use of drugs: for example, fluoroquinolones, steroids
Overuse: frequent micro-injury
Sport training errors
Sports shoes with AT impingement

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**Table 1.** Risk factors for AT tendinopathy/ruptures (Reprinted from: Alrashidi et al. Achilles tendon in sport. Sports Orthop Traumatol (2015) 31:282-292, Copyright (2018), with permission from Elsevier).

Traditionally, middle age group male subjects with irregular sports involvement were considered candidates for AT disorders [50, 51]. Neither age nor gender has been proven as a risk factor for AT pathologies [42, 52].

It has been observed that incidence of AT injuries increases dramatically with sport seasons and around 76% of AT injuries studied by Scott et al. were sport-related [52]. Hindfoot hypermobility as well as gastroc-soleus incompetence could be a factor in formation of AT tendinopathy among runners based on a biomechanical research study [53]. Malalignment of the hindfoot, particularly hyperpronation, is another identified contributing factor for AT tendinopathy [8].

Tendinopathy might be an element of what was described as “Haglund’s syndrome”. Repetitive contact between adjacent tissues at the AT/calcaneal attachment area could result in an abnormal mass formation “pump bump” and retrocalcaneal bursitis [54].

Histopathological studies demonstrated multiple forms of degeneration at the affected tendinous regions mainly hypoxic, mucoid, lipomatosis and calcification with predominance of hypoxic degenerative findings [55]. The classical ischemic degenerative hypothesis concerning pathogenesis of AT tendinopathy is not supported by robust scientific basis [56].

Individuals with metabolic conditions like diabetes, hypercholesterolemia, hyperuricemia and alkaptonuria could be more predisposed to have AT tendinopathy/rupture [57, 58].

It is advisable to exercise extra precautions for prevention of AT tendinopathy especially among susceptible athletes involved in AT-unfriendly sports such as running and soccer at the beach on the sand [8].

AT ruptures’ etiology is multifactorial, with participation of intrinsic and extrinsic factors, very important when referring to athletes [19, 59]. In sports, training errors may explain some of the injuries in the AT: too rapid increases or alterations in training routines neglecting recovery times, as well as soft training surfaces as track or sand or treadmill running, and unsuitable footwear can contribute to injuries in athletes [18, 26, 27, 33, 60, 61].

Other biomechanical alterations have been examined: forefoot varus seems to have a detrimental effect and hindfoot malalignment is believed to imply a rotational force into the tendon fibers [16, 53]. Many authors consider foot overpronation is related AT injuries although a recent review denies such an important effect [1, 9, 27, 62]. This is reinforced by biomechanical concepts in which, foot overpronation is accompanied by tibial internal rotation, a well know protective factor to AT injuries [27, 63]. Thus, foot pronation seems to display a moderate effect, and the correction should be taken cautiously given the contradictory result [27, 53]. Reduced stiffness during running has been related to AT injuries [37]. Low arch index is coupled with reduced stiffness in the lower limb, predisposing the athlete to suffer from them, while higher arches have a clear large beneficial effect [64, 65].

There are some theories which could explain the tendon degeneration prior to the rupture. Overuse tendon injuries have been described as those in which the tendon has been strained repeatedly, thereby generating cumulative microtrauma, until the tendon’s reparative ability is compromised, leading to injury [55]. Histopathologic studies on ruptured AT showed that



a high percentage (from 74% up to 97%) had clear definitory degenerative changes [66, 67]. This theory is also reinforced by different studies that support a poor blood supply due to the repetitive injuring mechanism might damage the tendon in the less vascularized areas—2–6 cm above the calcaneal insertion, precisely where the mid-portion AT ruptures most frequently occur [7, 66, 67].

