Achilles Tendon: Tendinopathy and Ruptures

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Keywords
Achilles tendinosis • Achilles tendon rupture • Conservative treatment • Surgical treatment

Introduction

The Achilles tendon (AT) is known to be the largest and strongest tendon of the human body; however, it is also the most frequently ruptured tendon with increasing incidence in the last decade [1, 2]. Investigators have seen that in men, the peak incidence occurs at the age of 30–39 years of age, while in women the incidence increases as one reaches the age of 60 years and above [3, 4]. Men are more common to rupture their AT with a male/female ratio of between 1.7:1 and 30:1 [5]. Sports activities are the most common cause of AT rupture (44–83 %) especially in athletes, but some biomechanical and biochemical changes seen in aging may also be a cause for the AT to be torn specially in sedentary individuals who occasionally play sports or even during normal activities [3, 6–8].

Anatomy

The AT is formed by the aponeurosis of the soleus muscle and the two heads of the gastrocnemius muscle at the lower ends of their muscle bellies [9, 10]. The AT functions as a plantar flexor of the ankle, but due to its insertion to the calcaneal tuberosity, which is slightly medial to the midline, it was found out that the AT can also produce an equinus and inversion force [9]. Other structures found near the AT are the plantaris muscle which is usually present in 93 % of lower extremities [10], a retrocalcaneal bursa which is situated between the AT and the calcaneus, and a subcutaneous bursa which is located between the tendon and the skin [11]. The sensory supply to the AT originates from the sural nerve through the attaching muscles and cutaneous nerves [12]. The AT has no true tendon sheath but has a paratenon which surrounds the whole tendon. This paratenon is composed of a superficial layer which is called the peritenon and a deeper layer

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which is in direct contact with the epitenon through the mesotenon [13]. In between the paratenon and the epitenon is a space filled with a thin, lubricating film of fluid which allows gliding of the tendon during motion.

The blood supply to the AT originates from different sources. On the proximal part of the AT, the musculotendinous junction provides most of the blood supply, while the surrounding connective tissue and the bone-tendon junction provide blood supply to the whole length and to the distal part of the AT, respectively [14]. Research has stated that at approximately 2–6 cm proximal to the insertion is an area of poor vascularity which may play an important role in the pathogenesis of its rupture [14, 15]. Studies have shown that blood flow to the AT decreases with increasing age, with gender (decreased in men), and with physical loading conditions [15, 16]. However, Langberg et al. have shown that exercise increases blood flow at 5 cm proximal to the insertion of the AT by fourfolds but only 2.5-folds 2 cm proximal to the insertion [17]. This was also supported by Astrom and Westlin [15] wherein they concluded that blood flow was evenly distributed throughout the tendon but has a significant lower blood flow to the distal portion when they compared competitive runners against healthy volunteers. They also concluded that symptomatic AT had an increase in blood flow to the affected area.

Tenocytes and tenoblasts account for 90–95 % of the cellular population, while most of the extracellular matrix is made up of type I collagen (70 %) and elastin proteins (2 %) [18, 19]. The type I collagen approximately rotates 11–90° in a medial direction with the proximal medial fibers becoming posteriorly located upon its insertion to the calcaneus [20]. This orientation provides mechanical advantage with rotational contraction; however, it may also be a potential area to strangulate the blood supply to the AT upon movement and eventually cause rupture [14]. It was noted by Mafulli and colleagues [21] that tenocytes from a ruptured AT produced more type III collagen, which are less resistant and elastic to tensile force than type I collagen. Normally, the AT is subjected to loads 2–3 times the body weight when walking and up to 10 times the body weight with certain athletic activities [8]. However, with a tear at the AT, people experience significant loss in plantar flexion strength which can lead to difficulty during sports and even climbing stairs [22].

Achilles Tendinosis

Introduction

Achilles tendinopathy is a painful condition that occurs commonly in active and even inactive individuals [23]. It was initially reported to be a tendon disorder which has multiple suggested pathologies which are based on poor scientific evidence as explained by Lake and Ishikawa [23]. But later researches have clarified the difference between an Achilles tendinitis and an Achilles tendinosis (Table 13.1).

Clinical Features

An Achilles tendinitis (tendonitis) occurs when there is a clinical presence of pain and swelling. It is an inflammatory process seen on a biopsy specimen of a diseased tendon. On the other hand, an Achilles tendinosis refers to a degenerative process of the tendon without histologic or clinical signs of intratendinous inflammation. Leadbetter [24] suggested that tendinosis is a failure of the cell matrix to adapt to repetitive trauma caused by an imbalance between the degeneration and synthesis of the matrix. An isolated pain at the insertion of the Achilles tendon to the calcaneus due to an intratendinous degeneration is referred to as insertional Achilles tendinosis, while a non-insertional

Table 13.1 Difference between Achilles tendinitis and an Achilles tendinosis

Achilles tendinitis	Clinical presence of pain and swelling
	Presence of inflammatory process seen on biopsy of a diseased tendon
Achilles tendinosis	Degenerative process of the tendon without histologic or clinical signs of intratendinous inflammation

Table 13.2 Etiology of Achilles tendinosis

Overuse
Decreased blood supply and tensile strength with aging
Muscle imbalance or weakness
Insufficient flexibility
Malalignment (hyperpronation)
Genetics
Endocrine disorders
Free radical production

(midportion) Achilles tendinosis occurs in the main body of the Achilles tendon.

Etiology

The etiology behind an Achilles tendinosis remains unclear, but there are many theories as to the cause of the disease which include overuse, decreased blood supply and tensile strength with aging, muscle imbalance or weakness, insufficient flexibility, and even malalignment such as hyperpronation [23]. Some also suggest that genetics, endocrine disorders, and free radical production can also produce tendinosis (Table 13.2).

Biopsies of diseased tendons revealed that there are cellular activation evidenced by an increase in cell numbers and ground substance, collagen disarray, and neovascularization. Prostaglandin inflammatory elements are not present, but neurogenic elements such as substance P and calcitonin gene-related peptides have been isolated [23]. Neurovascular ingrowth and glutamate (a potent modulator of pain) have also been noted in the diseased tendons, and they have been postulated to be the sources of pain in patients with Achilles tendinosis.

Tendon injuries occur in 30–50 % of all sports-related injuries. Sixty-six percent of joggers complain of Achilles tendon pain, and 23 % of them usually have insertional Achilles tendinosis [25]. Risk factors for the development of this condition include diabetes, hypertension, obesity, hormone replacement, and use of oral contraceptives [26]. One study even suggests that having a pes cavus could also cause Achilles tendinosis (Table 13.3).

Table 13.3 Risk factors for the development of Achilles tendinosis

Diabetes
Hypertension
Obesity
Hormone replacement
Use of oral contraceptives
Pes cavus

Imaging Studies

Ultrasonography and magnetic resonance imaging (MRI) are the most common tool in diagnosing an Achilles tendinopathy, and both have its advantages and disadvantages. The ultrasound is an inexpensive, reproducible and fast diagnostic tool for determining the thickness of an inflamed AT. However, it is user dependent, and the interpretation of the result is reliant on the experience of the examiner (Figs. 13.1, 13.2, and 13.3). The MRI on the other hand is more superior when it comes to detecting chronic inflammatory changes of the AT, but this modality is relatively expensive and cannot be used for dynamic assessment unlike the ultrasound [25] (Figs. 13.4 and 13.5).

Conservative Treatment

The first line of treatment for any kind of disease is still the noninvasive methods such as activity modification, orthotics, heel lifts, massage, hot and cold compresses, strengthening exercises, ultrasound, and nonsteroidal anti-inflammatory drugs (NSAID) or oral corticosteroids [25] (Table 13.4). Since there are no prostaglandin inflammatory mediators in an Achilles tendinosis, NSAIDs have been questioned with regard to its effectiveness [27]. In a randomized double-blind placebo-controlled trial done by Astrom and Westlin [27], it was concluded that piroxicam was no more effective than placebo. Though the traditional noninvasive methods are readily available and give most of the patients short-term pain relief, 30 % of them still find these alternatives as ineffective.

Corticosteroids are a class of medications that are related to a steroid called cortisone. They

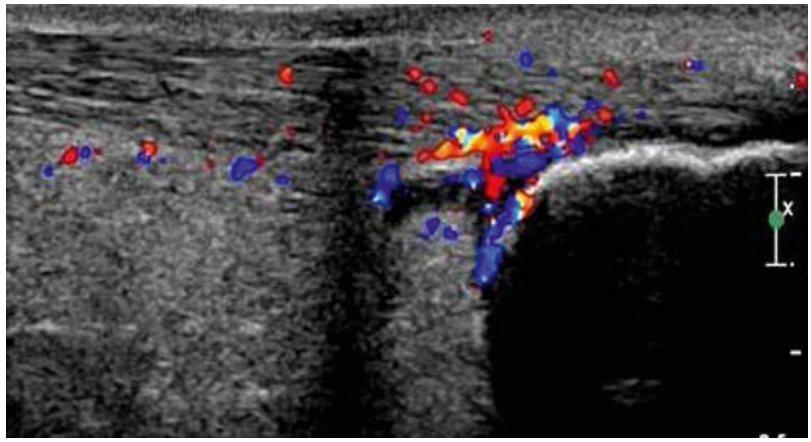


Fig. 13.1 Ultrasound image of an Achilles tendinitis with severe thickening and slightly heterogeneous low echogenicity of the distal Achilles tendon

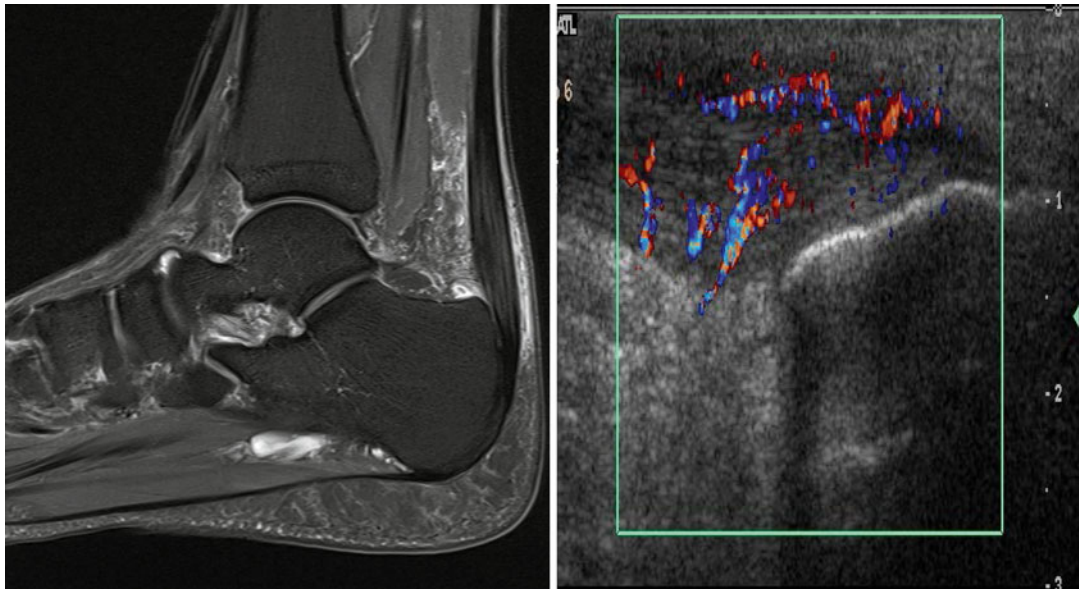


Fig. 13.2 This is a case of an Achilles tendinosis with retrocalcaneal bursitis, where on MRI (*left*) of the AT it showed that the distal portion to be swollen and there was a presence of diffuse enhancement together with diffuse peritendinous fat infiltration. There was also an existence of a retrocalcaneal bursal fluid on the image. On ultrasound (*right*), the AT was seen to have severe thickening with slight heterogeneous low echogenicity of the distal AT 5 cm in length and 10 mm in thickness. There was also a prominent increase in the vascularity of the distal AT on Doppler ultrasound

relieve pain by reducing the inflammation that occurs in a diseased tendon. There have been many conflicting studies regarding the use of corticosteroid injections in Achilles tendinosis since there is no inflammatory process. It produces short-term pain relief after peritendinous corticosteroid injection in one randomized control trial but another demonstrated no improvement at all [28]. Gill and colleagues [29] reported 40 % improvement without tendon rupture. Since their role in Achilles tendinosis has not yet been established, precaution is always advised as there have been multiple reports of complete or partial ruptures of the AT [28].

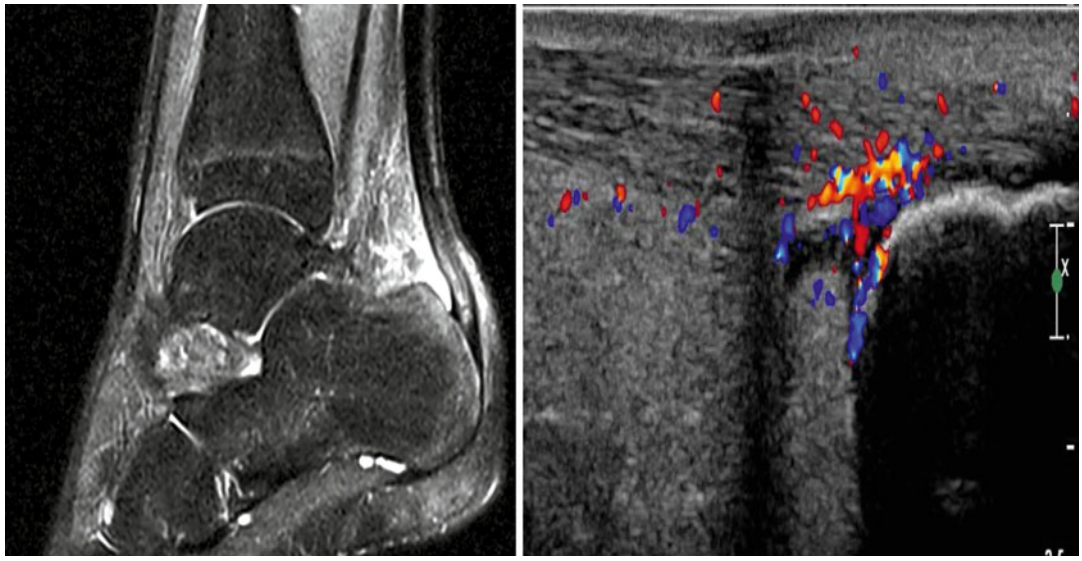


Fig. 13.3 This is a case of a 17-year-old male who was diagnosed to have bilateral inflammatory Achilles tendinitis with retrocalcaneal bursitis to rule out ankylosing spondylitis. Right Achilles tendon shows 5.8 mm thickness with equivocal thickening, and the retrocalcaneal bursitis is combined anterior to calcaneal attachment site with 7.5 mm thick fluid collection. Increased vascularity is confirmed by color Doppler in the retrocalcaneal bursa and distal Achilles tendon

