Ultrasonography-guided Intervention in the Achilles Tendon and Plantar Fascia

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Abstract

Tendinopathy is very common in the foot and ankle. Achilles tendinopathy is a painful overuse injury that often occurs in athletes, especially those who participate in running and jumping sports. Plantar fasciitis is the most frequent cause of plantar pain in the adult heel. Initial treatment of these conditions is conservative. However, in some cases symptoms only improve slowly, and many cases are intractable. When conservative management fails, ultrasonography guided injections are indicated. We discuss the main interventions performed in the foot and ankle for Achilles tendinopathy, retrocalcaneal bursitis, and plantar fasciitis. We describe the different agents that can be used and the various ultrasonography-guided procedures that offer technical and practical information to improve daily clinical practice.

Keywords

- ultrasonography-guided procedures
- ankle
- Achilles tendon
- foot
- plantar fascia

Achilles Tendon

Background

Achilles tendinopathy (AT) is a painful overuse injury that is extremely common in athletes, especially those who participate in running and jumping sports. However, AT is not purely an athletic injury; 65% of injuries diagnosed in a general practice setting are not sport related.[1]

The terminology used to describe and diagnose tendon injuries has changed in recent decades.[2] The term "tendinopathy" describes a *clinical syndrome* in which two components are present: pain and a histologic diagnosis of tendinosis. The term "tendinosis" indicates degenerative changes in the thickness of the tendon. It is not correct to use the term "tendinitis" because there is no inflammatory response in chronic tendinopathy.[3]

Achilles tendon injuries are classified according to the location of the lesion: insertional tendinopathy (20–25% of the injuries), midportion tendinopathy (55–65%), and proximal musculotendinous junction (9–25%) injuries.[4]

Reactive tendinopathy is defined as symptoms lasting 6 weeks, whereas a chronic tendinopathy lasts $\geq$ 3 months.[5]

Macroscopically, tendinopathy results in enlargement, disruption of the fibrillar pattern, and an increase in tendon vascularity. Histopathologically, there is evidence of disorganized proliferation of tenocytes, disrupted organization of collagen fibers, an increase in the noncollagenous matrix, and neovascularization. There is usually no evidence of inflammation, but the cause is considered a failed healing response.[6]

The exact pathophysiology of AT is still unknown; it is thought to have a multifactorial origin.[5] However, most authors believe repetitive microtrauma from unusual or excessive mechanical loading is a causative factor. The most common cause of tendinopathy in athletes is excessive loading with inadequate recovery between training sessions. At the Achilles tendon insertion, compressive forces on the Achilles tendon and calcaneus from footwear or activities that place the ankle in dorsiflexion (e.g., uphill running) or an anatomical abnormality (e.g., Haglund's deformity) may contribute to the development of pain. Enthesitis is the unifying clinicopathologic feature for all seronegative spondyloarthropathies.

The diagnosis of AT is usually made using clinical findings.[5] Pain and reduced function are the primary symptoms.

Recovery from AT can take a year or more.[1] Treatment initially is nonsurgical. The first advice for AT often consists of temporarily adjusting or stopping the sports load that probably caused the injury and wait and see.[5]

If there is still no improvement in patient symptom after 3 months of patient education, structural exercise therapy, and

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following loading advice, additional treatment options should be considered.[7]

Various additional treatments have been described:

- Extracorporeal shockwave therapy.
- Injection therapies, including injections with corticosteroid (CS), polidocanol, lidocaine, autologous blood, platelet-rich plasma (PRP), stromal vascular fraction, hyaluronic acid, prolotherapy, high-volume image-guided injection (HVIGI), or intratendinous needling.[5]

If > 6 months of basic management and additional therapies do not improve symptoms, surgery should be considered.[1,5]

The use of imaging-guided injection treatment in recalcitrant cases is controversial. A 2015 Cochrane review concluded there is insufficient evidence from randomized controlled trials to support the use of injection therapies.[8] However, different kinds of treatment have been described with promising results.

Imaging-guided interventional procedures in AT can be divided into two subcategories. The first are procedures in which some substances are injected into the retrocalcaneal bursa or pre-Achilles fascia, such as anesthesia, steroids, or (HIVGI). The second are procedures performed directly on the tendon, such as dry needling, prolotherapy, sclerosing polidocanol injection, or injection with PRP.

HVIGI involves a large volume of saline and local anesthetic with or without steroid injected into the interface between the midportion of the Achilles tendon and peritendinous tissue and Kager's fat pad. The high volume is thought to have a mechanical effect on neurovascular ingrowth and adhesions between the tendon and peritendinous tissue but may also have effects on pain and local sensitization.[9–12]

HVIGI seem to be effective at treating AT in the short term. Boesen et al[9] demonstrated that treatment with HVIGI or PRP in combination with eccentric training in chronic AT seems more effective in reducing pain, improving activity level, and reducing tendon thickness and intratendinous vascularity than eccentric training alone. HVIGI may be more effective in improving outcomes of chronic AT than PRP in the short term (6 and 12 weeks) but not in the medium term (24 weeks) in which the positive effect of treatment with PRP persists.

PRP is the cellular component of the plasma obtained by centrifuging whole blood, resulting in a higher platelet concentration, and contains various growth factors that have the potential to influence tissue regeneration.

Treatment of chronic tendon injuries by needling originated in veterinary medicine, in a practice known as "pin-firing,"[13] in which veterinarians used a firing iron to burn soft tissue and thus transform a chronic tendon injury into an acute one, producing an inflammatory state. This cruel method of treatment is no longer used. However, the idea of changing a chronic nonhealing injury (such as occurs in tendinosis) into an acute condition that may have greater healing potential is the basis of PRP treatment.

