Outcome of Percutaneous repair in Management of Achilles Tendon Ruptures

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Abstract

Background: Controversy exists regarding the optimal treatment for acute Achilles tendon ruptures. Conservative and surgical treatments have been reported with variable results and complications rates.

Objective: The aim of study is to assess the **Outcome of Percutaneous repair** in treatment of achilles tendon rupture.

Conclusion: Better outcomes are obtained by using percutaneous sutures technique in the treatment of achilles tendon ruptures.

Keywords: Achilles tendon rupture, Percutaneous repair

DOI Number: 10.14704/nq.2022.20.8.NQ44321 NeuroQuantology 2022; 20(8):2902:2910

Introduction

The Achilles tendon is the largest and strongest tendon in the human body. However, its rupture is common in middle-aged active men, especially athletes, with a male to female ratio of 4.8:1 [(1)].

Causes and etiology

The etiology and causes behind the AT rupture are not fully clear and there is no consensus in the literature. There are various factors affecting the tendon that may increase rupture risk such as poor vascularity of the tendon, gastrocnemius-soleus dysfunction, age, gender, previous injury and even changing of training pattern[(1)].

Some pathological conditions are associated with AT rupture as autoimmune diseases, arteriosclerosis, diseases associated with collagen abnormalities, infectious disease and neurological condition [(2)].

Two main theories were proposed to explain AT rupture the "degenerative theory" and the "mechanical theory". The degenerative theory is based on the degenerative changes that were noticed in the patients during the open repair which was done on day of injury, which mean that these changes were developed before the rupture. These abnormalities are not explained in literature, but probably it's due to the changing of the blood supply, hypoxia and the impairment metabolism [(3)].

Thedegenerative changes is usually found in people above 35 years old may be associated with accumulating of microtrauma and as it was demonstrated before that tenocytes regenerated from a ruptured tendon produces a collagen type III which disturb the tendon architecture makes it more susceptible to complete or spontaneous rupture as it becomes less resistant to tensile forces [(4)].

The mechanical theory explains the rupture of a healthy tendon as **Barferd**[(5)] demonstrated that an AT rupture may occur when is the tendon is obliquely extra loaded at a short initial length with muscle maximum contraction. These movements are present in almost all sports that requires rapid push-off.

Inglis and Sculco[(6)] proposed that the malfunction of the inhibitory mechanism which prevents the excessive muscle contractions that can cause rupture of a normal tendon. This represents the high risk of AT rupture in athletes who return to sport practice after a period of inactivity. The risk increases if an oblique stress applied to the tendon for example during the ankle eversion and inversion which commonly happens in sports and training errors.

Drug related tendon rupture

Most evidences support that steroids and fluoroquinolones are related to AT rupture. Both drugs resulting in collagen dysplasia which weakens the tensile strength of the tendon leading to increase the risk of tendon damage. The systemic and local corticosteroids participate in AT rupture. Available evidences recommends not to use intratendineous or peritendineous injections because it masks the symptoms of the damage tendon [(7)].

Corticosteroids decreases the tendon healing resulting in weakening of the tendon up to 14 days post injection. This weakening was found to be directly related to collagen necrosis and restoration of the tendon strength. That's why activity after corticosteroids injection should be avoided for at least two weeks giving the tendon time to heal and restore its strength[(8)].

Fluoroquinolones administration causes disruption of the extracellular matrix of the cartilage and chondrocytes necrosis leading to depletion of collagen which is associated with tendon rupture, as **Szarfman et al.** reported [(9)]. Other studies reported the bad effect of fluoroquinolones on the tenocytes[(10)].

Mechanism of injury

There are three main mechanisms of injury reported in literature [(11)]:

1- Weightbearing forefoot pushing off with an extended knee; this movement is commonly seen in starting sprint sports and jumping sport such as basketball.
2- Violent Sudden dorsiflexion of the ankle such as falling into a hole or falling downstairs.