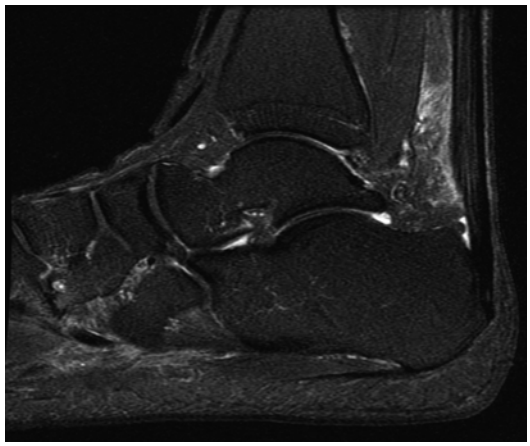


Fig. 13.4 This is a case of an Achilles tendinitis with paratendinitis of the left AT. MRI shows a fusiform swelling and enhancement of the Achilles tendon (9 mm thickness, 6 cm segment) with peritendinous fat infiltration in the Kager's fat fad

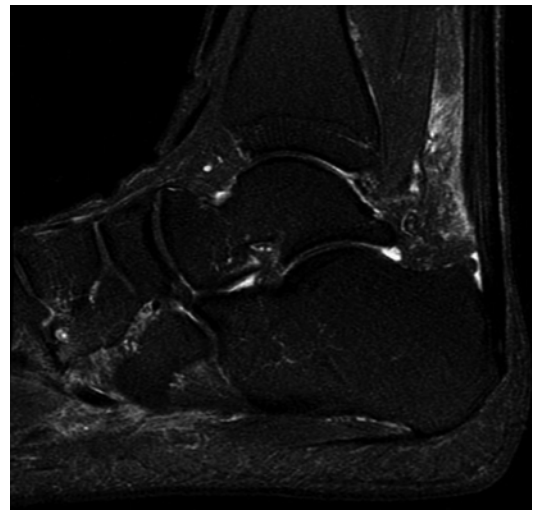


Fig. 13.5 Magnetic resonance image of an Achilles tendinitis with fusiform swelling and enhancement of the Achilles tendon together with peritendinous fat infiltration in the Kager's triangle

The eccentric training program developed by Stanish et al. promotes tendon healing by increasing the tendon volume and signal intensity which is thought to be a response to trauma [30]. But after a 12-week program, a decrease in size and a more normal tendon appearance were noted on ultrasound and MRI. With continuous eccentric loading of the AT, there will be lengthening of the muscle-tendon unit and increase in the tendon's capacity to bear load overtime. Repetitive eccentric training may cause damage

Table 13.4 First line of treatment for an Achilles tendinosis

Activity modification
Orthotics
Heel lifts
Massage
Hot and cold compresses
Strengthening exercises
Ultrasound
Nonsteroidal anti-inflammatory drugs (NSAID)
Oral corticosteroids

to the abnormal blood vessel and the accompanying nerves in the tendon thus eliminating the pain as concluded by Alfredson and colleagues [31]. With the 12-week program, it has produced 90 % good results with midportion Achilles tendinosis and 30 % good results in insertional Achilles tendinosis [32].

The extracorporeal shock wave therapy is another treatment option for Achilles tendinosis. It can be given as a low-energy treatment (which is a 3 weekly sessions without local anesthesia or intravenous anesthesia) or as a high-energy treatment (which is a single session but requires local or intravenous anesthesia). Microtrauma is produced by the repeated shock wave to the affected area which then stimulates neovascularization (Fig. 13.6). It is this new blood flow that promotes tissue healing and relief from pain. It can also inhibit afferent pain receptor function and produce a high number of nitric oxide synthase. In two non-randomized clinical trials, Furia [33, 34] reported that patients treated with high-energy ESWT had more successful results than those treated with other traditional nonoperative treatment. This result is in contrast to Costa and colleagues' two double-blinded randomized controlled trial where they reported no statistically significant treatment effects in 49 patients with Achilles tendinosis treated with low-energy ESWT [23].

Polidocanol is a sclerosing agent used to sclerose neovascularization. It causes thrombosis through a selective effect on the intima even when the drug is injected extravascularly. Based from European literature, when the process of neovascularization in the injured tendon is eliminated, the new blood vessels including the sensory nerves that are linked with them are destroyed producing pain relief to the patient [23]. Protocol states that the sclerosing agent is injected 2–3 times with 6–8 weeks apart [35]. Each injection is followed by a few days of rest, and high-impact activities are restricted for 2 weeks. However, there have been reports of tendon ruptures in elite athletes after multiple sclerosant injections [23].

Sclerosing thermal therapy uses a radiofrequency probe to carry out microtenotomies. Similar to sclerosing agents, the thermal energy applied to the diseased tendon destroys the new blood vessels together with the sensory nerves that accompany them. Boesen and colleagues [36] demonstrated good results after using an ultrasound-guided electrocauterization technique in 11 patients with chronic midportion tendinosis.

Glyceryl trinitrate is a prodrug of endogenous nitric oxide. It is commercially available as a topical patch to relieve nitric oxide which is a soluble gas that acts as a messenger molecule that can affect many cellular functions, including tendon healing [37]. It was believed that it increases collagen production by fibroblasts, cellular adhesion, and local vascularity. In a randomized double-blind placebo-controlled study by Hunte and Lloyd-Smith [38], a topical glyceryl trinitrate patch was more effective than placebo for reducing pain from chronic non-insertional Achilles tendinosis in the first 12 and 24 weeks of use. Another study showed excellent results (78 %) for ATs treated with 1.25 mgs of topical glyceryl trinitrate every 24 h for 6 months as compared to the placebo group (49 %).

Low-level laser therapy produces effects on a diseased tendon like enhanced ATP production, enhanced cell function, and increased protein synthesis [39]. It also reduces inflammation, increases collagen synthesis, and promotes angiogenesis. In a study done by Stergiolas and colleagues [40], they concluded that Achilles tendinosis patients who underwent eccentric

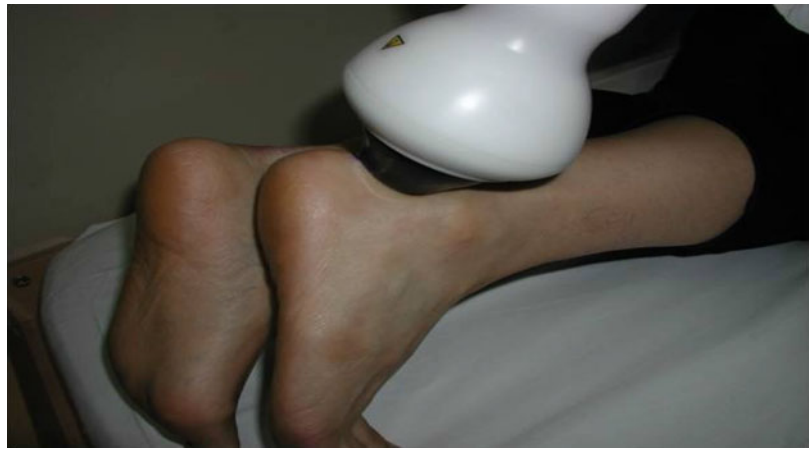


Fig. 13.6 Use of extracorporeal shockwave therapy on Achilles tendinosis patient

exercises together with low-level laser therapy showed decreased pain intensity, morning stiffness, tenderness to palpation, active dorsiflexion, and crepitus with no side effects as compared to those who underwent eccentric exercises only. However, there is still limited data to verify the effectiveness of low-level laser therapy for the treatment of Achilles tendinosis.

Prolotherapy is a series of a hypertonic glucose with lignocaine/lidocaine injection designed to sclerose the new blood vessels and nerves. A randomized control trial done by Sweeting and Yellard [41] compared the outcomes of eccentric loading exercises and prolotherapy injections used as a single dose and in combination for non-insertional Achilles tendinosis. It showed that as early as 6 weeks, the combination therapy produced more rapid improvements with regard to symptoms of pain compared to monotherapy in selected subjects.

In chronic Achilles tendinosis, there is an absence of inflammation and a paucity of platelets [42]. Platelet-rich plasma (PRP) injection works by increasing the concentration of platelets (through injection) in an injured tendon, which enhances the healing potential through stimulation of the revascularization process. Platelets when activated produce cytokines and granules that then produce growth factors that aid in the healing process [42]. There are no established indications for the use of PRP in Achilles tendinosis although the current best evidence suggests that patients will improve after platelet-rich plasma treatment (Fig. 13.7).

Fig. 13.7 Platelet-rich plasma (PRP) prepared from autologous blood of the patient

However, the improvement is not significantly better than physical therapy. De Jonge and colleagues [43] with a randomized control trial concluded that there were no clinical and ultrasonographic superiority of PRP injection over a placebo injection in chronic Achilles tendinosis at 1 year combined with an eccentric training program.

Operative Treatment

Operative treatment of Achilles tendinosis involves the removal of abnormal tissues and lesions, fenestration of the tendon through multiple longitudinal creations, and possibly stripping the paratenon. The goal of such management is to remove degenerative nodules, excise fibrotic adhesions, restore the vascularity, and stimulate viable cells to initiate an inflammatory response and reinitiate healing. Results showed that an open surgical treatment for an Achilles tendinosis produced 18.8 % unsatisfactory outcome in nonathletic subjects as compared to athletic subjects (8.9 %) [39]. A study also showed that reoperation is higher in women (12.2 %) as compared to men (6.7 %) [44].

Percutaneous Longitudinal Tenotomy

Testa and colleagues [45] developed a technique where multiple, ultrasound-guided, percutaneous incisions are made through the diseased AT. This procedure can be done as an outpatient surgery where the patient is placed on prone position. Local anesthesia is applied over the diseased area which can be identified through palpation or ultrasound. A stab knife is then used to make a longitudinal incision parallel to the long axis of the AT. With the knife pointing cephalad, the ankle is then fully dorsiflexed. Then with the scalpel pointing towards the caudal direction, the ankle is then fully plantarflexed. Four separate stab incisions are made approximately 2 cm apart; 1 medial and proximal, 1 medial and distal, 1 lateral and proximal, and 1 lateral and distal. The incisions are closed with adhesive strips. Early range of motion is encouraged, and full weight bearing is allowed after 2–3 days with an expected return to previous activity after 4–6 weeks. Testa achieved 56 % excellent results with this treatment and only 8 % poor results [45, 46]. Maffulli et al. [47] modified this technique in 2009 by adding another stab wound at the central portion of the diseased area (Fig. 13.8).

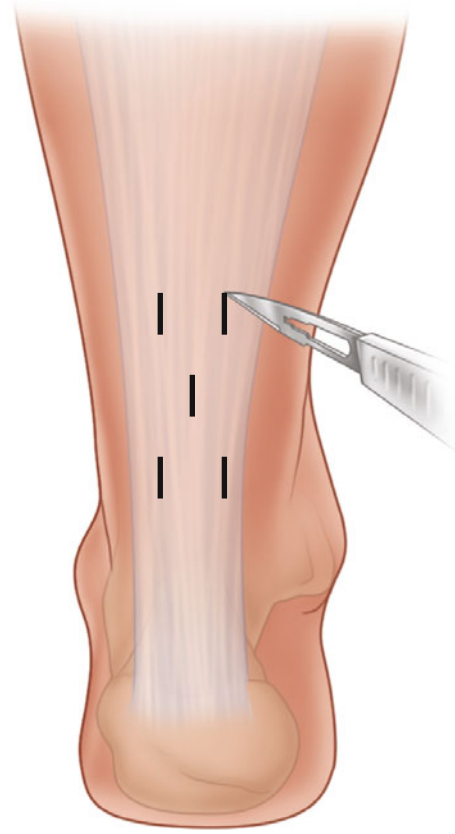


Fig. 13.8 Schematic representation of a percutaneous longitudinal tenotomy using a blade no. 11

Minimally Invasive Tendon Stripping

Longo and colleagues [48] introduced a technique of stripping the adhesions in the AT through a minimally invasive technique in 2008. This involves four 0.5 cm longitudinal skin incisions along the border of the AT. Two are made just medial and lateral to the origin of

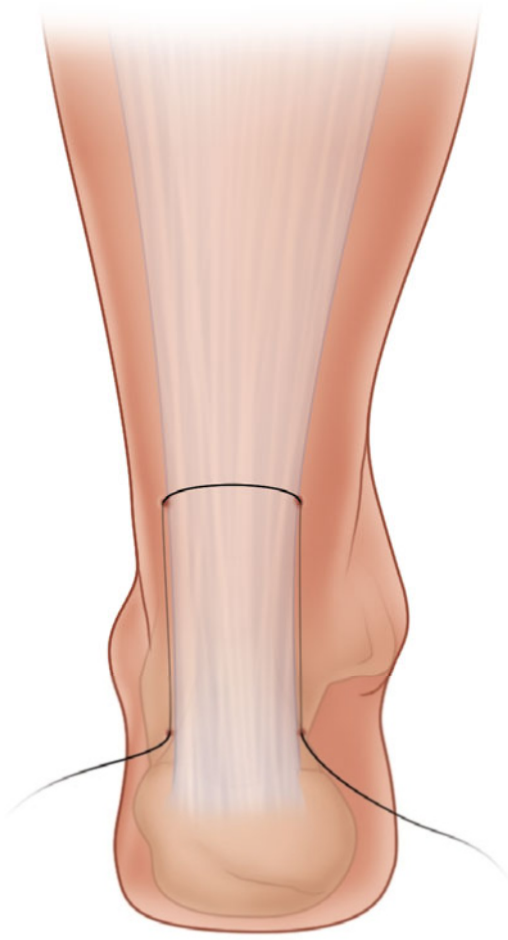


Fig. 13.9 Schematic representation of a minimally invasive tendon-stripping technique where an Ethibond is passed from the proximal incisions and is pulled caudally through the distal incisions

the tendon and two more at distal end of the tendon close to the insertion. A mosquito is then inserted through the incisions, and the proximal and distal portions of the AT are freed of peritendinous adhesions. A number 1 Ethibond thread is inserted at the 2 proximal incisions over the anterior aspect of the Achilles tendon. Ends of the Ethibond are then retrieved from the distal incisions. The Ethibond is then slid onto the tendon, causing it to be stripped and freed from adhesions at the anterior surface of the tendon. The procedure is repeated for the posterior aspect of the tendon (Fig. 13.9). This will disrupt the neovascularization of the damaged tendon and its accompanying nerve supply. After the procedure, the patient is allowed to do range of motion exercises and can be allowed to do full weight bearing [47].

Endoscopic Tendon Debridement

Small skin incisions are made and an arthroscopic shaver is introduced into the AT to debride the peritenon. This procedure produces decreased postoperative complication thus allowing the patient early return to previous activity [49]. Steenstra and van Dijk reported significant pain relief after 2–7 years of 20 patients who were able to return to sports after 4–6 weeks [49]. Maquirriain in a long-term follow-up study (5 years) reported a high rate of excellent results in patients with chronic Achilles tendinosis with 0 % infection and systemic complications [50]. There was however a report of delayed keloid lesion and a seroma with chronic fistula formation in his study postoperatively.