There is also a mechanical theory in which, the dysfunction of the musculotendinous unit is claimed to be the main cause of rupture, causing an dis-coordinated or excess of muscle contraction that leads to rupture [7]. Three main types of indirect trauma have been described to cause an AT rupture: (i) pushing off while extending the knee, this occurs at the beginning of a sprint, running and jumping (53%); (ii) violent dorsiflexion of the ankle joint in a plantarflexed foot, as occurs when jumping or falling from a height and landing with the foot plantarflexed (10%) and (iii) sudden unexpected dorsiflexion of the ankle, when slipping on a ladder or stepping into a hole or in an unexpected fall (17%) [26, 68].

## 2. Acute Achilles rupture

### 2.1. Presentation

Patients with acute AT tear commonly present with sharp acute-onset pain at the posterior heel associated with forceful ankle push-off or sudden ankle dorsiflexion. An abnormal pop might be felt by the patient. Immediate swelling and walking inability are usually accompanying complaints [69, 70].

Two descriptions related to mechanism of acute AT rupture were reported. AT is subjected to extra rotational forces beyond its strength as the foot is forced into extreme pronation. The second explanation is the occurrence of an abrupt interruption of triceps surae eccentric contraction during support phase [71].

### 2.2. Diagnosis

Diagnosis of AT ruptures is quite straightforward if an appropriate patient history assessment and clinical examination are carried out [67]. However, up to 25% of acute AT ruptures are missed by practitioners [7].

Patients often describe an abrupt 'pop' in the AT area associated with the feeling of being 'kicked by someone'. They usually report pain that diminishes sometime after the injury and they remain unable to bear weight or to perform heel rises with the damaged limb [72]. Nevertheless, some of the patients use the extrinsic foot flexors showing remnant function of the ankle.

Regarding clinical examination, edema and bruising are found in most of the patients. A palpable gap may be present—usually 2–6 cm proximal to the insertion of the tendon (**Figure 1**). The diagnosis should be completed with other confirmatory tests, such as:

- The popular Simmond's and Thompson's tests: squeezing the calf to check failure of plantar flexion in AT ruptures [69, 73]. Nevertheless, partial AT ruptures can be missed with this maneuver. A cadaveric study showed that loss of more than 25% of AT tendon substance is required to be detected on Thompson's test [74].
- Matles test: shows a discrepancy of passive plantarflexion between healthy and affected limb [75].
- O'Brien test: an invasive test that uses a needle going all the way through the skin, to the substance of the proximal tip of the tendon. Plantarflexion of the foot will not produce any movement in the needle, diagnosing the rupture [76].
- Copeland test, measuring the elevation of pressure with a sphygmomanometer. The increase of pressure will be close to none in ruptured AT tendons when plantarflexion is forced [77].

Because diagnosis of AT ruptures is mainly clinical, imaging studies have little role in this aspect and should be reserved for uncertain diagnosis or differentiating between partial and complete tears [40, 78]. Diagnosis of acute AT rupture should be made on clinical basis. Relying on imaging diagnostics is questionable [79]. Plain radiography could visualize the soft tissue defect and associated avulsion fractures, if present [26]. Disruption of Kager triangle or presence of Toygar sign is suggestive of AT rupture [24, 80].

Ultrasonography (US) is noninvasive, rapid, repeatable and it allows practitioners to perform a dynamic study [40]. It is also used as part of the treatment follow-up and to measure the gap in between the tendon ends; and may give information about the risk of re-rupture preoperatively. Tendon defects appear as hypoechoic areas on ultrasound images [81, 82].



**Figure 1.** A clinical photo of an acute AT rupture. There is an obvious discontinuity of the tendon (this photo is courtesy of Fernández Torres, MD).



Magnetic resonance imaging (MRI) is a much more expensive technique and it does not allow a dynamic examination. However, it is much more reliable than US to diagnose any AT pathology—including partial ruptures, a very common injury in athletes, with higher sensitivity and specificity than US [7, 83]. Thus, it has been recommended to use MRI for the definitive diagnosis, especially when a partial rupture is suspected. However, it is been demonstrated that not only is MRI expensive, but also it is time consuming, with a mean of 5 days to obtain the images that could mean a delay on the treatment, a crucial factor in recovery for athletes [84]. Moreover, acute AT tears can be demonstrated as focal or linear defects particularly on MRI T2 weighted studies. Bony edema and the retrocalcaneal bursa effusion are characteristic for insertional AT ruptures which can be identified on MRI images [85].