Diagnosis and presentation

AT rupture presents with sudden pain and swelling in the affected leg at the time of injury,

*e*ISSN 1303-5150

some patient report and audible snap. Usually patients are unable to bear weight and feel weakness of the affected ankle. However, they may be able to plantarflex the ankle using the flexor hallucis longus, flexor digitorum longus, tibialis posterior and peroneal tendons. Diagnosis of AT rupture seems to be easy and straightforward diagnosis, but 20%-25% of patients with AT rupture are missed during their first examination [(12)].

Clinical examinations are crucial in diagnosis of AT rupture [(13)]. Tests and signs should be performed to confirm the diagnosis:

Simmonds or Thompson calf squeeze test

Patient should be in a prone position, the examiner squeeze on the belly part of the calf muscle. In case of an intact AT the squeezing will cause a plantar flexion. The affected leg should always be compared to the normal side. A false positive test may occur in case of an intact plantaris tendon [(1)].

Matles Test

In this test patient asked to lie in prone position and actively flex his both knees to a 90 degree. If the forefoot is noticed to fall into a neutral or dorsiflexion position during this movement, the diagnosis of AT rupture may be established [(14)].

Figure (1): Matles test. Prone on the examination couch, patients are asked to actively flex their knees to 90 degree. During this movement, if the foot on the affected side falls into neutral or dorsiflexion, an Achilles tendon rupture can be diagnosed **[(15)].**

X-ray

X-rays to analyse the point of injury. This is not very effective at identifying injuries to soft tissue. X-rays are created when high energy electrons hit a metal source. X-ray images are acquired by utilising the different attenuation characteristics of dense (e.g. calcium in bone) and less dense (e.g. muscle) tissues when these rays pass through tissue and are captured on film. X-rays are generally exposed to optimisevisualisation of dense objects such as bone while soft tissue remains relatively undifferentiated in the background. Radiography has little role in assessment of Achilles' tendon injury and is more useful for ruling out other injuries such as calcaneal fractures [(16)].

Ultrasonography

Ultrasonography is an operator dependent. However, it's still a primary imaging method because it's not invasive. A normal AT appears on ultrasound as a hypoechogenic, ribbonlike image. A rupture AT is seen on the scan as an acoustic vacuum with thick irregular edges. The ultrasound could be also used for AT repair assessment [(16)].

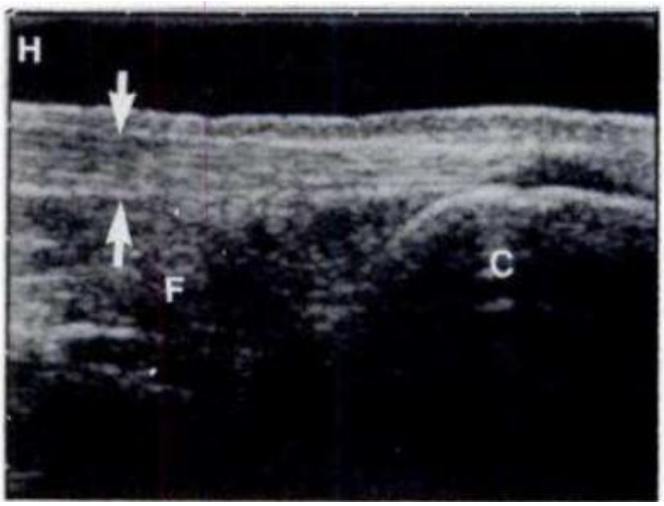

Figure (2): Normal Achilles tendon. Longitudinal scan shows the fibrillar architecture of the tendon with two slightly more echogenic bordering line (arrows). Note the hypoechoic distal insertion onto the calcaneus, whose posterior aspect is seen as a dense echogenic curved line. C=calcaneus, F=pretendinous fatty triangle, H=toward patient's head [(16)].