Open Tendon Debridement and Repair, with or without Augmentation

This procedure is the most commonly used surgical modality for chronic painful Achilles tendinosis. It is indicated for moderate to severe tendinosis when conservative measures fail and is especially suitable for the Achilles tendinosis with single or multiple discrete degenerated lesions. The paratenon is carefully incised and any inflammatory peritendinitis is removed (Cases 13.1 and 13.2). If on MRI or ultrasound there is an intratendinous nodule, or there is a palpable thickening within the tendon, excision is recommended until viable tissues are seen [51]. Any residual degenerated tissue increases the risk of persistent postoperative pain [51]. If there is a Haglund's deformity, it can also be excised at this point (Fig. 13.10) (Case 13.3). A turned down tendon flap may be necessary in case of significantly large defect in the tendon created by the excision. The flexor hallucis longus can be used to augment the AT by tendon transfer if more than 50 % of diseased

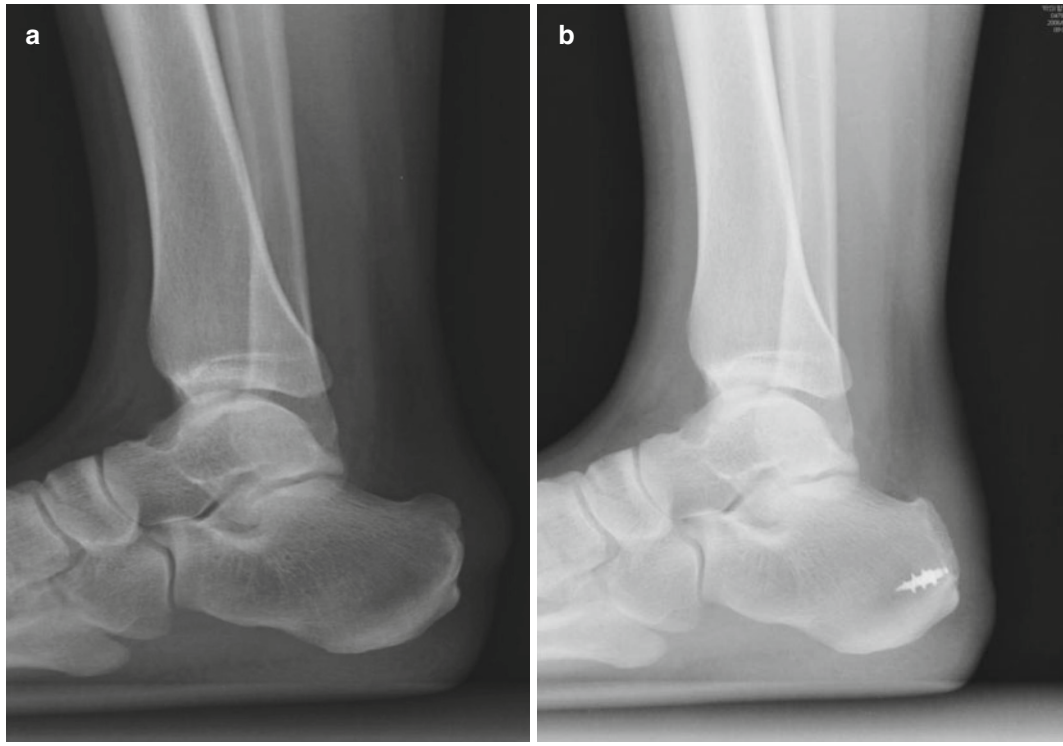


Fig. 13.10 (**a**) Calcaneus with Haglund's deformity. (**b**) Haglund's deformity excised and the Achilles tendon reattached with suture anchor

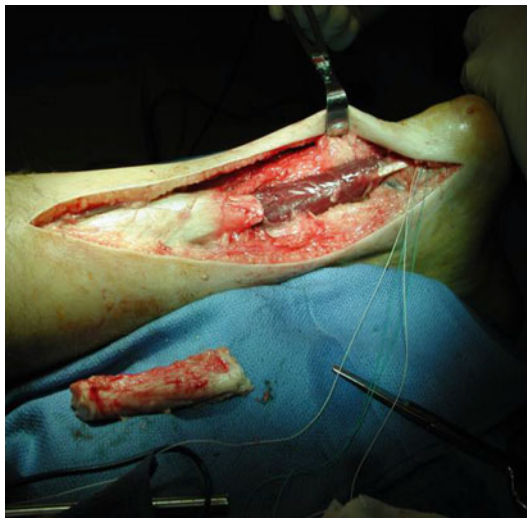


Fig. 13.11 Excision of diseased portion of the Achilles tendon with augmentation of the flexor hallucis longus

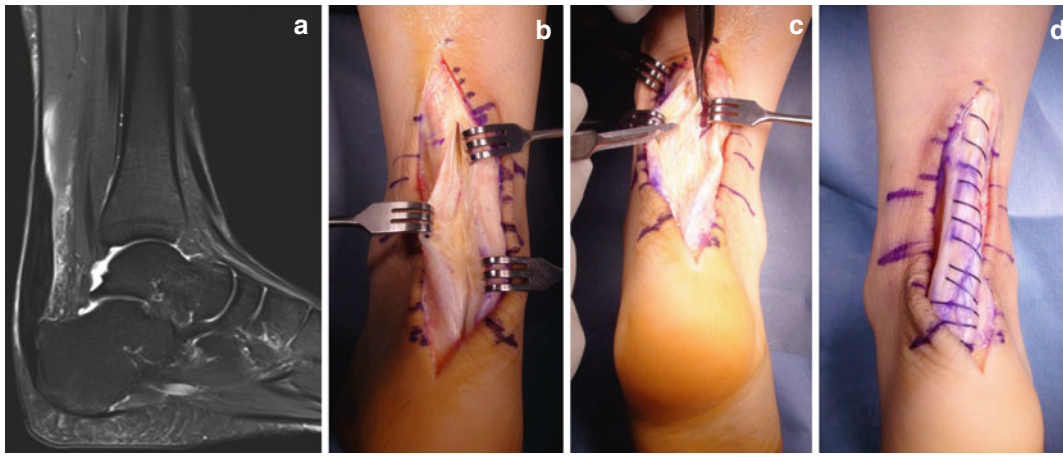
tendon was removed (Fig. 13.11). Schon and colleagues [52] reported significant improvement in terms of AT function, physical function, and pain intensity with this procedure in relatively inactive, older, and overweight patients. However, there are some reports of tendon rupture after an open debridement.

Tips

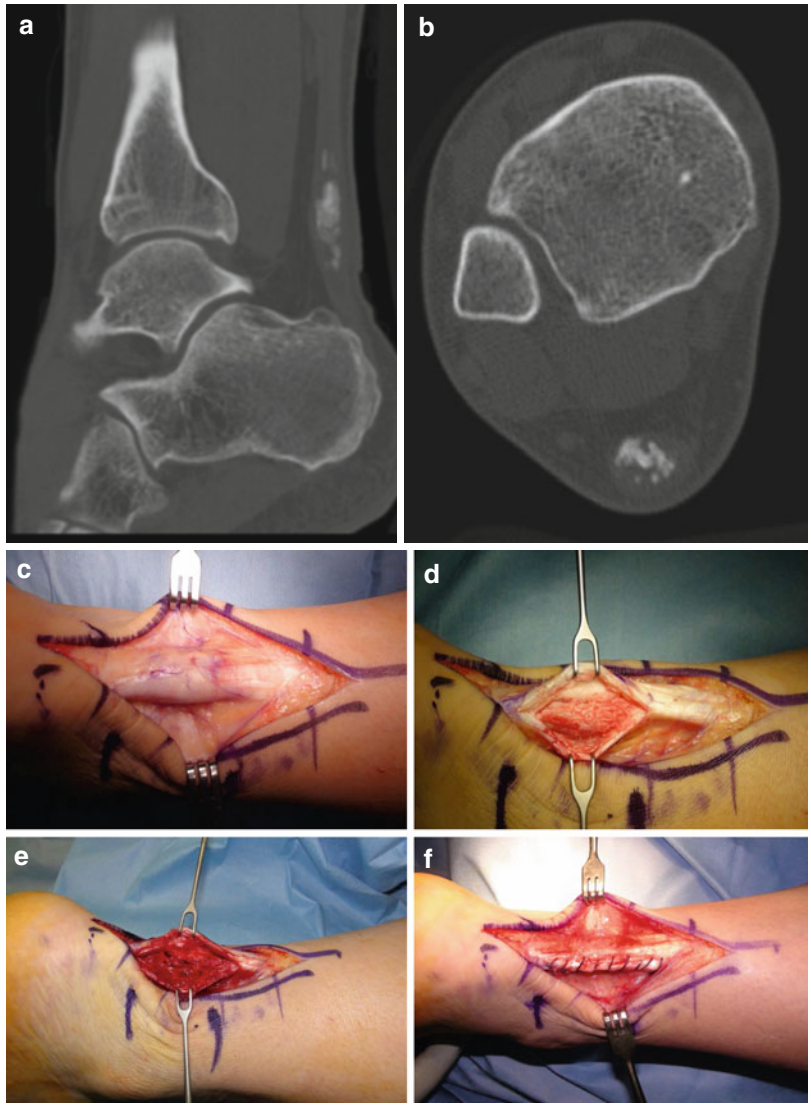
Achilles tendon debridement is often difficult to perform clearly because of the poor demarcation of the degenerative lesions or nodules in the Achilles tendinosis. Therefore, we recommend eccentric loading exercise and ESWT with silicon heel cup, NSAID medication, and restriction of overactivity as baseline treatments. Steroid injection is never prescribed for Achilles tendinosis due to potential rupture even though injected at the anterior region. The effects of PRP and stem cell injections should be discussed more in the future.

Cases

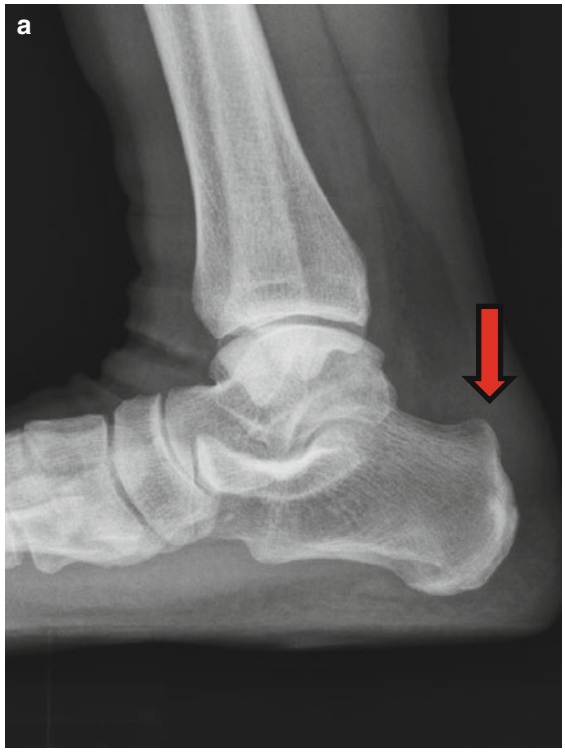
Case 13.1: A Chronic Achilles Tendinosis Debridement (**a**) A 39-year-old female hairstylist presented with a chronic Achilles tendinosis for almost 20 years which was aggravated by standing. Recent MRI showed that there is a thickened AT. She was initially treated conservatively with multiple ESWT and eccentric exercises without improvement. (**b**, **c**) Achilles tendon debridement through a post-midline approach showed yellowish diffuse degenerated inner portion. (**d**) Tendon repair with running suture using a 1.0 Vicryl was performed and a short leg cast was applied for 4 weeks.

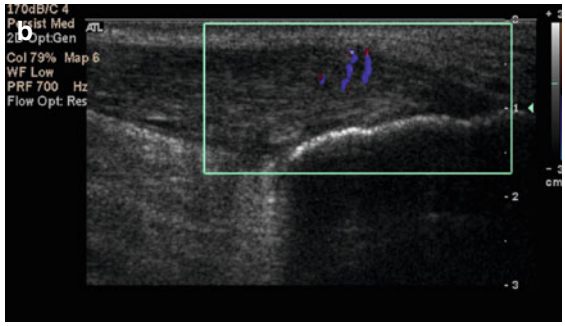


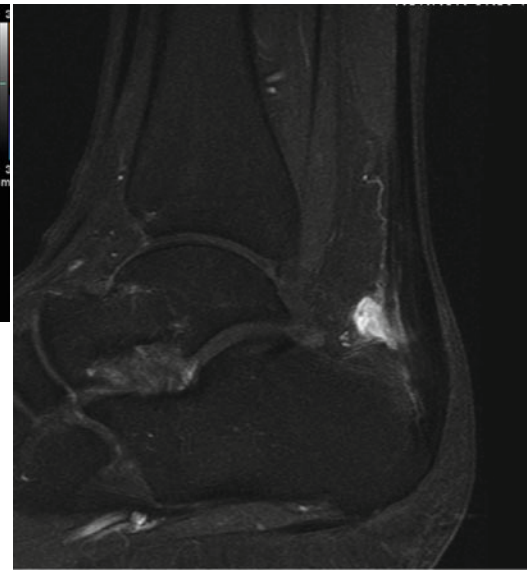
Case 13.2: A Calcified Mass in the AT (**a**, **b**) A 17-year-old male baseball player presented with pain and tenderness at the posterior ankle for almost 6 years. He was treated with ESWT for 8 times without improvement (VAS 7, AOFAS 72). CT showed a calcified mass in the intra-substance portion of the AT 6 cm proximal to the calcaneal tuberosity (chronic calcific tendinopathy). (**c**–**f**) The thickened AT portion approximately 5 cm long (**c**) was incised and the multiple calcified mass was identified (**d**) and excised, leaving a thin layer of AT remnant (**e**), which was repaired with multiple running suture using a 1.0 Vicryl suture.

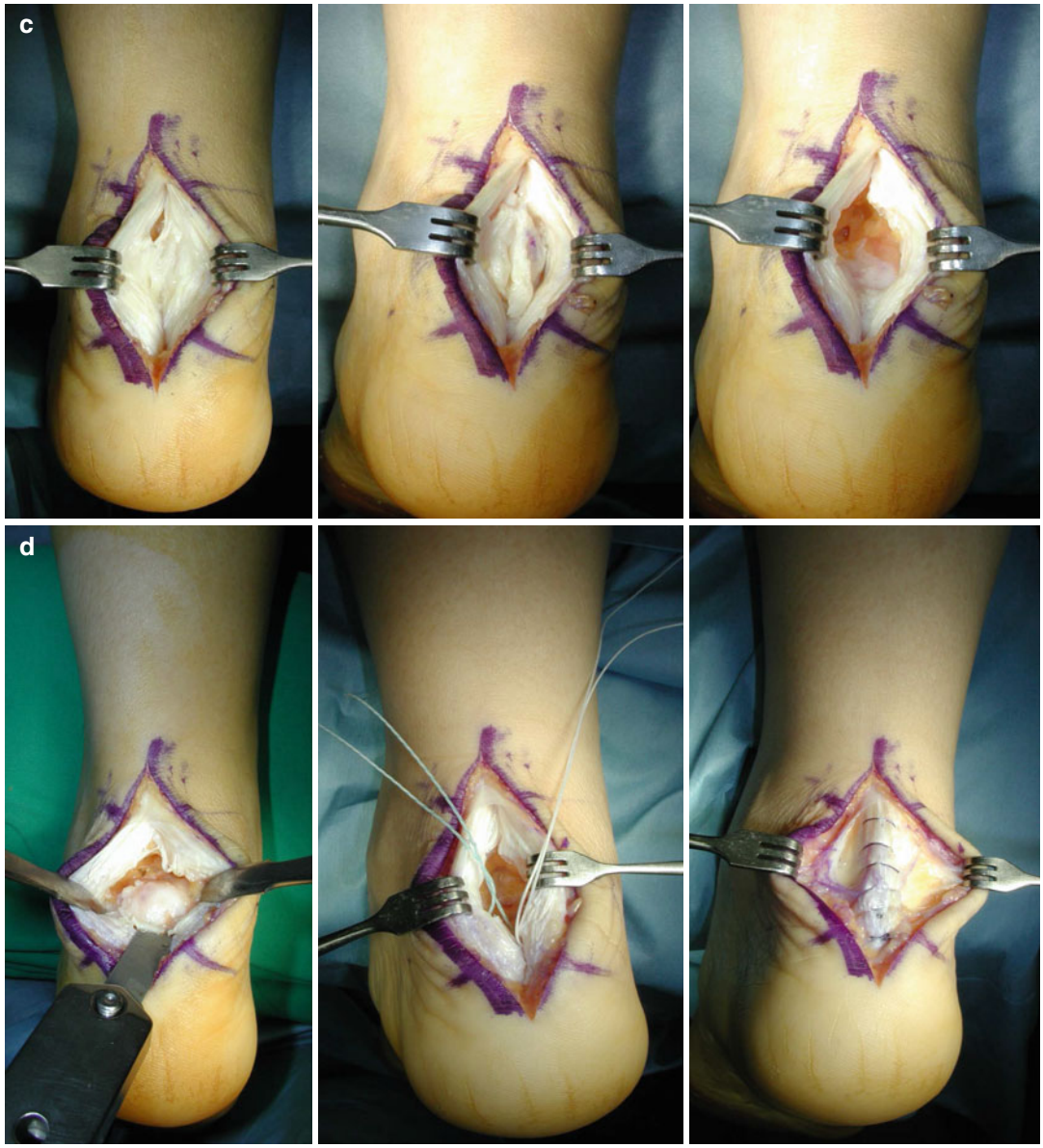


Case 13.3: Insertional Achilles Tendinopathy with Haglund's Deformity (**a**) This is a case of a 50-year-old female patient with insertional Achilles tendinopathy and a Haglund's deformity (*red arrow*). (**b**) MRI and US showed severely thickened distal part of AT with low echogenicity and increased vascularity combined with a retrocalcaneal bursitis. (**c**) A central midline approach showed tendinosis of the central part of the tendon. Debridement of the degenerated portion was then performed. (**d**) The Haglund's calcaneal tuberosity was adequately resected and the detached distal portion was firmly reattached to the calcaneus with a 3.5 mm suture anchor. The bisected AT was then repaired. (**e**) Post-op x-ray with resected calcaneal tuberosity.