The goal of PRP injected into areas of tendinopathy is to induce healing via cellular and humoral mediators.[9,14–18] The mechanism of PRP injections to treat chronic tendinopathy is believed to be a variety of growth factors, such as platelet-derived growth factor, transforming growth factor, and insulinlike growth factor, that promote a healing response. One of the main advantages is that PRP is autologous; therefore, it has an excellent safety profile with almost no side effects.[19] Some studies have reported promising results when examining the effect of PRP in chronic tendinopathies.[16,20–25]

In a study comparing PRP with blinded sham saline injections, de Vos[26] and colleagues reported significant improvement in both saline and PRP groups that slightly favored the PRP subjects but not by a statistically significant margin. However, saline injection may not be the best control group because it is likely active for tendinopathy; injecting saline into the tendon alters the pressure–volume relationship in a given space, thereby disrupting pathologic vascular and neural ingrowth. Injections into the tendon may induce bleeding, which in turn releases certain growth factors that stimulate the healing process with a similar mechanism of action as PRP.

*(CS) injection*s are a manufactured version of hormones normally produced by the adrenal glands. Steroids reduce redness and swelling in the nearby area. The infiltration with steroid can help relieve pain and stiffness.

Local CS injection is an effective and safe modality to treat plantar fasciitis of various causes.[8] However, only a few studies have evaluated this modality for the treatment of AT. Some authors have indicated that ultrasonography (US)-guided local CS retrocalcaneal injection is an effective and safe modality for refractory Achilles enthesis in patients with insertional AT and leads to reversion of acute changes at the enthesis site.[27] CS bursal injections rapidly help settle pain and swelling in tissue, restoring function and allowing individuals to participate in their rehabilitation.[28]

Side effects of steroid injection include local site infection, tendon rupture, skin depigmentation, and fat pad atrophy in cases of plantar fascia injections.[29] Tendon rupture, the most serious complication, is relatively rare with a reported incidence of 2.5 to 6.7% for injection for plantar fasciitis and is more associated with recurrent and blind palpation-guided injections. US guidance significantly decreases complication rates.[30]

Often combined with local anesthetic, these injections are described as having a diagnostic (from the anesthetic) and therapeutic (from the steroid) element. Commonly used injectable steroids in musculoskeletal medicine include triamcinolone acetonide, methylprednisolone acetate, and betamethasone. The choice depends on local availability and the site of treatment; for intra-articular injections, triamcinolone or methylprednisolone is typically used.[31] For the infiltration of the retrocalcaneal bursa CS injection, we prefer to use betamethasone.

To date, no definitive study has compared the efficacy of different treatment options in AT, and therefore no definitive superior method has emerged. Therefore, decision on technique remains a personal decision based on unique patient factors, imaging findings, and radiologist expertise/comfort level.

We divide the treatment of AT tendinopathy into three parts: HVIGI, PRP, and CS US-guided injection.

Recommended Clinical Indications

Clinical indications for HVI tendon stripping for tendinopathy (1) PRP injections for degenerative tendon disease with intrasubstance tearing; or (2) retrocalcaneal bursa injection for retrocalcaneal bursitis and Achilles enthesitis.

Pretreatment US Tendon Evaluation

The Normal Achilles Tendon

The Achilles tendon is the largest and strongest tendon in the body that connects the calf muscles to the heel. It is ~ 15 cm in length and begins in the mid-lower leg. The tendon is formed from the gastrocnemius and soleus muscles and inserts into the calcaneus. The tendon descends to its insertion on the calcaneus, an enthesis composed of fibrocartilage with direct meshing of the tendon fibrils into the marrow. The tendon rotates 90 degrees as it descends, with the soleus fibers twisting from anterior in the midcalf, to insert medially onto the posterior calcaneus, and thus the gastrocnemius fibers rotate from their posterior location to insert laterally into the calcaneus.[32] The Achilles tendon is covered by the paratenon; normally it is not visible on US. The paratenon is highly vascularized and thus is important in healing the Achilles tendon. Kager's fat pad (also known as the precalcaneal fat pad) refers to the fat within Kager's triangle, located in the posterior ankle joint, anterior to the Achilles tendon. The retrocalcaneal bursa is a synovial-lined structure occupying the space between the Achilles tendon and calcaneus. The superficial calcaneal bursa is located between the skin and the Achilles tendon insertion[33] (►**Fig. 1**).

High-resolution US demonstrates the dimensions and morphology of the tendon. The Achilles tendon is divided into the body, preinsertional zone (2 cm), and insertional zone. The fibrillary anatomical architecture can be appreciated as tightly packed thin echogenic lines on longitudinal scanning and echogenic punctate foci in the axial plane. The thin paratenon is usually seen surrounding the tendon as a slightly more echogenic border. The normal tendon shows no vascularity on color Doppler (►**Fig. 2**).

Paratenonitis

The inflammatory condition of paratenonitis is one of the most common findings in patients with a painful Achilles tendon. It is the first symptomatic stage of Achilles disorders and usually presents in cases of tendon degeneration. On high-resolution US, these changes appear as a thickened hypoechoic paratenon, mainly posteriorly. In the acute phase, fluid can be seen between the tendon and paratenon, along with an increased vascularity. The changes can extend into the local soft tissues around the tendon, including Kager's fat pad (►**Fig. 3**).