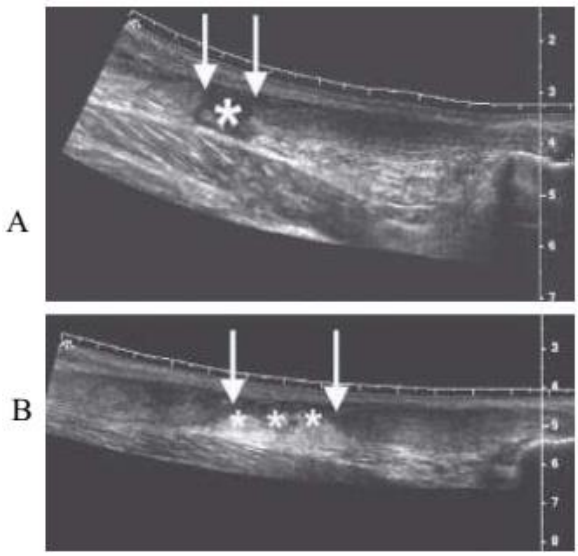

Figure (3): (A)Longitudinal extended field of view ultrasonographic image of a complete tendon tear showing the gap in the tendon (*) and the torn tendon ends (solid arrows). The muscle belly of flexor hallucis longus is well seen deep to the tear. (B) Longitudinal extended field of view ultrasonographic image of a complete tendon tear. Note more retraction, compared to case A, with a larger gap in the tendon, torn tendon ends (solid arrows), and echogenic (bright) fat herniating into the tendon gap (*) [17].

Magnetic Resonance Imaging (MRI)

In the MRI the tendon is seen well delineated by the high the signal intensity of the fat pad of Karger's triangle. If any increased intensity of the intratendinous signal should be considered as abnormality. The sagittal and axial planes are used to evaluate the AT rupture, as it appears as a disruption of the tendon signal [18].

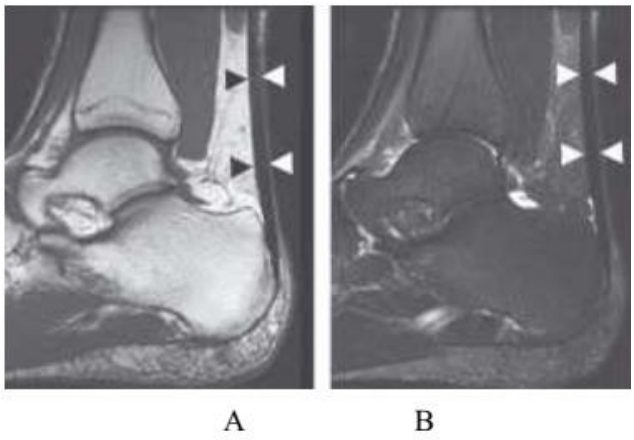

Figure (4): Sagittal T1 (A) and T2 with fat saturation (B) images, of a normal Achilles tendon. Note the parallel anterior and posterior tendon surfaces (arrowheads) and its low signal (black) appearance on both sequences [18].

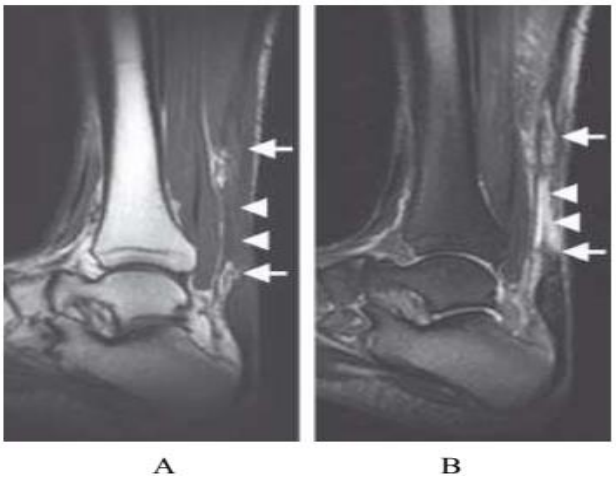

Figure (5): Sagittal T1 (A) and sagittal T2 with fat saturation (B) weighted images in a patient with an Achilles tendon rupture. Note discontinuity of fibers, high signal within the tendon gap (arrowheads), and the torn tendon ends (solid arrows) [18].