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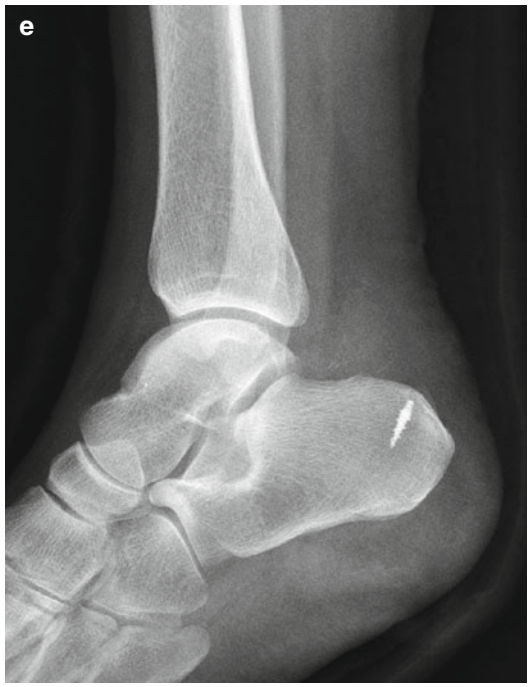


Achilles Tendon Ruptures

Risk Factors for an AT Rupture

Studies have shown that recreational athletes as well as "weekend warrior" athletes are more prone to rupture the AT due to a sedentary life combined with intermittent activities as compared to professional athletes who are consistently exercising. With regular exercise, it has been shown that it allows the diameter of the AT to thicken and to decrease the chance of rupture due to an atrophied AT. Other risk factors associated with a ruptured AT are advancing age, extreme change in training level, tight gastrocnemius, poor vascularity, overpronation, cavus foot, and recent steroids or fluoroquinolone therapy. Pathologic conditions such as infectious diseases, neurologic conditions, arteriosclerosis [53], genetically determined collagen abnormalities, and high serum lipid concentration are also associated with AT ruptures (Table 13.5).

Table 13.5 Risk factors for an Achilles tendon rupture

Recreational athletes (weekend warriors)
Advancing age
Extreme change in training level
Tight gastrocnemius
Poor vascularity
Over pronation
Cavus foot
Recent steroids or fluoroquinolone therapy
Infectious diseases
Neurologic conditions
Arteriosclerosis
Genetically determined collagen abnormalities
High serum lipid concentration

Acute Versus Chronic AT Rupture

By definition, an AT rupture is a disruption of the continuity of the AT which is most likely the result of a combination of mechanical stress and intratendinous degeneration. Weber and colleagues [54] noted that a tendon loses its normal configuration when it is stretched by more than 2 %. When the tendon is stretched by 3–4 % its normal length, some microscopic disruptions can then be noticed, and when stretched further by 8 %, macroscopic tears then develops.

In a study by Arner and Lindholm [55], three main mechanisms were reported with 53 % of all AT ruptures occurred during weight bearing with the forefoot pushing off while the knee is in extension as seen in athletes who are into sprints and jumping activity. Sudden unexpected dorsiflexion of the ankle (falling from a stair) accounted for 17 % of ruptures and 10 % occurred when there was a violent dorsiflexion of a plantarflexed ankle as seen in patients who fell from a height.

Most of the acute AT rupture occurs during sports activity which accounts to 35 % of all tendon injuries [56]. Once undiagnosed, which usually accounts to 25 % of the patient population [57], these tears may go on to be considered chronic or neglected ruptures. However, their transition is still arbitrary and not well defined based on present literature [22, 58]. But the consensus is that AT tears which are diagnosed at

4–6 weeks from the time of injury could already be classified as a chronic [22].

Pathophysiology of Acute at Rupture

Three theories have been postulated for the occurrence of an acute AT rupture, and these are the degenerative theory, the mechanical theory, and the drug-related tendon rupture theory.

Degenerative Theory

The degenerative theory was postulated due to an observation that the AT undergoes chronic degeneration which leads to a rupture even without excessive loads being applied. Chronic overloading, microtrauma, and physiologic alterations in the tendon were just some of the factors associated with degeneration of the tendon [3]. Some authors also hypothesized that alterations in the blood flow with subsequent hypoxia and impaired metabolism were also factors for the development of degenerative changes at the AT. Waterston et al. [59] also noted that ruptured and tendinopathic tendons produce type III collagen which are less resistant to high loads and tensile forces. This theory was first noted by Arner and colleagues [60] when they hypothesized that prior to the rupture, all of their patients already presented with degenerative changes along the AT. This theory was also supported by Jozsa and Kannus [61] as they observed marked degenerative changes and collagen disruption in all of the ATs they operated within 24 h.

Mechanical Theory

Sporting activities play an important role in the development of problems with the AT, especially when training sessions are wrongly performed. Based on this theory, the AT can rupture due to frequent and repetitive microtrauma to the tendon without enough time for the tendon to repair itself even in healthy tendons without prior degenerative changes. In healthy ATs, the tendon could rupture due to a violent muscle contraction in the presence of an incomplete synergism of agonist muscle contraction, inefficiency of the plantaris muscle to act as a tensor of the AT, and also if there is a discrepancy between the thickness quotient of the gastrocnemius muscle and the AT itself. Uncoordinated muscle contraction as a cause of rupture of the AT was also proposed by Inglis and Sculco in 1981 [62]. They also noted that athletes who return to sports too early after a prolonged period of inactivity are at greater risk to rupture their ATs [62]. Literature also noted that functional overpronation, gastrocnemius/soleus insufficiency, overtraining, and repeated microtrauma from an eccentric loading of a fatigued muscle are also possible causes of rupture.

Drug-Related Tendon Rupture

It has been written in many researches that anabolic steroids and fluoroquinolones are widely implicated in the rupture of ATs, and in a study by Longo and colleagues in 2013 [3], they found that such drugs decrease the tensile strength and increase the risk of the AT to rupture due to dysplasia of the collagen fibrils. In addition, corticosteroids have also been found to cause collagen necrosis which then interferes with the healing of tendons. Distefano and Nixon [63] also concluded that corticosteroids may mask or delay the onset of symptoms of a problematic tendon due to its analgesic effect, which may encourage individuals to continue their high level of activity even when the tendon is already damaged.

In 1987, McEwan and Davey [64] reported on the first case of tendon rupture associated with ciprofloxacin. Fluoroquinolones like ciprofloxacin, pefloxacin, ofloxacin, levofloxacin, and norfloxacin are the most commonly associated antibiotics with tendon disorders [3]. In an animal study done by Szarfman et al. in 1995 [65], they were able to show that administration of fluoroquinolone produced disruption of the extracellular matrix of the cartilage, depletion of collagen, and necrosis of chondrocytes.

Pathophysiology of Chronic at Rupture

Most of the chronic AT ruptures are usually misdiagnosed as reported by Ingles and colleagues

[66] and are attributed to physicians and patients being misled by the inconsequential nature of the trauma reported, lack of significant pain, and the patient's ability to weakly plantarflex the ankle. During an acute AT rupture, appreciation of a gap along the tendon could be difficult to be palpated due to significant swelling which could also lead to misdiagnosis [57].

Once an acute AT rupture is misdiagnosed, it was observed by Carden and colleagues [67] that within 1 week from the acute trauma, enough granulation tissue has formed between tendon ends to prevent their apposition in a closed manner. The granulation tissues will then develop into fibrous tissues which will then be gradually stretched due to continuous contraction of the calf muscles [53, 68]. As the tendon heals in an elongated position, it then loses its mechanical efficiency and eventually produces ankle plantar flexion weakness and its associated gait disturbances [69]. If the plantaris muscle is present, it was observed to be hypertrophied leading to believe that this muscle synergistically assumes the lost function of the ruptured AT [22]. If a gap within the tendon is appreciated, this can be attributed to the continuous contraction of the gastrocnemius muscle [70]. However, the size of the gap can be variable, and the associated retraction of the proximal stump of the tendon can prevent end-to-end apposition at this point. Furthermore, the proximal stump could have adhered to the posterior fascia making it difficult to identify.

Patient Evaluation

Patients who suffered from an acute AT rupture usually report of a sharp stabbing pain or an audible snap on the affected leg and would usually describe the incident as being kicked at the posterior aspect of the ankle. Patients also complain of inability to bear weight due to weakness or stiffness of the affected ankle. However, some patients will still have the ability to plantarflex the ankle by using their long/short toe flexors and even their peroneal muscles [3]. On physical examination, diffuse swelling and hematoma formation can be observed. However, an appreciable gap can also be seen or palpated 2–6 cm proximal to the insertion of the tendon if there is minimal swelling.

On the other hand, symptoms are usually vague for patients who suffer from chronic or neglected AT rupture. Pain and swelling can be noticed by the patient; however, the sense of fatigue and weakness with prolonged ambulation are the most common reasons for consult [58, 71]. On physical examination, calf atrophy and a calcaneal gait with loss of normal push-off during ambulation are evident [72]. Presence of a gap can be variable and will be dependent on the reparative process. If tissues do fill the gap, no palpable defect can be appreciated [73]. But due to the loss of the normal restraint of the AT, there will be increased passive dorsiflexion at the ankle. Inability to perform single leg heel raise of the affected leg will also be pronounced due to plantarflexion weakness [53].

Special Tests

Thompson Test

This test has been used to detect AT ruptures, was first described by Simmonds [74] in 1957, and was popularized by Thompson and Doherty [75] in 1962. The patient is laid on a prone position with both feet hanging over the edge of the examining table. The calf is then squeezed by the examiner to cause shortening at the Achilles tendon. A normal response upon doing this procedure is to generate plantar flexion at the ankle in an intact AT, and failure to do so is considered diagnostic of an AT rupture [71] (Fig. 13.12).

Matle's Test

The patient is placed on a prone position with both feet over the edge of the examining table. The patient is then asked to actively flex or bring the knee to 90°. A normal response to this test is to produce active plantar flexion of the foot caused by shortening of the gastrocnemius muscle during active knee flexion. A ruptured AT is considered when the foot falls into neutral or slight dorsiflexion [72] (Fig. 13.13).

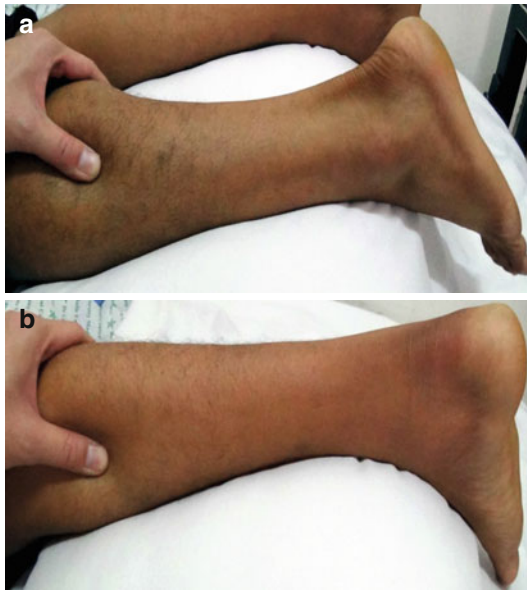


Fig. 13.12 **Thompson Test:** (**a**) Plantarflexion of the ankle upon squeezing the calf muscle in an intact Achilles tendon. (**b**) Absence of plantarflexion of the ankle upon squeezing the calf muscle in a torn Achilles tendon

O'Brien's Test

The patient is placed in a prone position with both feet over the edge of the examining table, and a needle is inserted approximately 10 cm proximal to the calcaneal insertion of the tendon. The needle is introduced through the skin of the calf at a right angle just medial to the midline, and the needle tip is estimated within the substance of the tendon without transfixing the tendon to adjacent structures. An intact tendon will show that the tip of the needle will point proximally upon passive dorsiflexion of the ankle [76] (Fig. 13.14).