Based on American Academy of Orthopedic Surgeons (AAOS) recommendations, at least two of the following clinical findings are required along with full medical history to establish a diagnosis of acute AT rupture: palpable gap, increased ankle dorsiflexion with gentle passive motion, weakness of ankle planter flexion and positive Thompson's (Simmonds's) test [79].

### 2.3. Treatment strategy

Non-operative treatment involving weeks of limb immobilization using a plaster or brace is known to have high re-rupture rate, which may lead to loss of considerable time off-athletic activity, which is probably not acceptable to athletic people [86]. Prolonged immobilization has been found to cause atrophy of calf muscles and relatively weak healing of tendon [87, 88]. Adding to the previous factors and high expectations of such patients, there has been a tendency to achieve optimal outcomes and lessening the risk of re-rupture through surgical repair [29, 87, 89]. However, certain conditions may make surgical options unfavorable such as diabetes mellitus, neuropathy, immunodeficiency, elder people (age above 65), smoking, sedentary lifestyle, high body mass index, peripheral vascular disorders or regional/systemic dermatologic diseases [79].

Acute AT ruptures with small defects within one-centimeter length usually heal adequately with immobilization in plantarflexion (**Figure 2**) [90, 91]. Conservative treatment course usually lasts for 8–10 weeks. The amount of plantarflexion is decreased gradually in a stabilizing boot to neutral position. A suggested program includes physiotherapy and serial adjustments from plantarflexion of 30° for 2 weeks adjusted to 15° for additional 2 weeks reaching plantigrade foot by the 5th week. Conservative method has increased risk of AT re-rupture and atrophy of calf muscles [22, 92–95]. Non-operative strategy requires rigorous follow-up and skilled orthopedic surgeon [96, 97]. Adjunctive use of platelet-rich plasma (PRP) in acute Achilles rupture is not yet proven [98].

Management of AT rupture in athletes is challenging to the surgeon owing to the high importance of sports worldwide and possible social and financial consequences of an injury to both the player and the team. Thus, new surgical techniques aim at attaining quick recovery with good outcome [86, 89].

Surgical options include open, mini-open and percutaneous techniques [99]. Open repair has shown good outcome postoperatively, but carries high risk of wound complications [24, 86, 95, 100]. Open repair of acute AT rupture is considered the standard surgical intervention.



**Figure 2.** A cast, which is applied in equinus as part of conservative treatment of acute AT rupture (this photo is courtesy of Fernández Torres, MD).



**Figure 3.** An illustration of a patient with acute AT rupture showing hematoma and degeneration of the tendon fibers (this photo is courtesy of Fernández Torres, MD).

Many techniques and modifications have been described in this setting [24, 101–103]. Open method offers full exploration of the injured tissues, adequate debridement, good assessment of tendinous defect and reliable repair strength (**Figure 3**).

Percutaneous techniques have demonstrated an improved outcome since its introduction [104]. Some studies recommend to do percutaneous repair in athletes rather than open [89]. Percutaneous techniques are advantageous in decreasing soft tissue damage, which consequently may improve time to recovery and rehabilitation [24, 89]. Adding to that, some studies demonstrated better outcome of percutaneous repair in acute AT ruptures in terms of less expected infection, adhesions, deep venous thrombosis and have less costs and quicker recovery period [31]. Other studies showed no difference regarding results of both techniques [99, 105]. In contrary to open repair, percutaneous repair has a comparable re-rupture rate [95, 99, 106]. On the other hand, sural nerve injury and inability to address the torn soleus component are

possible drawbacks of percutaneous methods. Hence, some surgeons do not prefer such a method as it is believed that soleus contributes significantly to the overall AT strength with no less than 40–52% [8, 10, 22, 99].