Management of AT rupture

The treatment of acute AT rupture has no agreed protocol based on high evidence, and it has been always based on surgeon's preference. The management can be classified into two main groups:

A) Operative treatment
B) Nonoperative treatment

The operative is sub-grouped into open repair or percutaneous repair. The nonoperative is sub-grouped into immobilization in cast for 8 weeks or functional ankle brace [19].

The treatment of acute AT rupture has changed with time as more evidence that supported the operative percutaneous treatment over the open repair and the early mobilization over the prolonged immobilization [20].

The nonoperative treatment was preferred back in 1970s. For the last two decades the operative treatment the treatment of choice in athletics and young active patient, while the nonoperative method of treatment was reserved for nonathletic old patient [21].

Studies that have been conducted shown that the open repair of acute AT rupture significantly

reduces the risk of re-rupture compared with nonoperative treatment, as it allows end to end healing and more secure application of early ofmotion. However, the open repair has high risk of skin complication and wound healing 44. The reviews reported that the open repair wound complications maybe reduced by performing percutaneous AT repair [22].

The treatment goal of acute AT rupture is to have best functional outcomes and achieve patient satisfaction and to minimize the morbidity of the injury and prevent the complication. The modern study for evaluation of AT rupture outcomes are the complications of the treatment itself, calf muscle atrophy, wound complications, endurance, sport participation and patient satisfaction [23].

The major limitation for these studies is absence of a global outcomes scores that adequately assess all the necessary outcomes and can be used for all the studies.

Surgical treatment

Percutaneous repair

In **1977, Ma and Griffith** [24] was the first to develop a new percutaneous technique as an alternative for the open repair and the cast immobilization. The technique based on making six small stab incisions along the medial and the lateral borders of the AT, then passing a suture through the tendon using these incisions. They reported 18 patients treated by this technique and were only two minor noninfectious skin complications and no re-ruptures.

FitzGibbons et al. [25] reported good results in 14 patients who had percutaneous repair by same technique, only one patient had sural nerve injury. 38 patients with AT tendon rupture were treated with Ma and Griffith technique in **Klein et al.** [26] study and reported a 13% had sural nerve injury.

In a study done by **Rouvillain**[27] reported a series of 60 patients with AT rupture underwent a percutaneous repair by Ma and Griffith technique without any sural nerve injury and two cases with re-rupture at two and five months of follow up and good functional outcomes.

Webb and Bannister [28] described a technique for the percutaneous repair which carried out under local anesthesia where a three midline transverse 2.5 cm incisions over the posterior aspect of the AT tendon is done. They reported no sural nerve injury either late re-rupture in a 27 patient. They return to work at four weeks and sports activities at four months.

Later **McClelland and Maffulli,** [29] described a modification for **Webb and Bannsiter**technique by using these three small transverse incisions. The first incision is made directly over the palpable defect. The other two incisions are made four cm distal and proximal to the first incision. The most proximal incision is made to medial side of the tendon to minimize the risk of sural nerve injury – the sural nerve crosses the lateral border of the AT 10 cm to its calcaneal insertion- then a four-strand repair is done.

Minimally invasive technique

Assal et al. described a new minimally invasive technique using the AchillonR system for AT repair that reduce the risk of sural nerve injury and minimize the adhesion and wound complication [30].

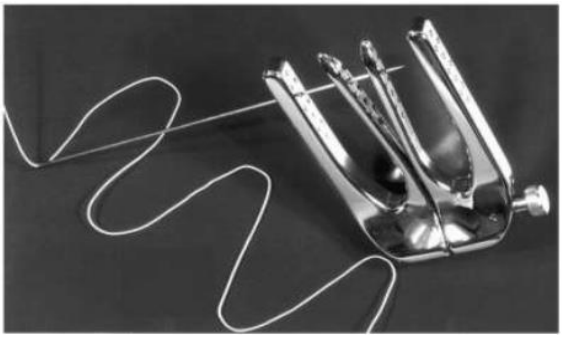

Figure (6): The guiding instrument, with a straight needle and suture passed through one of the levels of holes [30].