Maffulli [77] in a prospective study in 1998 noted that the Thompson test is highly reliable in acute AT ruptures after applying the test on 174 patients. However, the same reliability cannot be expected in chronic AT rupture [72] as ruptured tendons at this point may have adhered to surrounding structures leading to a weak plantar flexion response as noted by Thompson and Doherty [75]. Maffulli [77] also proposed that

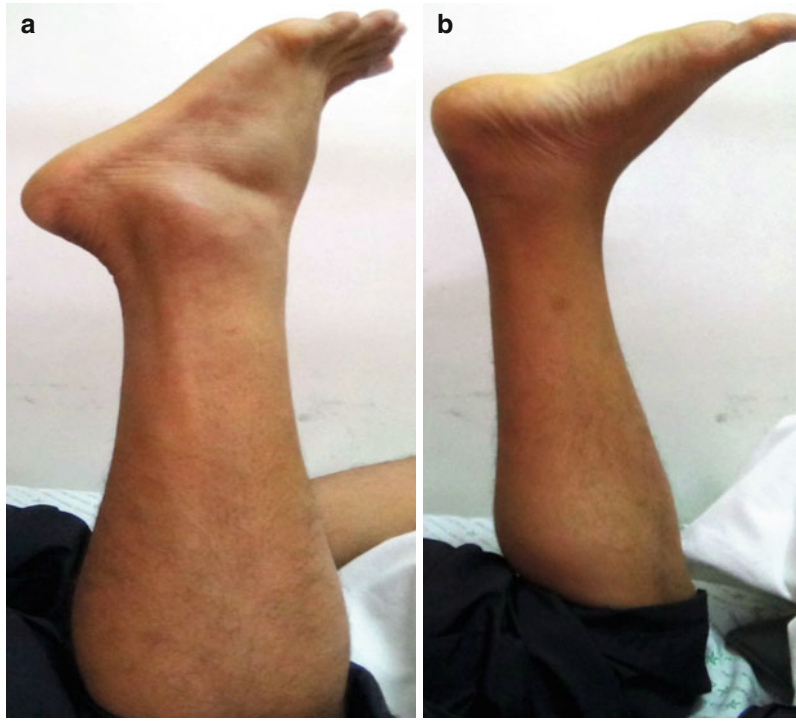


Fig. 13.13 **Matle's Test:** (**a**) Plantarflexion of the ankle upon active flexion of the knee of an intact Achilles tendon. (**b**) Absence of plantarflexion of the ankle upon active flexion of the knee in a torn Achilles tendon

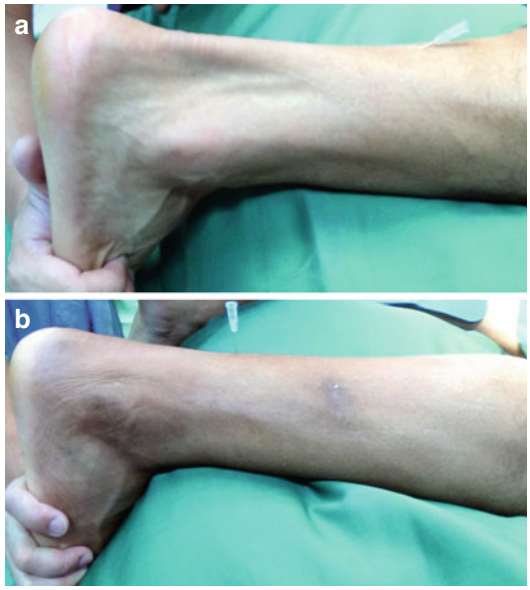


Fig. 13.14 O'Brien's Test: (**a**) Needle moving superiorly upon passive dorsiflexion of the ankle in an intact Achilles tendon. (**b**) Needle maintained its position upon passive dorsiflexion of the ankle in a torn Achilles tendon

the plantar flexion of the foot may also be caused by the reorganization of the hematoma overtime leading to some continuity within the chronic ruptured tendon.

Imaging Studies

A standard radiographic lateral view of the foot can be used in the diagnosis of either an acute or a chronic AT rupture. Arner et al. [60] found that through plain radiograph, the distal segment of the tendon will be deformed due to loss of AT tone in the acute setting. The pathognomonic sign of a loss of the normal configuration of the Kager's triangle can also be seen [3], and the Toygar sign (Fig. 13.15), which involves measuring the angle of the curved posterior skin surface, will reach 130–150° indicating a ruptured AT. In the chronic setting, a lateral radiograph may show an avulsion fracture of the calcaneus [71] or a calcification at the proximal stump of the tendon [78] (Fig. 13.16).

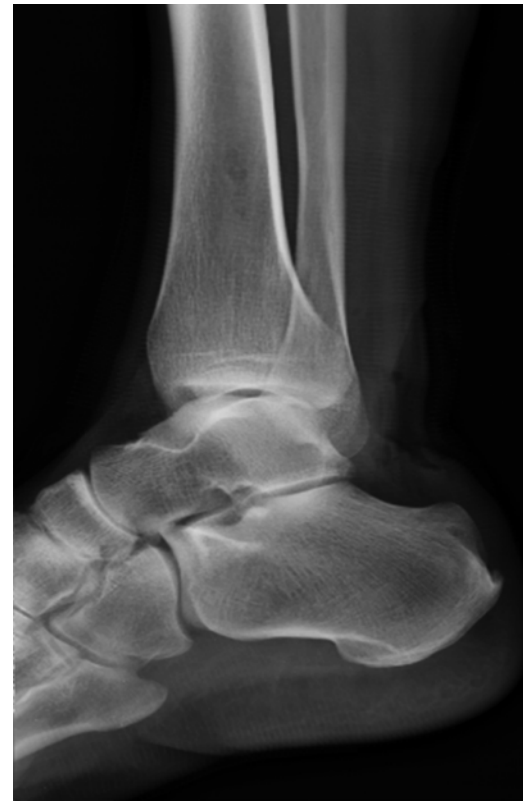


Fig. 13.15 Lateral x-ray view of the Kager's triangle and the presence of Toygar sign

According to Bleakney and colleagues [79], the primary imaging modality for the diagnosis of an AT rupture is ultrasonography. A real-time high-resolution ultrasound of a ruptured AT may reveal an acoustic vacuum at the rupture site with the presence of thick irregular edges on the tendon ends. However, the disadvantage of an ultrasound is that it is highly user dependent and one needs significant experience to correctly interpret the images [73] (Fig. 13.17).

A magnetic resonance imaging (MRI) can also be used in either an acute or chronic AT rupture and can properly estimate the measurement of the gap formed between the ruptured ends [73]. In an acute AT rupture, T1-weighted images will show disruption of the signal within the tendon, while T2-weighted images of the ruptured site will show retraction of torn ends and a generalized increase in signal intensity, which is due to the edema and

hemorrhage. In the chronic setting, T1-weighted images will show a low-intensity signal at the ruptured area and alterations in T2-weighted signal (Figs. 13.18, 13.19, and 13.20).

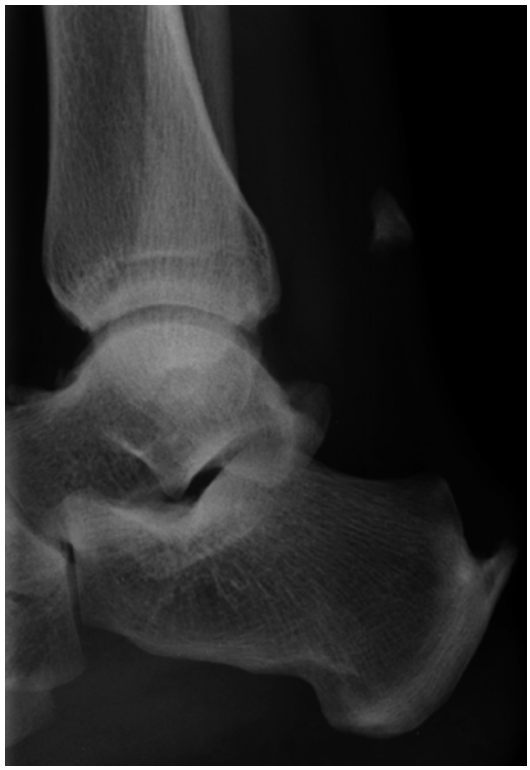


Fig. 13.16 Avulsion fracture at the insertion of the Achilles tendon

Both ultrasonography and the MRI are useful in differentiating between a partial and a complete tear of the AT, and both can also give a more detailed evaluation of the tendon in the chronic setting. However, the advantage of ultrasonography over an MRI is that it is less expensive and easy to use and allows dynamic imaging of the injured tendon.

Treatment of Acute AT Rupture

At present there are a lot of treatment options for an acute AT rupture, and these can be broadly

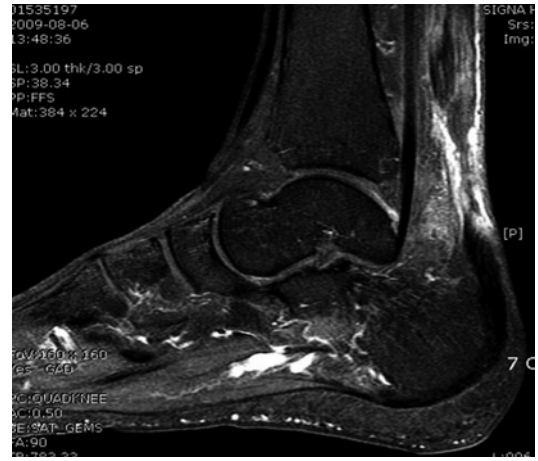


Fig. 13.18 Magnetic resonance image of a 72-year-old male with a partial tear of an Achilles tendinitis

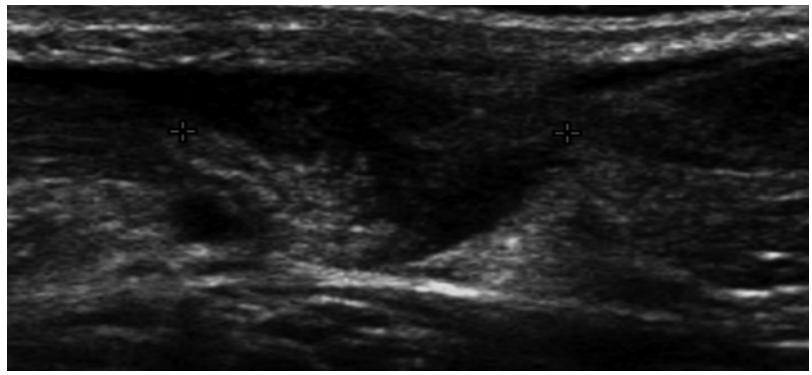


Fig. 13.17 This is a case of a 67-year-old male patient who presented with a left ankle pain after slipping from a height. On physical examination there was a presence of dimpling along the AT and was positive for the Thompson test. Ultrasound showed that there was a complete tear of the AT measuring 1.7 cm between the tendon ends located 5 cm proximal to the os calcis. The tear was then treated with the Achillon system

classified into operative and nonoperative management. The operative treatment can either be an open surgical approach or a percutaneous repair, while functional bracing or immobilization can be utilized as a nonoperative approach [3]. However, the choice of treatment will depend on the preference of both the surgeon and the patient as well as the patient's age, occupation, and the level of sporting activity.

Most investigators recommend the use of the nonoperative treatment option especially for nonathletes [54, 80]. However, the risk of rerupture is more commonly seen in cast immobilization because of the increasing length of the Achilles tendon which in turn reduces its strength against deforming forces after removal of the cast [80]. For the past two decades, open surgical treatment has been the method of choice for acute AT rupture in both athletes and young patients as well as for cases of delayed ruptures [80]. Though the open surgical techniques significantly reduce the risk of rerupture, it is well known that these procedures generate high rates of complications such as wound healing problem. Based on present studies, the percutaneous suture techniques have a more favorable outcome as they are simple and use minimally invasive techniques, slightly lower incidence of complications, and with identical functional recovery as compared to the open approaches [81].

Cast Immobilization/Functional Bracing

At present, the most common nonoperative protocol is to put the affected extremity on a below-knee plaster cast with the foot in gravity equinus position for 4 weeks. This will then be changed with the foot in a more neutral position

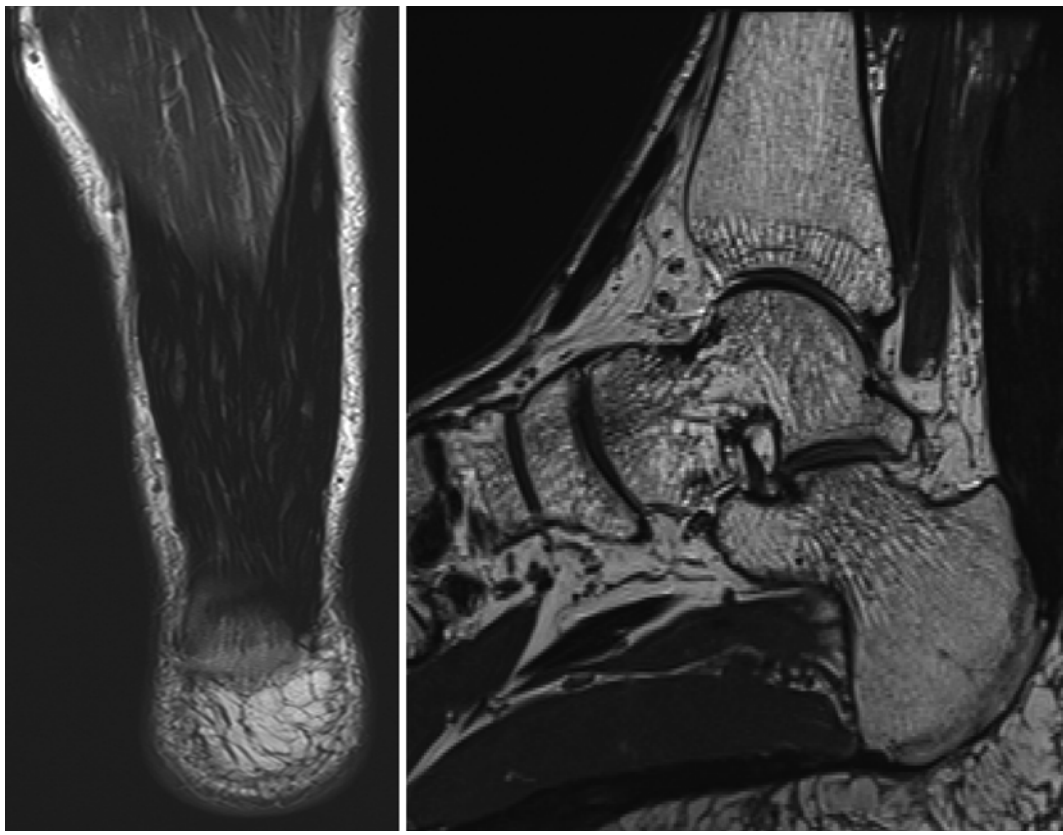


Fig. 13.19 MRI shows a case of a 54-year-old male with xantoma of the AT. He was treated conservatively with observation and medications for endocrine disorder

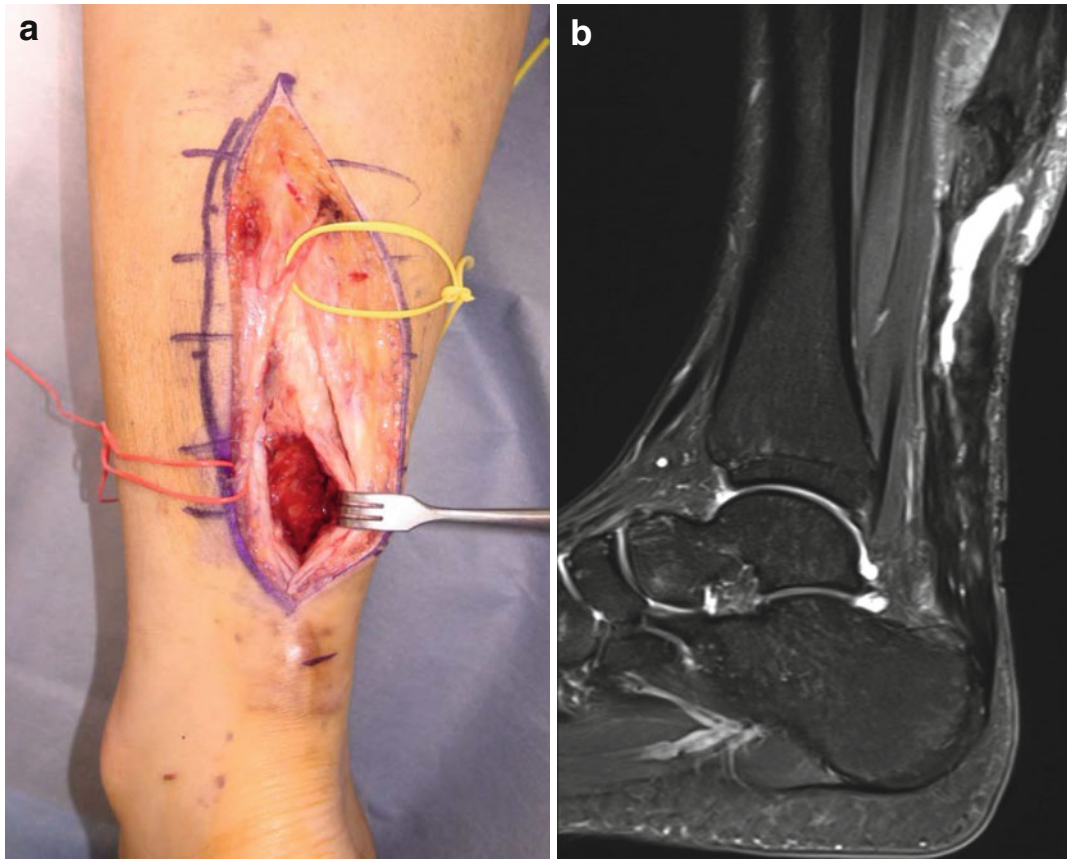


Fig. 13.20 (**a**) This is a case of a 24-year-old male who was initially diagnosed to have a partial rerupture of a previously repaired left AT (3 months postoperative). (**b**) MRI showed that the rerupture occurred proximal to the previous operative site

for another 4 weeks. Transition to a walking boot with a heel lift and active range of motion exercises can then be done after the casting period [82]. Stein and Luekens [83] reported that with the use of this protocol, the clinical outcome is comparable with an operative treatment. However, studies by Lea and Smith [84] and Persson and Wredmark [85] showed a rerupture rate of 13 % and 35 %, respectively, when this treatment option was used. The use of a below-knee cast has shown that the gastrocnemius muscle, which is biarticular, was still able to move and could have an effect on the healing tendon [86]. It was also seen that with immobilization, the muscle morphology and physiology were profoundly altered. This effect does not only influence the gastrocnemius muscle but also the soleus muscle, which contains a high proportion of type I muscle fibers that are susceptible to atrophy if immobilized.