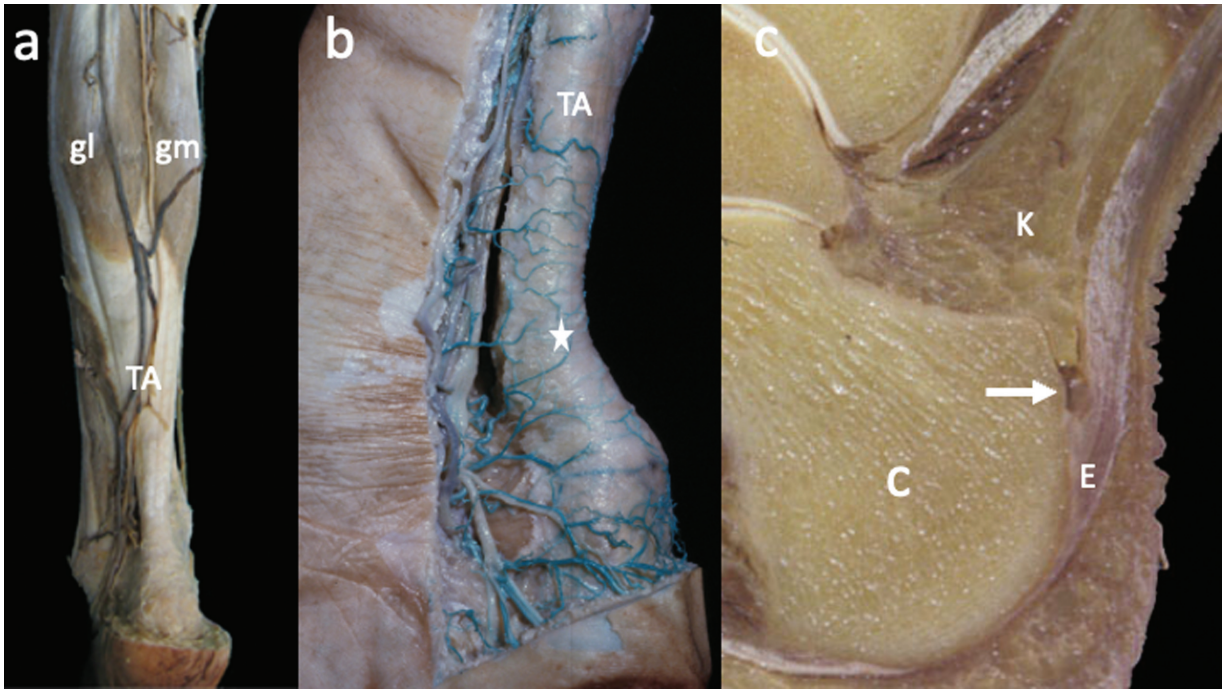

Fig. 1 Gross anatomy of the Achilles tendon (AT). (**a**) Posterior view. The AT is the distal insertion of the triceps surae that consists of the two heads of the gastrocnemius muscle and soleus muscle. Note the relationship of the AT with the lateral gastrocnemius (gl) and medial gastrocnemius (gm) muscles. (**b**) Sagittal view shows the highly vascularity of the paratenon. Note the relatively poorly vascularity of the midregion (star). (**c**) Sagittal section through the calcaneus (C) and distal AT shows the AT enthesis (E) and the prominent Achilles fat pad (K). Note the retrocalcaneal bursa (arrow) between the AT and the superior tuberosity of the calcaneus (figures courtesy of Dr. Alfonso Rodriguez, University of the Balearic Islands).

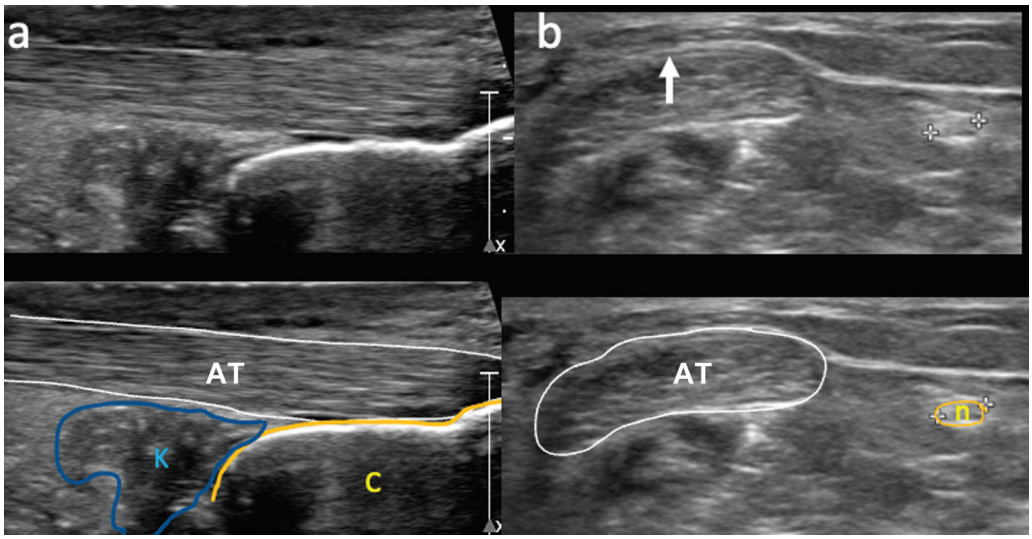

Fig. 2 High-resolution ultrasonography (US) demonstrates the dimensions and morphology of the tendon. (**a**) Longitudinal and (**b**) transverse US view of the normal Achilles tendon (AT). The tightly packed thin echogenic lines on the longitudinal view and echogenic punctate foci in the axial plane indicate the fibrillary anatomical architecture. The thin paratenon is usually seen surrounding the tendon as a slightly more echogenic border (b, arrow). Note the distention of the retrocalcaneal bursa (K) in (a). Note the sural nerve (n) in the axial plane in (b). C, calcaneus.