In professional athletes with acute AT rupture, some surgeons prefer to do mini-open approach to reduce skin complications and allow for faster recovery. Postoperative care program consists of 8–10 weeks of immobilization in an adjustable boot with intermittent physiotherapy. Functional rehabilitation protocols have been established to achieve a fast and successful recovery, showing a reduction of complications associated with cast immobilization without increasing the re-rupture rate. These protocols vary depending on the chosen surgical technique, being the percutaneous surgeries the ones who allow the patient do prompt mobilization and weightbearing [95, 99, 107–109]. Initially, the foot is kept plantarflexed then gradually stretched to neutral position [8].

Time to return to sports ranges from 4 to 6 months based on the sport type. Sanchez et al. claimed that addition of PRP injection along with acute surgical repair can shorten the time to return to sports [110]. However, a randomized controlled trial has shown no significant acceleration in healing of acutely repaired AT [111].

Regardless to treatment approach, resumption of pre-injury normal walking could happen within 12 weeks [112, 113]. Involvement in a functional rehabilitative program remarkably shortens this time to 8 weeks [113, 114]. At least, 4–6 months are required to return to sports. Contact sports need longer periods [8].

Outcomes of treatment of acute AT can be assessed with clinical examination particularly in unilateral cases where the normal side is available for comparison [115]. No significant consistency was appreciated between the clinical scoring systems and biomechanical studies outcomes in treated ruptured AT cases. It seems that trauma to the AT inherently lowers its biomechanical properties to a certain extent. Specific peculiarities were observed during gait analysis in injured AT kinematics. Excessive eversion and diminished peak planter flexion torque (PPFT) at stance phase were noticed in this group of patients [71, 116]. Maximum calf circumference (MCC) correlated well with PPFT and push-off force (POFF) [116]. AT total rupture score (ATRS) is a validated scoring system which provides a reliable instrument to evaluate torn AT post-surgical repair [117, 118].

Heel rise height can be used as an indicator of postoperative functional performance. Heel rise height tends to get less as age increases. Young men scored higher on 12-week-evaluation in terms of functional outcomes. Obese individuals were more symptomatic. Both surgical and non-surgical treatment provided no clue about the final functional outcome. Type of treatment could predict moderately the intensity of subsequent symptoms [119, 120].

### **3. Chronic Achilles rupture**

#### **3.1. Clinical presentation and diagnosis**

Up to authors' knowledge, there is not yet a consensus on the time-limit after which AT injury could be accurately described as chronic. Generally, AT rupture can be labeled as chronic after

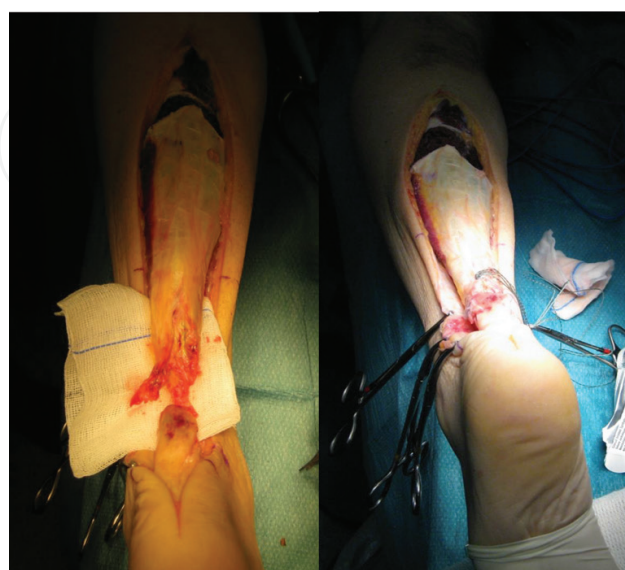
4–6 weeks have passed after the injury [2, 6, 8, 85]. Moreover, cases which did not show healing signs or presented after 4 weeks of initial injury is thought to be “chronic” irrespective of rupture etiology [3, 51].