Recently, two studies have shown that with the use of a functional bracing protocol, which combines early motion and early weight bearing, detrimental alterations in muscles can be prevented and have a more favorable influence in the maturation of collagen fibers in the healing tendon [70, 87]. With early weight bearing, increase in strength of the healing tendon is postulated as this is due to the fibroblasts and collagen fibers within

the tendon gap reorienting themselves along the long axis of the AT in response to the mechanical stress. McComis and colleagues' [88] study of 15 patients using functional bracing were able to achieve good results and concluded that such protocol in selected cases can be a viable alternative in the treatment of acute AT ruptures.

Comments
It must be emphasized that in treating acute Achilles tendon rupture by nonoperative functional bracing and early weight bearing, elongated tendon healing must be avoided with strict follow-up utilizing ultrasonography. Elongated healing leads to plantar flexion weakness in later stage, which may be difficult to address.

Open Surgical Repair

Open surgical repair remains to be the gold standard in the management of an acute AT rupture, and with the advent of more advanced postoperative rehabilitation protocols, more surgeons are encouraged to apply this technique in their patients [53]. And due to the excellent results of surgical repair such as low rerupture rates and increased tendon strength, many athletes return to their preinjury physical activities at a much faster rate. Comparative studies have showed that operative treatment has a rerupture risk of 3.5 % against 12.6 % following nonsurgical treatment.

End-to-End Repair: Surgical Technique

The patient is placed on the operating table in a prone position. The affected extremity as well as the contralateral leg can be prepped and draped for comparison. A posterior-medial midline incision (approximately 6–10 cm) is placed over the Achilles tendon while carefully protecting the sural nerve. Dissection is carried down through the subcutaneous tissue and fat until the crural fascia. The paratenon and the crural fascia are then carefully incised and reflected to expose the torn tendon. The paratenon should be properly identified to ensure anatomic layered closure upon closing the skin. Once the torn tendon is exposed, ruptured ends are identified. Stretching for a short period could also be done for the proximal portion prior to approximation of both ends. If adequate length is achieved, end-to-end reapproximation is carried out with the foot in equinus position; otherwise, other options like tendon lengthening, turndown or rotational flap, and even transfer procedures can be done at this point, but the authors never had to perform such procedures.

Simple end-to-end repair is carried out using either the Bunnel, Kessler, or Krackow technique. However, studies have shown that the Krackow technique is more superior in biomechanical and cadaveric models and has an advantage of allowing four threads (double Krackow technique) across the rupture site if desired [89] (Fig. 13.21a). Plantaris coverage can be performed to prevent the repaired tendon adhesions (Fig. 13.21b). Anatomic layered closure is then done upon conclusion of the procedure.

Platelet-rich plasma (PRP) has also been used as an augmentation tool in the repair of acute AT rupture. However, randomized control trials have shown that PRP is not useful in the management of these cases and even in chronic AT tendinopathies [90].

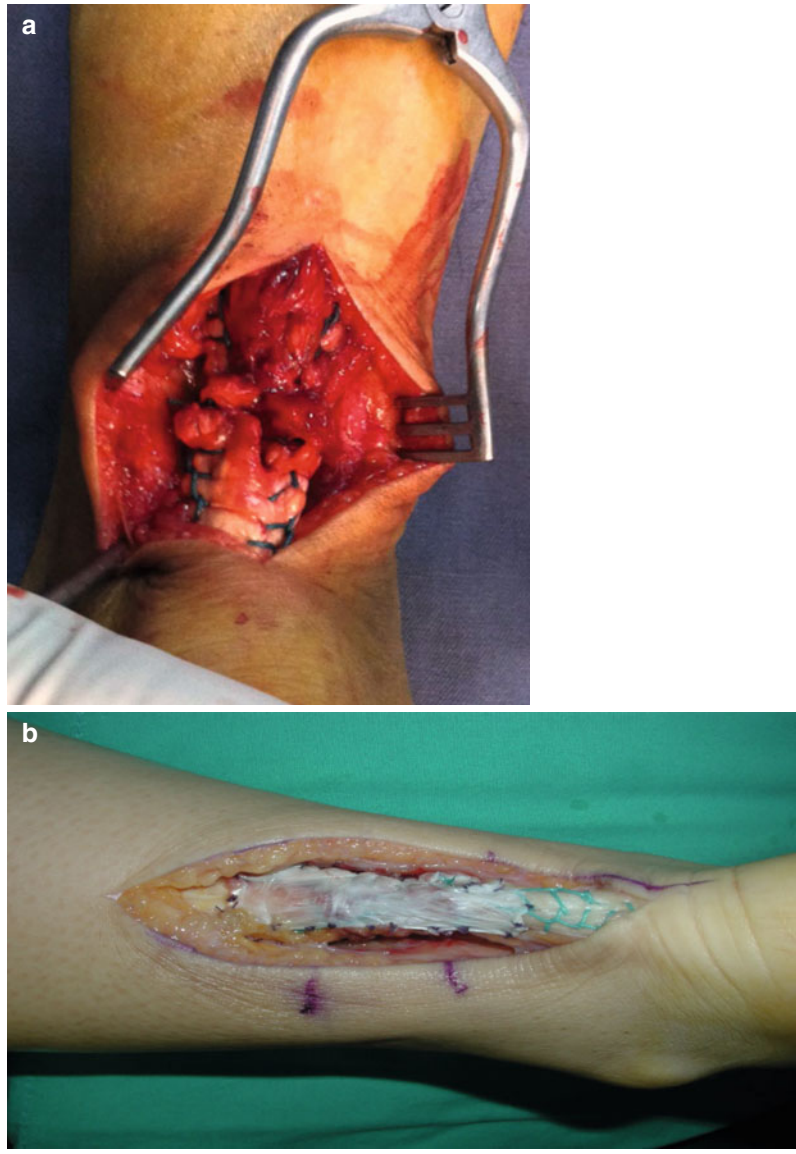
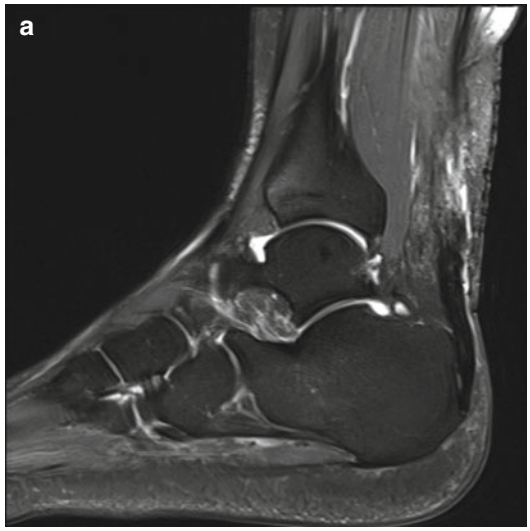
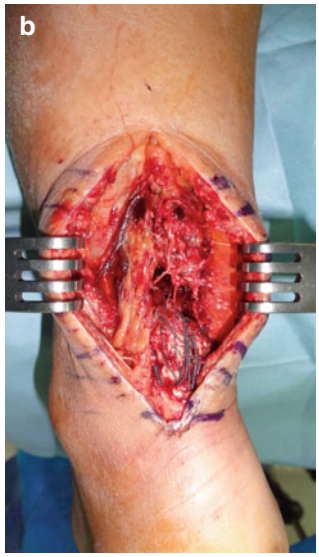
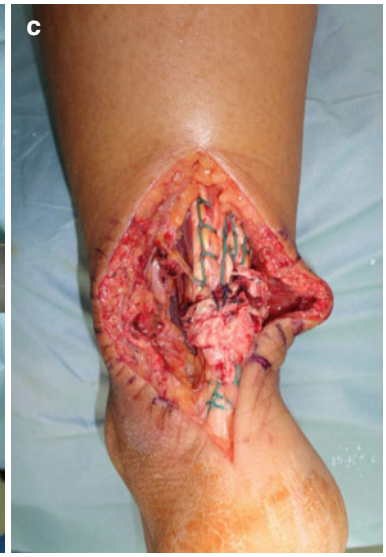


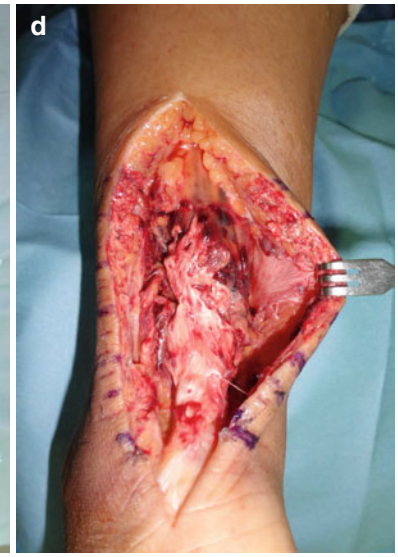
Fig. 13.21 (**a**) End-to-end repair of AT rupture using a Krackow suturing technique, (**b**) Plantaris tendon coverage over Krackow suture

Case 13.4 (**a**, **b**) A 35-year-old female had rerupture of the AT 10 days after slipping. The tendon ends were shredded and the sutures were found to be totally torn. (**c**) The previous suture materials were removed, and the tendon rupture was firmly repaired with double Krackow suture technique using a 1.0 Ethibond and (**d**) covered with the plantaris tendon for the prevention of adhesion. Although the author prefers mini-incision indirect repair using an Achillon as primary repair, it was not appropriate for this case and not recommendable for other redo-repair situations.









Percutaneous Repair

The first percutaneous repair for the management of an acute AT rupture was first introduced by Ma and Griffith in 1977 using the abovementioned technique [91]. In their study, they were able to report no reruptures but with two minor noninfectious skin complication. FitzGibbons and colleagues [92] in 1993 were also able to report no rerupture and skin infections, but there was a reported case of sural nerve injury. However, in recent reports, sural nerve entrapment as high as 13 % [93] and higher rerupture rate have been described. A newer technique was later introduced in 2005 by Webb and Bannister [94] using 3 midline transverse incision (2.5 cm) over the posterior aspect of the AT which produced no rerupture or sural nerve injury after a 35-month follow-up.

Mini-Open Indirect Repair (Achillon System)

We almost routinely treat the acute AT total rupture patients with mini-open indirect repair using the Achillon system. Unless the Achilles tendon has a longitudinal type of tear, which cannot withhold the transversely inserted 3 lines of sutures, other techniques are utilized.

Mini-open technique has many advantages. Since it makes use of a small incision of about 2–3 cm, less scar with better cosmesis and less morbidity with less discomfort are expected. Therefore, the postoperative wound complication of dehiscence or infection is infrequent. The indirect suture fixation is also quite stable that rehabilitation in the angle-adjusting boots with tolerable weight bearing is possible. However, the indirect repair by Achillon system is less powerful than the Krackow technique that when the AT has a previous history of marked Achilles tendinosis, successful tendon healing cannot be expected with the Achillon due to delayed healing. Thus, a more powerful suture fixation with double Krackow technique is selected.

The mini-open technique using the Achillon system (Newdeal, Lyon, France) showed decreased wound complication due to a smaller incision [95]. With the use of the Achillon system, Rippstein and Easley [96] were able to report early postoperative range of motion and earlier return to sporting activities in their patients. Comparing the Achillon system against the Ma and Griffith technique, Cecarrelli and colleagues [97] were able to show similar results in the time taken to return to sports or work and similar AOFAS score values.

Operative Technique: With the patient prone and under general anesthesia, the defect in the ruptured tendon is palpated and the incision site is marked. After applying a tourniquet on the thigh of the injured leg, a longitudinal incision is made at the center of the soft spot of the tendon and extended to about 2–3 cm. Both stumps of the ruptured tendon are then identified next to the incision of the sheath. Gentle continuous traction is then applied to the proximal stump of the ruptured tendon, and the Achillon® device is introduced maximally through the incision between the tendon and the paratenon sheath. Three no. 2 Ethibonds (EB; Ethicon, Inc., Somerville, NJ) are then placed through the instrument guide step by step. The instrument guide is then withdrawn, and the distal stump is then managed in the same manner. Suture pairs are then tightened in pairs such that both ends of the ruptured stumps meet correctly under proper tension with the ankle in 20° of plantar flexion. Additional 1.0 Vicryl sutures are then placed to reinforce the reconstruction site depending on the characteristics of the ruptured tendon. Finally, the tendon sheath and skin are then closed by layers (Fig. 13.22a–f).

Postoperatively, patients are immobilized with a non-weight-bearing short leg splint in approximately 20° of plantar flexion for the first week. This is then changed to a walker boot with the ankle in equines position accordingly, and partial weight bearing is allowed as tolerated. The angle is gradually changed to neutral at postoperative 4–6 weeks. The boots are maintained for 6–8 weeks postoperatively, after which an ankle strap brace is applied for 2–4 more weeks.

Comments

Our primary surgical option for acute Achilles tendon rupture is mini-open suture with Achillon system. It is functionally quite stable with low morbidity and cosmetically superior. However, in some selected cases, Achillon indirect sutures do not hold the strength and is changed to Krackow suture technique.