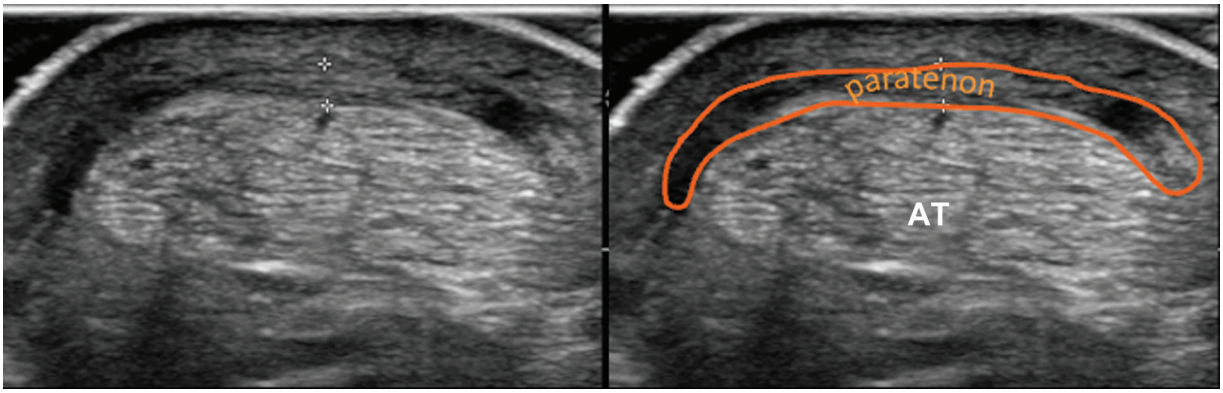

Fig. 3 High-resolution ultrasonography (US) of the Achilles tendon (AT) in the axial plane showing paratenonitis. A thickened hypoechoic paratenon is best appreciated posteriorly. On US, these changes appear as a thickened hypoechoic paratenon.

Achilles Tendon Injuries

Achilles tendon injuries can be separated into insertional tendinopathy (20–25% of the injuries), midportion tendinopathy (55–65%), and proximal musculotendinous junction (9–25%) injuries, according to the location of the pain (►**Fig. 4a**).

In *insertional tendinopathy,* symptoms localize within the first 2 cm of the attachment of the Achilles tendon to the calcaneus (►**Fig. 4b**). There may be a tendinopathy of the Achilles tendon insertion and associated prominence of the calcaneus (Haglund's morphology) and/or an associated retrocalcaneal bursitis. *Noninsertional tendinopathy* is the tendinopathy located at the midportion of the tendon, localized > 2 cm above the distal attachment (►**Fig. 4c**). The distinction between these two subclassifications is also justified because there seems to be a difference in prognosis during nonsurgical treatment.

On US, imaging findings of AT depend on the stage of tendon degeneration. The pathologic process usually starts as myxoid or hypoxic intrasubstance degeneration (or a combination), with an accumulation of mucoid material throughout the affected portion of the tendon. With tendinosis progression, there is a coalescence of microscopic foci of mucoid material, collagen fiber separation, and disruption. The degeneration can progress through the stages of microtears, intrasubstance tears, partial tear, and complete tear.[32] Mucoid accumulation results in tendon thickening that may be diffuse, fusiform, or, less commonly, nodular.[32,33]

On US evaluation, symptomatic tendinopathy usually shows tendon neovascularization. Dystrophic calcification can occur in degenerative tendon, often progressing to ossification, with distinct cortex and trabecula.

Ultrasonography Treatment Procedure

Patient Positioning

The patient is placed in a prone position, with the leg fully extended and the foot hanging off the end of the bed table (►**Fig. 5**). This exposes the tendon at the posterior aspect of the ankle.

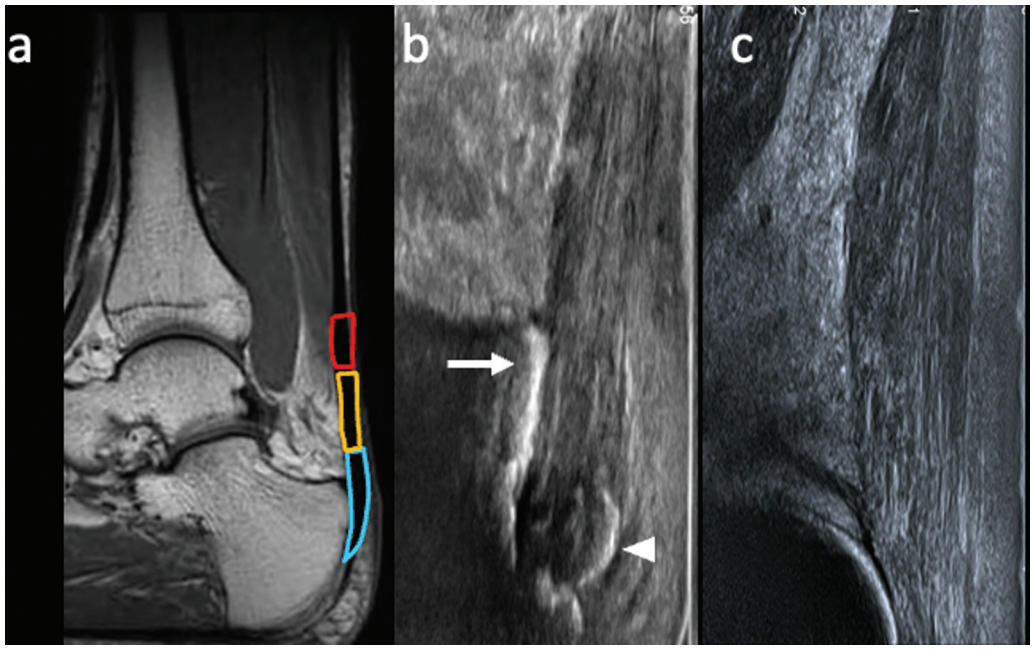

Fig. 4 (**a**) Sagittal DP magnetic resonance image of a normal Achilles tendon (AT). AT injuries can be separated into insertional tendinopathy (blue), midportion tendinopathy (yellow), and proximal musculotendinous junction (red) injuries. (**b**) Insertional tendinopathy: The injury is localized within the first 2 cm of the attachment of the AT to the calcaneus. High-resolution ultrasonography (US) in the longitudinal plane shows the AT enlarged and hypoechoic with heterogeneous echotexture. Note the associated prominence of the calcaneus (Haglund's morphology) (white arrow) and the enthesopathic calcification at the tendon insertion (arrowhead). (**c**) Noninsertional tendinopathy is the tendinopathy located at the midportion of the tendon, localized > 2 cm above the distal attachment. High-resolution US in the longitudinal plane shows the tendon diffusely enlarged.