Chronic AT ruptures can present with pain, gait changes, calf muscle wasting and impaired push-up. Adding to that, dramatic effect of physical or athletic activities such as walking, jumping or using of stairs becomes obvious [2]. In some occasions, the rupture defect may be filled with a scar tissue [2, 3, 50]. Such a scar is often not of the same physiological properties (e.g. elasticity and excursion) to substitute a normal AT tissue and subsequently cause a noticeable effect on gait [121].

Chronic AT tears are visualized as low-signal lesions on MRI images. Furthermore, amount of tissue loss can be measured. Postoperative evaluation of repaired or transferred tissues can be obtained through MRI [85].

### 3.2. Treatment strategy

Non-operative treatment of chronic AT ruptures commonly ends with unsatisfactory outcomes. Consequently, most surgeons prefer surgical treatment approach for cases of chronic AT tears [122]. Chronic AT ruptures with unremitting pain, instability or functional limitations in terms of daily activities or sports performance are considered reasonable indications for surgical reconstruction [121]. Defects of less than 3 cm can be amenable to end-to-end anastomosis. Gastrocnemius-soleus V-Y advancement is useful to manage gap within 2–6 cm in length (**Figure 4**). Gastrocnemius turndown procedure is preferred to address Achilles defects of greater than 6 cm. Plantaris tendon can be employed to augment the repair area [8, 122]. Anterior paratenon harbors the key blood supply to the repair area and should be protected [51].



**Figure 4.** A clinical photo demonstrating V and Y technique for treatment of chronic AT rupture (this photo is courtesy of Fernández Torres, MD).



Chronic AT ruptures are categorized into 3 types according to Myerson: type I includes defects under 2 cm; type II which has a defect ranging from 2 to 5 cm; and type III defect which exceeds 5 cm in size [85].

Tendons of adjacent muscles can be incorporated within AT reconstruction avoiding free graft complications. Sadek et al. reported good satisfaction among 18 patients with Myerson type III AT defects following local reconstruction with triple loop of plantaris tendon along with turndown flap [85]. Instead of removing scar tissue filling the tendon defect, Khaimi et al. advocated including tubular scar tissue within AT reconstruction procedure. Khaimi and colleagues performed shortening Z-plasty of the fibrotic tissue across the defect and augmented that with free sural triceps graft [121]. Besse et al. reported satisfactory results in six subjects with long-standing terminal ruptures of AT using bone-tendon autograft obtained from knee extensor mechanism [123].

For the sake of having more reliable repair, Esenyel et al. attempted adding mesh (Hyalonect) to the gastrocnemius turndown flap in 10 patients. Those patients scored significantly higher postoperatively on AOFAS (American Foot and Ankle Society) score [124]. Similarly, Ibrahim et al. reported good outcomes combining surgical repair with a synthetic polyester graft in 14 chronic AT ruptures [3].

Allografts and synthetic grafts could reduce the operative time by eliminating the harvest time. Avoidance of donor site complications is an advantage of using allografts. Moreover, allografts are relatively biologically active. Allograft-related disadvantages include risk of disease transmission and graft-versus-host disease [2]. Nellas et al. indicated reasonable results following AT reconstruction with freeze-dried allogenic AT grafts [51]. AT reconstruction using polypropylene mesh (Marlex) was reported by Choksey et al. in five cases with promising outcome [125].

Idealization of tension across the repair site of AT is of paramount importance. Weak push-off is predicted with very lax repaired AT. Equinus deformity might be due to overtightening of AT repair. Knupp and Hintermann emphasized using the contralateral side as a control to optimize the desired amount of tension [122].

Paavola et al. followed up 432 patients with surgical AT procedures for 1 year looking for complications. There were 46 patients suffered from complications which required re-operation in 11 of them. The main complications identified were superficial infection, transient sural nerve palsy, incomplete re-rupture and thromboembolism [126].