Treatment of Chronic AT Rupture

The choice between operative and nonoperative management in patients with chronic AT ruptures will depend on the overall health status of the patient, assessment of risk factors for healing, and the patient's level of activity [73]. Nonoperative treatment is usually indicated for patients who have associated comorbidities like peripheral vascular disease, those who have a relatively sedentary lifestyle that can accommodate with the loss of strength of the Achilles tendon, and for those who do not have any significant functional deficits and are able to perform their activities of daily living [53, 71]. By using pain and Achilles function as a criteria for an operative management in chronic AT ruptured patients, Christensen [98] was able to report 90 % improvement in the operative group and 70 % in the nonoperative group. He also observed that in the nonoperative group, only 56 % were able to return to their preinjury level, while in the operative group it was 75 %.

Myerson [53] suggested that the use of a molded ankle foot orthosis (AFO) with or without a hinge may be useful in patients with chronic tears as these AFOs are tolerable and add stability to the leg of the patients. Padalinam [71] also suggested the use of a structured rehabilitation program to strengthen the remaining flexor muscles in order to compensate for the Achilles function loss for these patients. However, most authors still suggest the use of an operative technique in the management of chronic ruptures [22, 66, 78] with restoration of the normal length and tension of the Achilles tendon as the main objective [57, 99]. There are various operative techniques for such cases; however, there are only a little to no data at all which compare the

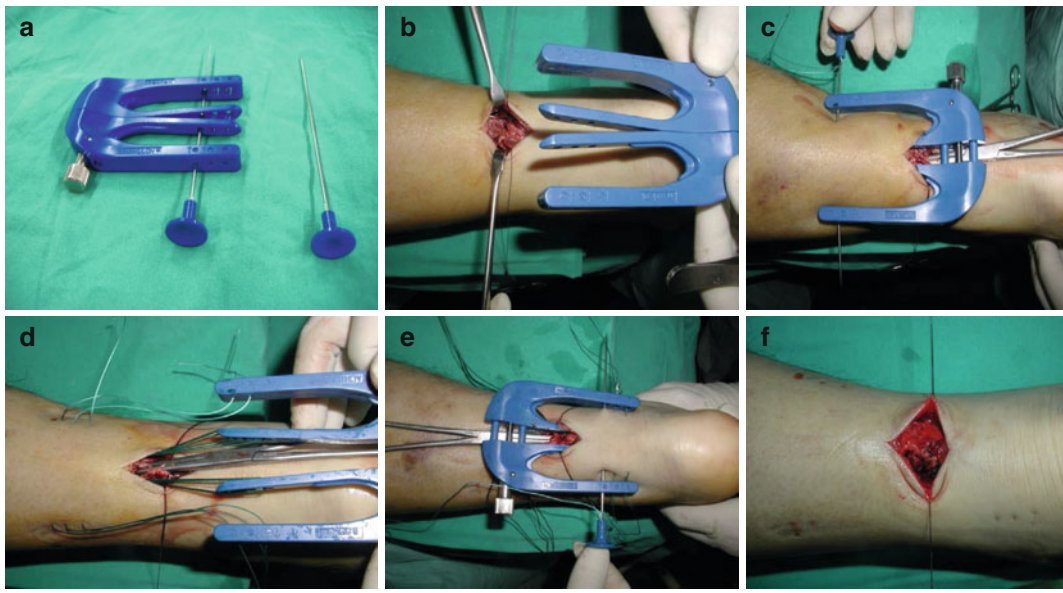


Fig. 13.22 (**a**–**f**) Surgical procedure of indirect repair of an acute Achilles tendon rupture by mini-incision technique utilizing the Achillon system

Table 13.6 Treatment recommendations for chronic Achilles tendon ruptures according to Myerson [53]

<2 cm gap	2–5 cm gap	>5 cm gap
End-to-end repair	V-Y lengthening	FHL transfer
Posterior compartment fasciotomy	FHL transfer if the gastrocnemius muscle is not healthy appearing	Can add V-Y advancement to transfer

different techniques [71]. Tissue expanders can also be used to allow tension-free wound closure in some cases [58] (Table 13.6).

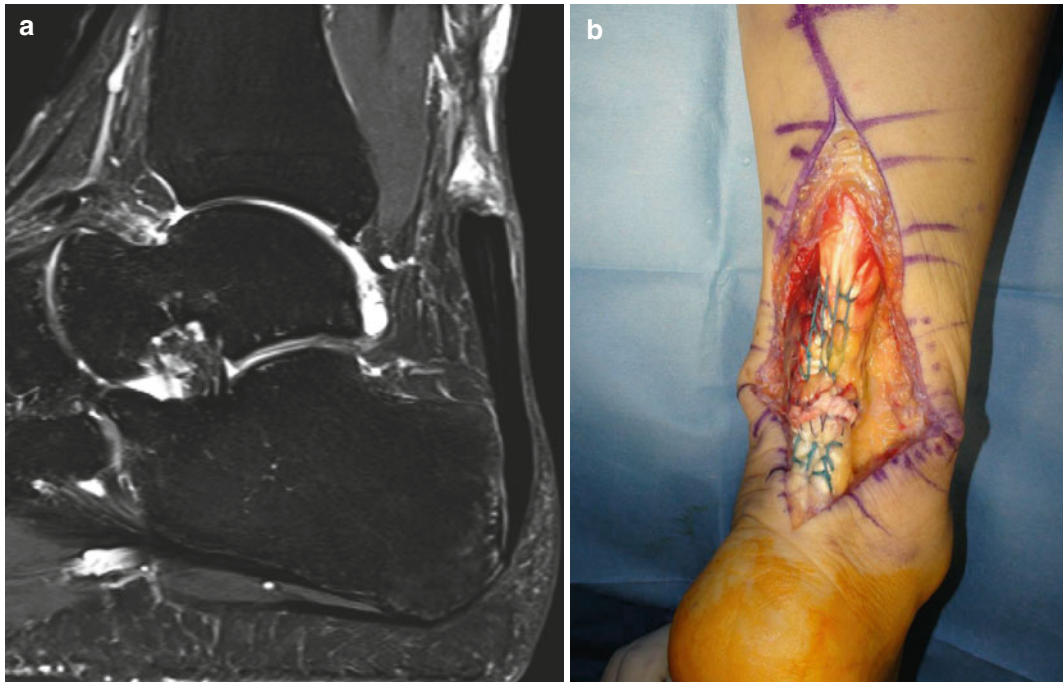
Tendon Insufficiency

As previously discussed, some patients will have no demonstrable gap within the ruptured tendon. In fact, what can be observed is lengthening of the AT with loss of its mechanical efficiency and power. And upon exploration, there is evidence of extensive scarring between the ruptured ends and tendon sheath [53].

Treatment options for such cases include a z-shortening with placement of the ankle in a slightly greater equinus than the contralateral side as described by Mafulli and Ajis [73]. Porter and colleagues [100] also reported their technique wherein upon freshening the tendon ends of 2–4 mm of fibrous scar tissues and prior to proximal release, they found an average gap of 3–5 cm between tendon ends. The tendon ends were then apposed while the ankle is in 20–30° of plantar flexion and the existing fibrous scar was used as local reconstruction tissues. Incorporation of the fibrous scar during the procedure was supported by the biopsy result of the scar tissues, wherein they were able to observe the presence of vascular granulation tissues within the rupture site. Yasuda and colleagues [69] concluded that the technique by Porter can be used if on preoperative MRI the T2 or gradient echo images showed the tendon ends to be thickened and fusiform in shape with diffuse intratendinous high-signal changes; otherwise, other reconstructive methods should be considered.

If a gap does exist within the ruptured tendon, mobilization can be used to appose the tendon ends. Gaps, which are less than 2 cm, can be repaired with simple end-to-end repair [58, 73]. Slightly larger gaps can be closed by placement of a Krackow stitch, and manual traction for several minutes can help stretch the proximal musculature prior to apposition. Bluntly freeing the tendon ends from the surrounding soft tissues can also help to appose the tendon ends.

Case 13.5: Primary Repair of a Chronic AT Rupture (**a**) A 46-year-old male presented with a chronic AT rupture that had been neglected for almost 2 months. Preoperative MRI showed a ruptured AT with a retracted proximal end. (**b**) Although being a chronic rupture for 2 months, it was possible to bring both ends together and be repaired with a double Krackow technique.



Turndown Techniques

Bosworth's technique [71] starts with a posterior longitudinal midline incision from the calcaneus to the proximal one third of the calf. Upon exposure of the ruptured tendon, the scar is excised between the ends. A strip of tendon (1.5 cm wide and 22.5 cm long) is freed from the median raphe of the gastrocnemius muscle while leaving it attached just proximal to the site of rupture. The strip of tendon is then distally turned and passed transversely through the proximal tendon and anchored in this position with absorbable sutures. The strip is then passed distally and then through the body of the distal tendon. Prior to bringing the strip back to the proximal end, the strip is passed again at the distal end from anterior to posterior. While holding the knee at 90° and the ankle in plantar flexion, the fascial strip is drawn tightly and secured with chromic sutures. The strip is then passed transversely through the proximal tendon and carried down distally and sutured to itself. The wound is then closed, and a long leg cast is applied with the knee in flexion and the foot in plantar flexion.

Christensen [98] reported 75 % satisfactory outcome using a similar technique wherein a 2 × 10 cm flap was formed from the proximal tendon fragment and was turned down to be used as a cover for defects in both acute and chronic ruptures. Rush [101] on the other hand used an inverted "U" from the proximal fascia and then sutured the ends together to create a tube. However, mild-to-moderate stiffness, weakness, and discomfort have been reported after the use of such techniques [101–103]. Studies have also shown that there was no significant functional difference among the various turndown techniques [104]. It was also noted in a study by Maffulli and Leadbetter [99] that the quality of the proximal stump is suboptimal to be used as a turndown flap and concluded that these techniques are using avascular autologous tissues to fill the gap between the ends of the ruptured tendon.

V-Y Tendon Advancement

A lazy "S" incision is started from the lateral aspect of the Achilles tendon insertion and ending at the medial part of the calf. Dissection is carried down up to the paratenon while identifying and protecting the sural nerve. The scar tissues are then excised from both tendon ends and the tendon defect is then measured with the knee in 30° of flexion and the ankle in 20° of plantar flexion. An inverted V incision is done through the proximal aponeurosis of the gastrocnemius with the apex at the central part and its arms at least one and a half times longer than the tendon defect. The flap or the proximal tendon is then brought down and approximated against the distal tendon with interrupted nonabsorbable sutures. The proximal part of the incision is then repaired in a Y configuration, while the paratenon is sutured together with interrupted nonabsorbable sutures. Closure by layer is then done, and the affected extremity is put in a long leg cast with the knee in 30° of flexion and the ankle in 20° of plantar flexion (Fig. 13.23).

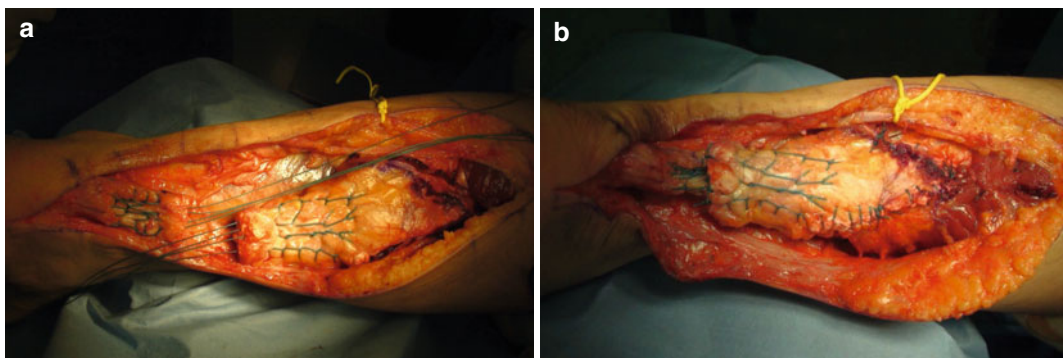
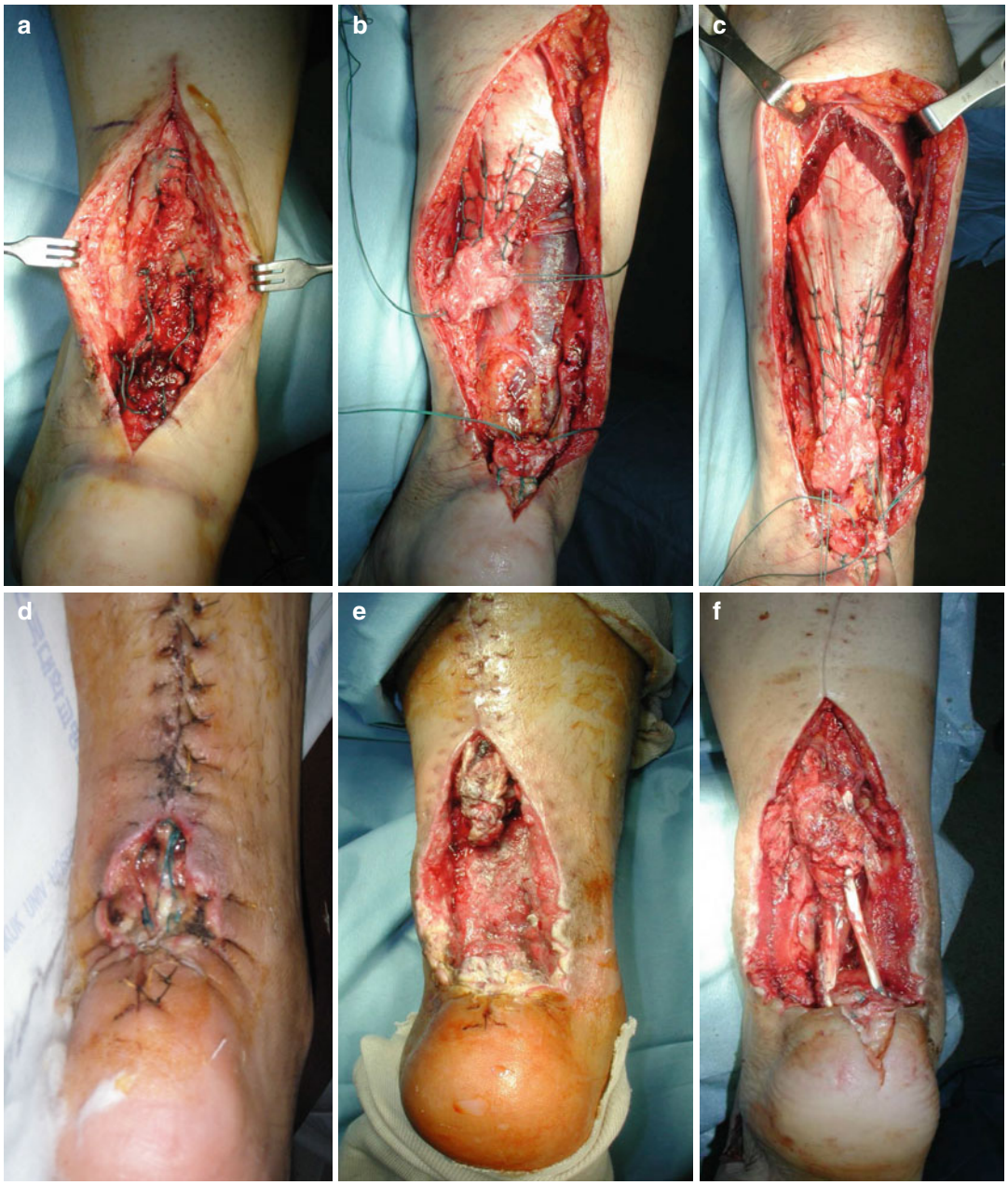