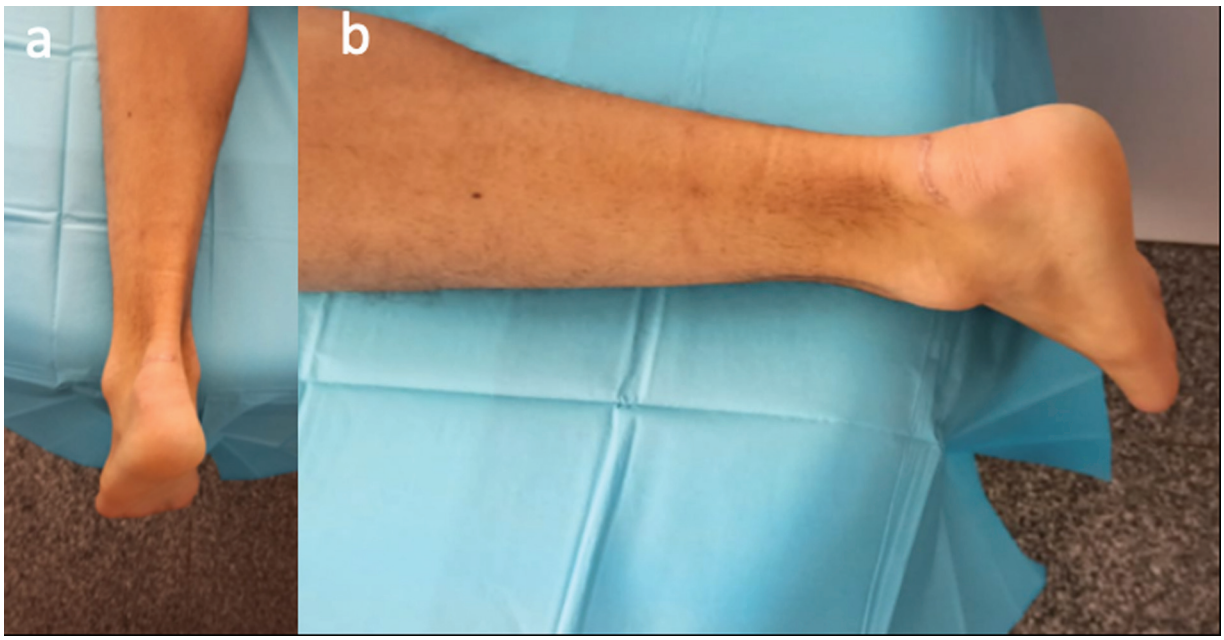

Fig. 5 In the prone position, the knee is fully extended and the foot hangs off the end of the bed. The injection can be administered with the transducer in a sagittal or axial orientation over the Achilles tendon, and the needle is introduced using an in-plane technique. For high-volume image-guided injection, the needle is placed between the tendon and Kager's fat pad; for platelet-rich plasma injections, it is taken into the tendon itself.

Fig. 6 Recommended transducers. (**a**) Linear, 6–15 MHz. (**b**) Hockey stick, 8–18 MHz.

Skin Antisepsis and US Probe Disinfection

Ordinary antisepsis is generally sufficient to guarantee a safe procedure for both the patient and operator. Preliminary disinfection of instruments is mandatory before starting the procedure.[34] For skin cleaning, we use uncolored disinfectant (►**Fig. 6**).

We use a sterile probe cover to protect the probe. The use of a sterile lubricating gel is advisable to optimize the contact between probe and skin.

High-Volume Injection

Equipment Needed

A. Transducers: Linear, 6–15 MHz; hockey stick, 8–18 MHz (►**Fig. 6**)

B. Nonsterile materials (►**Fig. 7**)
- Nonsterile gloves
- Bandages
- Anesthetic spray
- Lubricating gel
- Uncolored disinfectant.

C. Expendable materials (►**Fig. 8a**)
- One 21G needle
- One 18G needle
- Four- to five Cono Luer Lock (10 mL) syringes
- Sterile gel
- Tubing
- Sterile gloves
- Gauzes
- Sterile probe cover
- Sterile dressing pack

D. Medications (►**Fig. 8b**)
- Sterile saline solution (40 mL)
- Local anesthesia: Lidocaine 1% (10 mL)
- Steroid

US-guided HVI Procedure

Our US technique is identical to that suggested by other authors.[9–12,35,36] In general, a medial-to-lateral approach is preferred to avoid the superficial located sural nerve. The choice of lateral-to-medial or medial-to-lateral direction depends on the location of the sural nerve. Under US control we identify the point where we want to perform the injection and mark the skin with a needle cap and marker.

Using an aseptic technique and helped by an assistant, using a transverse needle approach, a 21G needle is inserted between the anterior aspect of the Achilles tendon and Kager's fat pad. The needle is attached to a connecting tube and inserted under real-time US guidance. A mixture of 8 mL 1% or 2% lidocaine and 2 mL of 12 mg betamethasone is injected (total: 10 mL),

Fig. 7 Nonsterile material. Nonsterile gloves. Bandages. Anesthetic spray. Lubricating gel. Uncolored disinfectant.