The Achilon jig figure (6) (Integra Lifescience Corporation, USA) has been developed from the minimally invasive technique; a transtendinous sutures can be placed through the targeting holes in the jig across the skin, fascia, paratenon and the proximal part of the ruptured then the jig is pulled from the proximal part and the sutures remains under the paratenon, as the jig is inserted between the tendon and the paratenon. There is an obvious chance to injure the nerve when the needle is inserted through the jig but as the suture is removed from the same puncture site, so it reduces the risk of permanent nerve symptoms [30].

A study on 25 patients with AT tendon rupture used the Achillon jig with early active rehabilitation reported return back to sport activity after five months with no wound complications and either sural nerve injury or re-rupture [31].

Ismail et al. [32] reported on the Achillon minimally invasive technique comparing the biomechanical properties of the tendon suture done by Achillon and Kessler method and concluded a close tensile strength results of both.

McWilliam and Mackay [33] in 2016 described a new technique the InterBrace (IB) using the PARS and Two 4.75- mm anchors. Two longitudinal incisions are made on the posterior aspect of the heel, approximately 1.5 to 2 cm apart, just below the midline of the convex surface of the posterior calcaneus. A 3.4-mm drill hole is made in each incision with the drill angled distally and toward the midline. During anchor insertion, distal tension is placed onto the sutures such that the amount of ankle equinus of the injured limb is equivalent to the uninjured limb to prevent the over-tension. This IB technique provides a knotless approach.

Open repair

Open surgical repair was the treatment of the choice for decades in young fit individuals with acute AT rupture and range from simple end to end suturing, with Bunnell or Kesslertype sutures, to more complex repair with the use of fascial reinforcement or tendon graft [34]. The new postoperative rehabilitation protocols and good functional outcomes with the early rehabilitation have encourages many surgeons to favor the open repair [35].

Furthermore surgical repair decreases the rerupture rate to 1% and less calf muscle atrophy, also helps a higher number of athletes to return back to their pre-injured level of sport activity faster [35].

The open repair can be performed by various of sutures types. Krackow suture type is the most commonly used due to its biomechanical tensile strength and less suture burden [36].

Elevation of the paratenon then repairing it is important to decrease the adhesion around the tendon [37].

Several authors have opposed the open repair because its main disadvantage the high complications rate. They reported, in a series of 86 patients underwent operative repair, reported 24% of complications; two patients had deep vein thrombosis which one of them resulted in pulmonary embolism and death; three patients had wound infection; 11 patients suffered from wound necrosis and four cases of rerupture[1].

Soldatis et al. [38]**,** in study of 23 patients who had open repair reported only two case with delayed wound healing. The explanation behind that maybe referred to a greater operative experience combined with techniques improvement.

Primary augmentation for acute AT ruptures has no evidence to have better outcomes than the nonaugmented repair. The augmentation is mentioned only in the recurrent ruptures. In 2007**, Aktas et al.** [39] reported no significant functional difference between the end-to-end

repair technique by Krakow sutures only and augmentation with plantaris tendon. More randomized controlled trails reported augmented repair of a fresh total Achilles tendon rupture does not have any advantage over simple end-to-end repair [(40)].

Complications of an Achilles tendon injury

Complications of an Achilles tendon injury may includePain, which can be severe, difficulty walking or being active, warping of tendon area or heel bone, tendon rupture from re-injury, other complications can happen because of the treatments used to treat an Achilles tendon injury. For instance: sometimes, cortisone injections can cause the tendon to tear. Surgery can lead to pain and infection [(41)].

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*e*ISSN 1303-5150