#### **4. Achilles tendinopathy**

AT tendinosis is a term that includes a series of different degenerative processes without clinical or anatomopathological signs of inflammation [25, 127]. It is been suggested that tendinosis is the ultimate consequence of repetitive stress applied to the tendon, that is unable to create a homeostasis between synthesis and degeneration of the cell matrix [128].

Grossly, AT tendinopathy can be classified as insertional (pain at the insertion of the tendon to the calcaneus) and non-insertional (lesion found in mid-portion of the tendon).



#### 4.1. Presentation

A detailed medical history should be elaborated including data about the timing and nature of injury, any history of infection, use of steroids, sport shoe type of usual use and any previous orthopedic interventions for the same injury [8].

In 1998, Maffulli et al. has described AT tendinopathy as heel pain and swelling associated with decreased performance of the tendon [127]. An exostosis may be felt at the postero-superior aspect of calcaneus which was called “Haglund’s deformity”. Mechanical irritation from such a prominence may lead to retrocalcaneal bursitis [54]. In 2011, new terms have been proposed by van Dijk et al. for better guidance of diagnosis and treatment of different AT disorders. Such disorders are summarized in **Table 2** along with their anatomical location and clinical manifestations [129].

#### 4.2. Diagnosis

Clinical examination should aim at identifying any systemic or local risk factors of AT tendinopathy (**Table 1**) [8]. The site of pain is localized, whether insertional (within 2 cm from insertion) or mid-portion (within 2–7 cm above the insertion) tendinopathy; any palpable gap, swelling, crepitus or nodules [129–131].

Calcifications of AT in addition to bony spur (Haglund’s deformity) can be appreciated from plain radiographs. Some radiographic parameters have been suggested to diagnose bony spur such as Fowler’s angle [132, 133].

Disorder	Anatomical location	Manifestations	Clinical signs
Mid-portion tendinopathy	2–7 cm from AT insertion	Pain, swelling and impaired performance	Diffuse or localized swelling
Acute paratendinopathy	Around mid-portion of AT	Edema and hyperemia	Palpable crepitations and swelling
Chronic paratendinopathy	Around mid-portion of AT	Exercise-induced pain	Crepitations and swelling less pronounced
Insertional tendinopathy	Within 2 cm of insertion onto calcaneus	Pain, stiffness, sometimes a (solid) swelling	Tenderness of AT insertion at mid-portion of posterior aspect of calcaneus. Swelling may be seen and a palpable bony spur may be found
Retrocalcaneal bursitis	Retrocalcaneal recess	Painful swelling superior to calcaneus	Painful soft tissue swelling, medial and lateral to AT at level of posterior superior calcaneus
Superficial calcaneal bursitis	Bursa between calcaneal prominence or AT and skin	Visible, painful, solid swelling postero-lateral calcaneus (often associated with shoes with rigid posterior portion)	Visible, painful, solid swelling and discoloration of skin. Most often located at postero-lateral calcaneus; sometimes posterior or posteromedial

**Table 2.** Achilles tendon disorders and their anatomical location, symptoms and signs (adapted from van Dijk et al. [129]).

AT disorder	Plain radiography	US	MRI
Mid-portion tendinopathy	Deviation of soft tissue contour is usually present.  In rare cases calcifications can be found	Tendon larger than normal in both cross-sectional area and antero-posterior diameter.  Hypoechoic areas within the tendon, disruption of fibrillar pattern, increase in tendon vascularity (Echo-Doppler) mainly in ventral peritendinous area	Fat-saturated T1 or T2 images: fusiform expansion, central enhancement consistent with intra-tendinous neovascularization
Acute paratendinopathy	—	A normal Achilles tendon with circumferential hypoechoic halo	Peripheral enhancement on fat-saturated T1 or on T2 images
Chronic paratendinopathy	—	A thickened hypoechoic paratenon with poorly defined borders may show as a sign of peritendinous adhesions; increase in tendon vascularity (Echo-Doppler) mainly in ventral peritendinous area	
Insertional tendinopathy	May show ossification or a bone spur at the tendon's insertion; possibly deviation of soft tissue contours	Calcaneal bony abnormalities	Bone formation and/or on STIR (short tau inversion recovery) hyperintense signal at tendon insertion
Retrocalcaneal bursitis	A postero-superior calcaneal prominence can be identified; radio-opacity of the retrocalcaneal recess; possibly deviation of soft tissue contours	Fluid in the retrocalcaneal area/bursa (hyperechoic)	Hyperintense signal in retrocalcaneal recess on T2 weighed images
Superficial calcaneal bursitis	Possibly deviation of soft tissue contours	Fluid between skin and Achilles tendon	Hyperintense signal between Achilles tendon and subcutaneous tissue on T2 weighed images