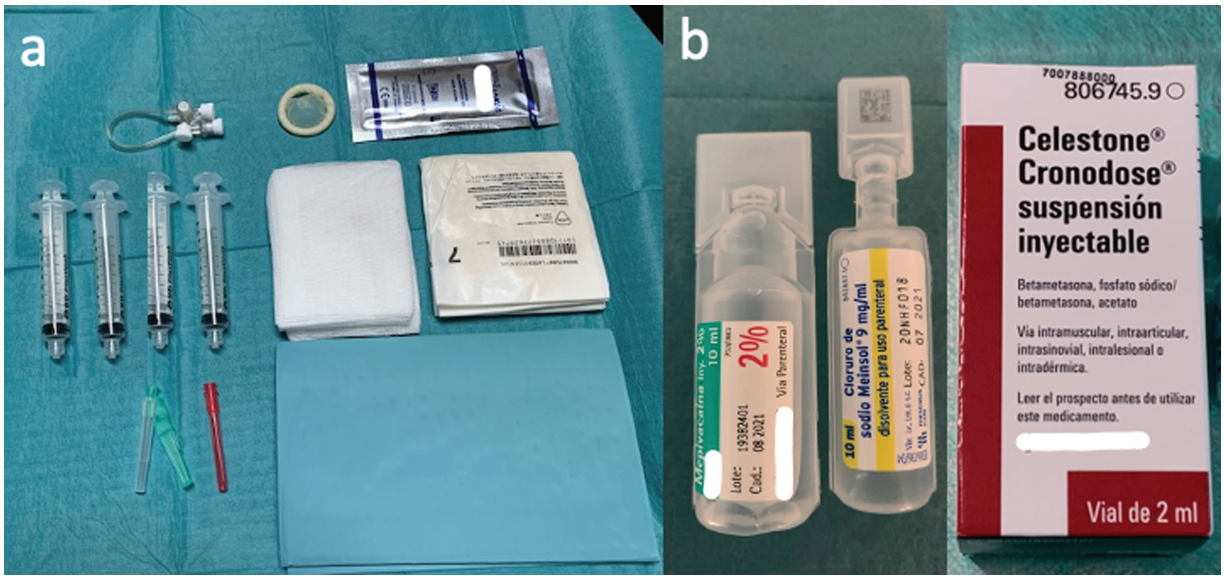

Fig. 8 Equipment preparation for high-volume image-guided injection tendon stripping. (**a**) Expendable material: One 21G needle. One 18G needle. Four to five Cono Luer Lock (10 mL) syringes, sterile gel, tubing, sterile gloves, gauzes, sterile probe cover, sterile dressing pack. (**b**) Medication: sterile saline solution (40 mL), local anesthesia, steroid.

immediately followed by 10 mL injectable normal saline three to four times depending on the characteristics of each patient (total volume: 40–50 mL). The position of the needle is monitored continuously by US during this phase, and the needle is moved gently across the anterior aspect of the tendon to ensure uniform effect over the symptomatic area. The saline is injected in bursts to maximize potential mechanical effect (►**Fig. 9**). Note: Some authors have described using more or less volume and without adding CS.[37]

Platelet-Rich Plasma Injection

Equipment Needed

A. Transducers: Linear, 6–15 MHz; hockey stick, 8–18 MHz (►**Fig. 6**)

B. Nonsterile materials (►**Fig. 7**)
- Nonsterile gloves
- Bandages
- Anesthetic spray
- Lubricating gel
- Uncolored disinfectant

C. Medications (►**Fig. 10**)
- PRP (1–3 mL)
- Lidocaine 1% (10 mL)

D. Expendable materials (►**Fig. 10**)
- One 21G needle
- One 18G needle
- Two Cono Luer Lock (10 mL) syringes
- Sterile gel

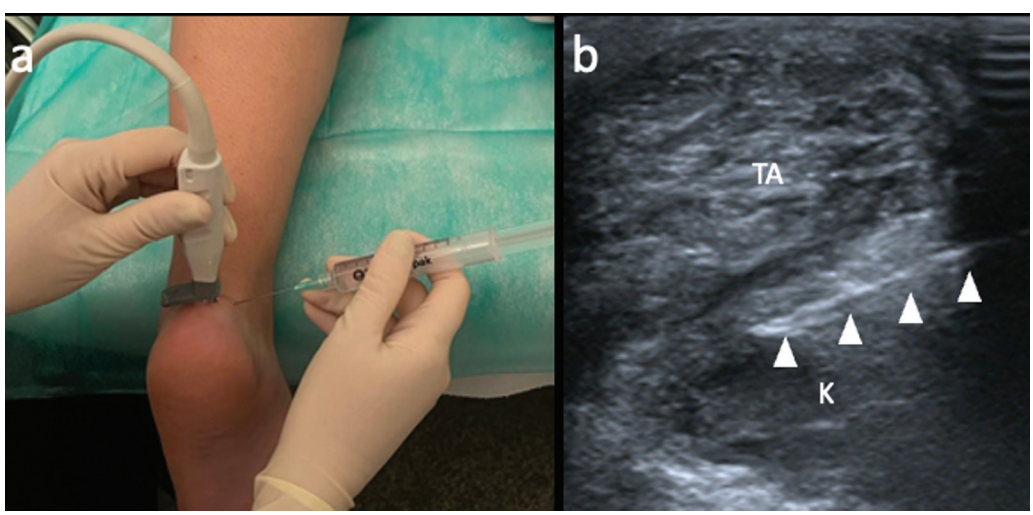

Fig. 9 Ultrasonography (US)-guided high-volume image-guided injection. (**a**) In the axial orientation, the needle is introduced at 15 to 20 degrees using an in-plane technique from the lateral aspect of the ankle. (**b**) Needle is inserted in the interface between the anterior aspect of the Achilles tendon (AT) and Kager's fat pad (K; arrowheads); the needle is attached to a connecting tube and inserted under real-time US guidance. A total volume of 40 to 50 mL is injected.

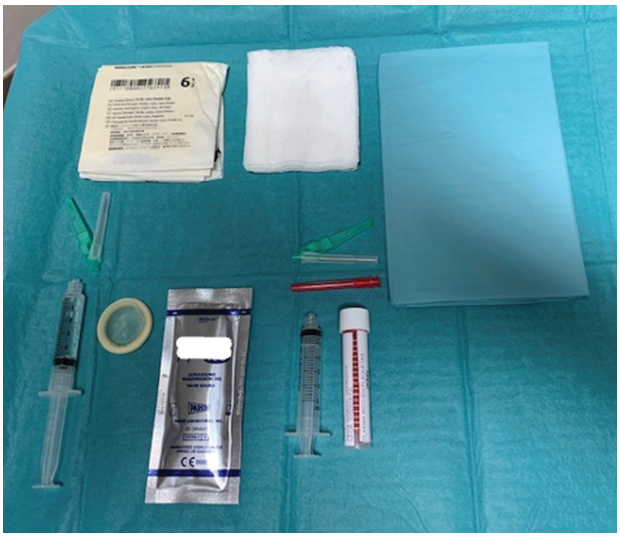

Fig. 10 Equipment preparation for platelet-rich plasma (PRP). Expendable material and medication: One 21G needle, one 18G needle, two Cono Luer Lock (10 mL) syringes, sterile gel, sterile gloves, gauzes, sterile probe cover, sterile dressing pack. Medication: PRP (1–3 mL), lidocaine 1% (10 mL).