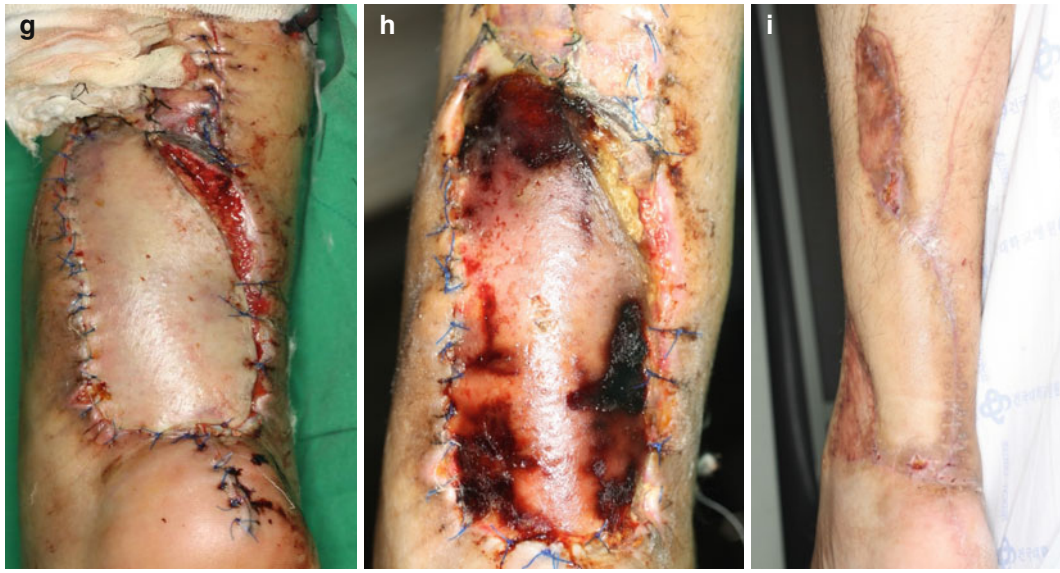
Fig. 13.23 (**a**, **b**) Achilles tendon advancement using the V-Y technique

Reports have shown that the V-Y advancement technique's advantage includes a healthy tendon-to-tendon apposition, less tension at the repair site, it allows intrinsic healing (increased elasticity, strength, and mobility), and sacrifice of other tendons are spared (tendon transfers or free tendon grafts).

The V-Y tendinous flap was first described by Abraham and Pankovich [103] in response to their poor experience with fascial turndown flaps and fascia lata transfers. They were able to report good results in four of their patients with three of them regaining full strength. They also recommended that the arms of the incision have to be extended through the aponeurosis and the underlying muscle tissue along the side of the flap, and sometimes it may be necessary to dissect the flap almost completely free for end-to-end repair to be obtained. Myerson [53] recommended the use of this technique if the gap measured up to 5 cm and even as long as 8–10 cm according to Leitner and colleagues [105].

Case 13.6 V-Y after for infected Achilles tendon. (**a**–**c**) This is a case of a 34-year-old male patient who had an AT rerupture 3 months after the primary repair due to a possible evidence of mild infection. Debridement and irrigation of the rerupture site was performed and was repaired with V-Y advancement and double Krackow suture. (**d**) However, 2 weeks post-op, the wound was found to be infected and caused dehiscence. (**e**) Aggressive repeated tendon debridement left a large Achilles tendon defect. (**f**) After infection control, the 7 cm defect was reconstructed with an FHL tendon transfer, and (**g**) the large soft tissue defect was covered with myocutaneous free flap. (**h**) There was marginal skin necrosis of the free flap at 1 week post-op for which debridement and skin graft were performed. (**i**) At 2 months post-op, the wound eventually healed and the patient actively participated in his rehabilitation program from thereon.

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Tendon Transfers

Chronic AT rupture can also be reconstructed with the use of other tendons, and it was Wapner and colleagues [106] who popularized the use of the flexor hallucis longus (FHL) tendon in these cases. In their seven patients using the FHL tendon, they were able to achieve three excellent, three good, and one fair outcome. All subjects were noted to have some loss of flexion in the interphalangeal joint of the hallux, but these were unnoticed by the patients.

FHL Tendon Transfer Technique

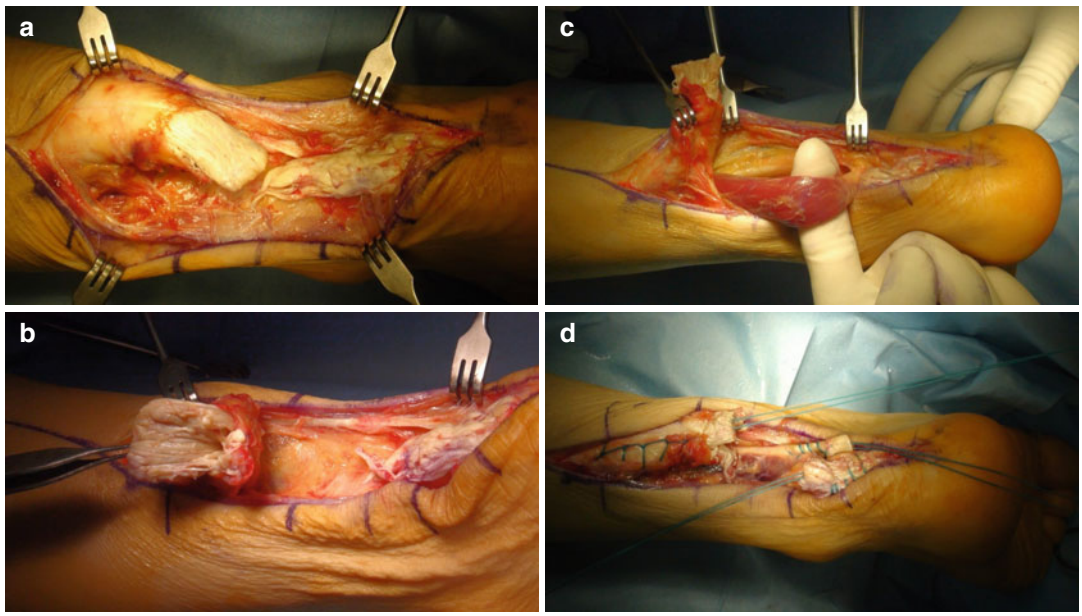
The FHL tendon is harvested by making a longitudinal incision at the medial border of the foot just above the abductor muscles and extending from the navicula up to the first metatarsal head. Dissection is then carried down sharply, and the abductor muscle together with the flexor hallucis brevis is reflected plantarward. The FHL is then identified and isolated against the flexor digitorum longus (FDL). The FHL is then divided as far distally as possible and tagged. The distal end of the FHL can then be sutured together with the FDL with the toes in neutral position. A separate posteromedial incision is made 1 cm medial from the AT starting 2.5 cm below the calcaneal insertion up to the musculotendinous junction. The incision is then carried down sharply up to the paratenon creating a full-thickness flap. After removing the fibrous tissues within the gap, a longitudinal incision is done at the deep fascia of the posterior compartment. The FHL is then exposed from this incision and retracted from the midfoot. A transverse hole is then drilled distal to the insertion of the Achilles tendon halfway from medial to lateral. A second hole is also drilled vertically deep to the insertion and joined with the previous hole. The formed tunnels are then enlarged using a towel clip. The tagged FHL is then passed through this tunnel in a proximal to distal manner and then weaved through the distal tendon up to the proximal tendon until the full length of the harvested tendon is used. The weave is then secured with nonabsorbable sutures, while the paratenon is closed with absorbable sutures. The leg is then placed in a non-weight-bearing cast with the ankle in 15° of plantar flexion, and then after 4 weeks, the cast is replaced with a short leg walking cast with the ankle in neutral position which is to be worn for another 4 weeks.

Alternatively, the FHL can also be harvested from the posterior incision. However, reports have shown that by harvesting the FHL distal to the knot of Henry, through a separate incision at the midfoot, it will provide approximately 10–12 cm length of the FHL tendon or an additional 3 cm length [106] which could allow weaving of the tendon through both stumps and may enhance the repair. With the use of the FHL tendon for repair, reports have shown good to excellent results and with low morbidity, except for one study which reported injuries to either the medial or lateral plantar nerves during the FHL harvest [107].

Case 13.7: Achilles tendinopathy with tendon rupture (**a**) A 76-year-old male patient was admitted with an acute AT rupture. He initially had a chronic painful Achilles tendinopathy and was treated with multiple injections of corticosteroids. The tendon was found to be severely degenerated and devascularized. (**b**) This portion was then resected. (**c**) The flexor hallucis longus (FHL) was then identified and isolated. (**d**) Primary repair of the AT was performed using a Krackow suture technique and was augmented with an FHL transfer to the calcaneus.

The peroneus brevis (PB) tendon was also used to repair AT ruptures, and it was reported to produce 28 excellent and 2 good results in 30 patients [56]. Even with a modification of this technique, Turco and Spinella [9] were able to achieve similar results with most of their patients returning to their activities without significant functional limitations. Though there was a report of mild weakness in eversion strength (14.9 %), still there was no functional loss as compared with the normal contralateral side [56]. The FDL has also been used by Mann and colleagues [78] for the reconstruction of chronic AT ruptures and noted the following advantages like avoidance of host rejection, revascularization not needed, and similarity of the FDL to the Achilles with regard to biomechanical characteristics.

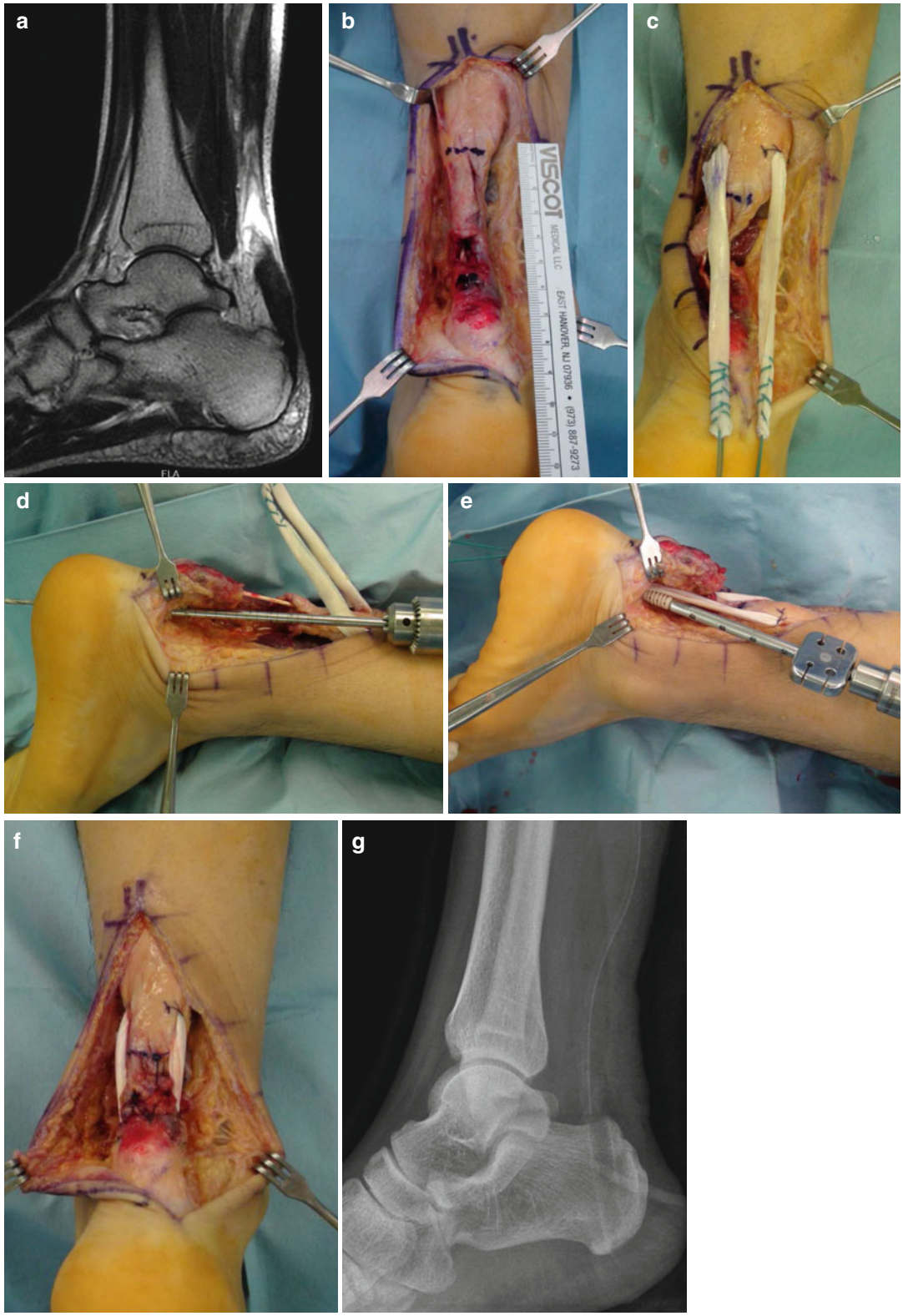
Research by Wapner and colleagues [106] has reported on the advantages of using the FHL tendon for the reconstruction of AT rupture against the PB and FDL tendon which includes the greater strength of the FHL against both tendon and avoidance of the neurovascular bundle at the lateral compartment due to the anatomic proximity of the FHL to the Achilles. It was also reported that the distal musculature of the FHL can provide some vascularity to the Achilles tendon as compared with the other tendons.



Free Tendon Transfer

The fascia lata has been used in the treatment of chronic AT ruptures, and as reported by Tobin [108], this technique produced good results with delayed wound healing in some of his patients. Others also reported satisfactory results with no problems with adherent scars with use of this tissue. Free gracilis tendon grafts has also been reported as an alternative, and results have shown that patients returned to work and resumed preinjury activities by 2 years with some patients experiencing superficial wound infections [99]. Achilles tendon allograft has also been used which can act as a scaffold for migrating host cells [109]. Advantages of the allograft include avoidance of morbidity from harvesting an autogenous autograft, decreased operative time, and excellent mechanical properties [109]. However, the potential risk of disease transmission and the high cost for procuring the allograft were seen as its disadvantages [71].

Case 13.8: Achilles Reconstruction with TA Allograft (**a**, **b**) A 29-year-old male presented with a chronic AT rupture for 1 month. He was previously treated for chronic Achilles tendinopathy for almost 3 years. Thompson test was positive, and his MRI showed a complete tear of the Achilles tendon 4.1 cm distal from the calcaneal attachment with a rupture gap of about 5 cm. (**c**) A tibialis anterior (TA) tendon allograft was whip stitched at both ends after approximating the estimated tendon lengths to be embedded in the calcaneus and passed transversely through the proximal stump. (**d**) A 7–9 mm drill hole was made at the calcaneus, just anterior to the AT insertion. (**e**) Both ends of the allograft tendon were pulled through the hole, and under submaximal tension, the 7 mm Bio-Tenodesis screw was inserted to stabilize the allograft. (**f**) The scar tissues at the ruptured ends were preserved and repaired. (**g**) A lateral x-ray showed the bone tunnel holding the TA tendon reconstruction of the Achilles tendon.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