**Table 3.** Radiologic signs of AT disorders (adapted from van Dijk et al. [129]).

MRI and US can help in diagnosis of different AT disorders. The radiographic signs of AT disorders are summarized in **Table 3** [129, 134]. In case of suspected underlying metabolic disease, laboratory studies should be considered to predict any healing problems associated with those diseases and further treatment, if indicated [58].

**4.3. Treatment strategy**

Initial treatment regimen of AT tendinopathies includes a course of AT eccentric exercises and/or extracorporeal shockwave therapy (ESWT). Surgery is considered if no significant response to non-operative treatment [135–137].

Rompe et al., in RCT, found a comparable outcome of AT eccentric exercises and low shock-wave therapy at 4th month of outpatient visit [135]. Another high-level study did not show any superiority of heavy slow resistance exercises or eccentric exercises over another in cases of mid-portion tendinopathy, but slow resistance exercises showed a higher patients' satisfaction after 12th week of outpatient follow-up [138].

A recent systematic review has suggested that low-energy ESWT is successful in reducing manifestations of both insertional and mid-portion AT tendinopathies if used over a period minimum of 3 months. Better results are expected if AT eccentric exercises are performed during ESWT treatment period [139]. Another study, an RCT, suggested that physiotherapy and the use of custom made insoles for period of 4 weeks showed a significant alleviation of pain among athletes who were diagnosed as chronic AT tendinopathy, without modification of their athletic activity during treatment period [140]. Modification of shoe wear (e.g. Rocker shoe) and using shock-absorbing insoles may help in prevention of AT tendinopathy [141, 142].

In RCT, by de Vos and colleagues, PRP injection as an additional modality to exercises did not show any significant effect on subjects suffering from mid-portion AT tendinopathy in contrast to patients managed by exercises and placebo [143]. It is claimed that concentrates of platelets were found to have in vivo potential to enhance creating the granulation tissue and helping in repair of AT tendon defects. The latter process was not shown to be applicable in AT tendinopathy [144]. Another double blinded study by de Vos et al. involved 54 patients with chronic AT mid-portion tendinopathy and followed for 24 weeks. They found no significant difference between the PRP group (PRP injection and eccentric exercises) and the placebo group (placebo injection and eccentric exercises) in terms of alteration in tendon ultrasonic picture or vascularity [145].

Operative treatment is indicated in patients who are not responsive to conservative protocols (3–6 months). Generally, surgical option is selected according to the clinical and radiological signs of individual cases. Insertional AT tendinopathy can be treated by open or endoscopic techniques which may include removal of retrocalcaneal bursa, tendon debridement, detachment and reattachment of tendon, intra-tendinous bone excision and/or removal of postero-superior calcaneal prominence [8]. Radiological finding of postero-superior calcaneal prominence (Haglund's exostosis) is not an indication per se for operative treatment and may not explain the reason behind patient's manifestations [54].

Non-insertional AT tendinosis can be addressed via different surgical options. All of them try to remove the abnormal tissue on the tendon itself and the paratenon and promote the healing process through origination of new viable tissue and vascularization [146]. These options include percutaneous tenotomy, tendon stripping through MIS and endoscopic and open tendon debridement with or without augmentation techniques [147–150].

## Conflict of interest

All authors declare that there is no conflict of interest related to this manuscript.

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