- Sterile gloves
- Gauzes
- Sterile probe cover
- Sterile dressing pack

US-guided PRP Procedure

PRP can be obtained by manual procedures (open technique) or disposable kits (closed technique). According to the platelet concentrations, PRP can be classified as low, intermediate, or high concentrations. The correct platelet concentration must be ~ 1 million.

Depending on the presence of white blood cells, PRP can be classified into "pure PRP" without leukocytes and "L-PRP" with leukocytes.

PRP can be liquid without activation but can also be used after activation, resulting in a gelatinous substance. Platelet-rich fibrin is a solid material and not an injectable platelet suspension.

The PRP we use is prepared manually by the Hematology Department. The PRP obtained is nonactivated L-PRP.

Under US control, we identify the point where we want to perform the injection and mark the skin with a needle cap and marker.

We can optionally use anesthetic spray. In our experience, the procedure is painful, so we inject anesthesia into the peritendinous level but not into the tendon.

For PRP injections, the Achilles tendon can be viewed in the sagittal plane and the needle can be introduced intratendinous from the dorsal aspect at 15 to 20 degrees directly into the tendon (►**Fig. 11a**). Alternatively, an axial approach can also be used with the needle again guided into the tendon itself. We prefer to use the sagittal approach. In both positions, the PRP can be delivered using a fenestration technique.

Using an aseptic technique and under real-time US guidance, < 5 mL 1% lidocaine is injected into the peritendinous level (►**Fig. 11b**). Then we locate the lesion with US and infiltrate it with the PRP (►**Fig. 11c**).

The optimal dose, number, and interval of injections is controversial. We administer 1 to 3 mL PRP depending on the size of the lesion and repeat the procedure 12 weeks later if necessary. Most recent studies suggest that a single infiltration with 1 mL PRP is enough.[16,22,25]

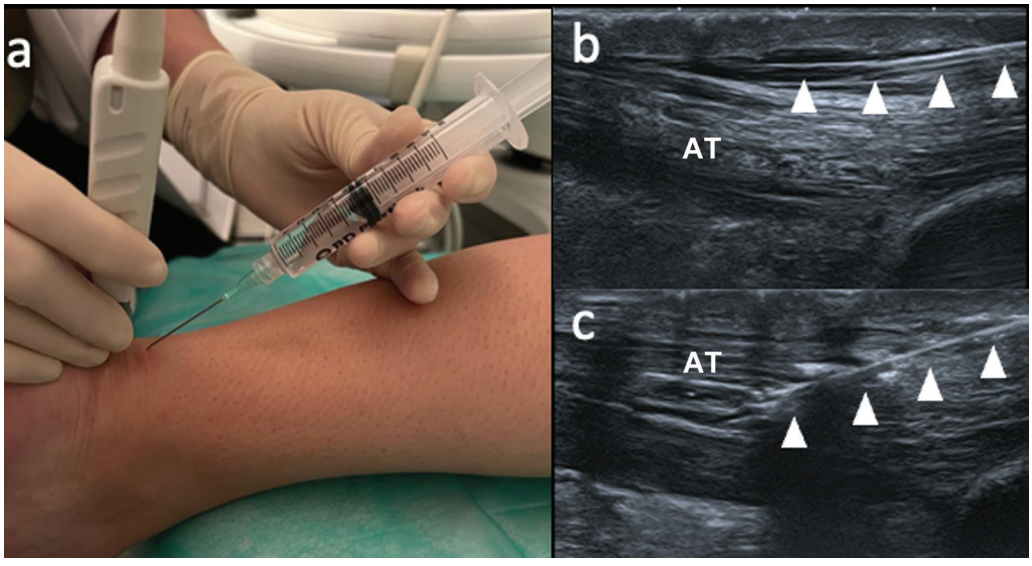

Fig. 11 Ultrasonography (US)-guided platelet-rich plasma (PRP). (**a**) In the sagittal orientation, the needle is introduced from the dorsal aspect at 15 to 20 degrees using an in-plane technique. (**b**) High-resolution US axial view. Under real-time US guidance, < 5 mL 1% or 2% lidocaine is injected into the peritendinous level (arrowheads). (**c**) Then 4 to 5 minutes later, with the needle under US guidance, PRP is injected into the tendon itself. The PRP can be delivered using a fenestration technique (arrowheads). AT, Achilles tendon.

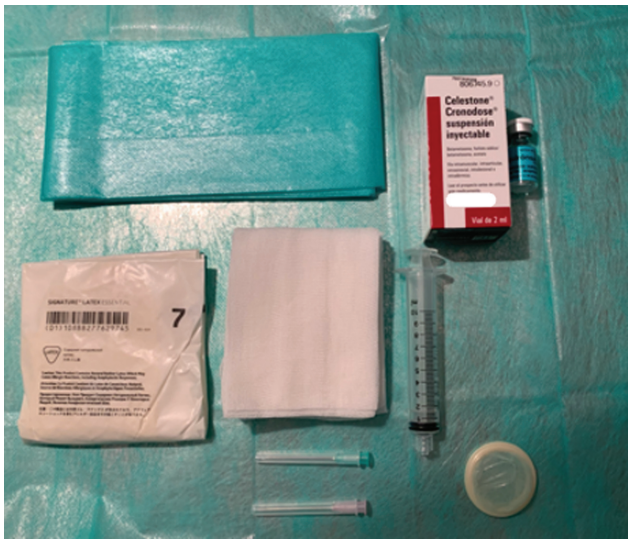

Fig. 12 Equipment preparation for corticosteroid injection. Expendable material: one 21G needle, one 18G needle, one syringe Cono Luer Lock (5 mL), sterile gel, sterile gloves, gauzes, sterile probe cover, sterile dressing pack. Medication: local anesthesia and steroid.

Corticosteroid Injection

Equipment Needed

A. Transducers: Linear, 6–15 MHz; hockey stick, 8–18 MHz

B. Nonsterile materials (►**Fig. 7**)
- Nonsterile gloves
- Bandages
- Anesthetic spray
- Lubricating gel
- Uncolored disinfectant

C. Medications (►**Fig. 12**)
- Betamethasone 12 mg/2 mL or triamcinolone acetonide 40 mg/1mL.
- Lidocaine 1% or 2% (10 mL)

D. Expendable materials (►**Fig. 12**)
- One 21G needle
- One 18G needle
- One Cono Luer Lock (5 mL) syringe
- Sterile gel
- Sterile gloves
- Gauzes
- Sterile probe cover
- Sterile dressing pack

US-guided Retrocalcaneal Bursa Corticosteroid Procedure

In general, a medial-to-lateral approach is preferred to avoid the superficially located sural nerve. Under US control, we identify the point where we want to perform the injection and mark the skin with a needle cap and marker.

Using an aseptic technique and with a transverse needle approach, a 21G needle is inserted in the retrocalcaneal bursa (►**Fig. 13**). A mixture of 2 cc betamethasone and 2 cc 1% or 2% lidocaine or 1 cc triamcinolone acetonide and 2 cc 1% or 2% lidocaine is introduced into the retrocalcaneal bursa.

Postprocedural Treatment

How patients are managed after treatment varies widely.

- High-volume injection

Patients are allowed to walk on the injected leg immediately but are advised strictly to refrain from high-impact activity, such as running or jumping for 72 hours. After 72 hours, patients are sent to their referring clinicians for advice about physiotherapy and returning to sport.

- PRP injection

After treatment we suggest that the patient rest for 72 hours. It is recommended not to locally apply ice. The patient is advised not to take nonsteroidal anti-inflammatory

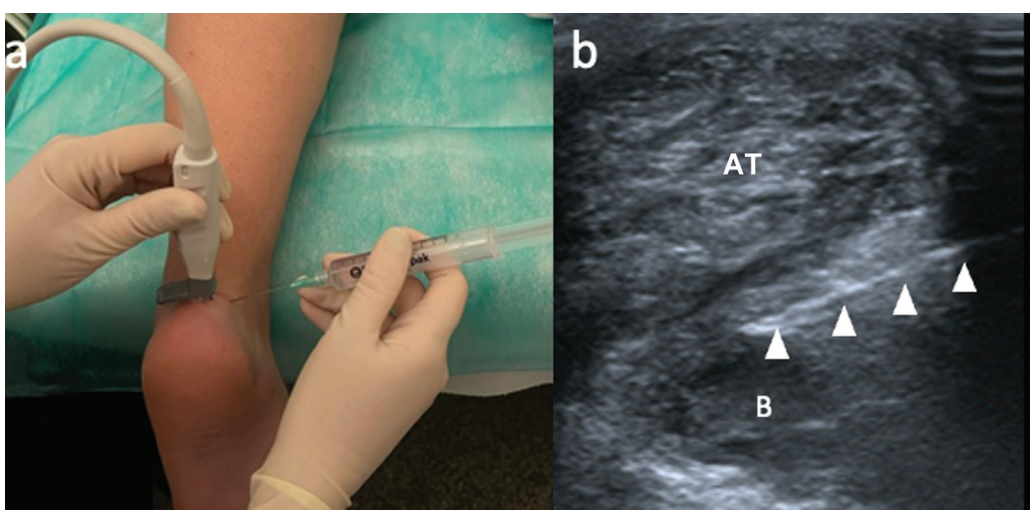

Fig. 13 Ultrasonography (US)-guided corticosteroid injection. (**a**) In the axial orientation, the needle is introduced at 15 to 20 degrees using an in-plane technique from the lateral aspect of the ankle. (**b**) The needle is inserted in the retrocalcaneal bursa (arrowheads). A mixture of 2 cc betamethasone and 2 cc 1% or 2% lidocaine or 1 cc triamcinolone acetonide and 2 cc 1% or 2% lidocaine is introduced into the retrocalcaneal bursa.

drugs (NSAIDs) for 1 week before and 2 weeks after the procedure. Finally, a written recommendation of non-NSAID oral painkillers is given to patients in case of pain.

- Corticosteroid injection

Patients are asked to avoid weight-bearing for the following 48 hours and to continue their baseline medications including NSAIDs.

Clinical Effectiveness

Scales used to evaluate the effectiveness treatment of Achilles tendon injuries vary widely among different authors. The visual analog scale (VAS) is a simple technique to measure the subjective experience of pain. A score of 0 means "no pain"; a score of 10 means the "worst pain imaginable."[38]

The Victorian Institute of Sport Assessment–Achilles questionnaire (VISA-A) is designed to evaluate pain and symptom severity with activity in patients with AT. The VISA-A contains eight questions, and scores range from 0 to 100, with 100 indicating no symptoms and full participation in physical activity.[39]

In cases after HVIGI and CS injection, imaging is not routinely used during follow-up. After treatment, patients are sent to their referring physician; a short treatment of physiotherapy is recommended. Patients are instructed to call our department if fever or pain persist 2 months post-treatment. We then perform US to detect the presence of complications, and in some cases a second US-guided injection is performed.

In the PRP procedure, we perform a clinical control (VAS and VISA-A questionnaire) and US 12 weeks after treatment. A second intratendinous infiltration of PRP is sometimes indicated.

Plantar Fascia

Background

Plantar fasciitis (PF) is the most common cause of plantar pain in the heel of the foot in adults, $\sim$ 7% of all foot pain in adults $>$ 65 years of age, and 24% in athletic individuals.[40] The main stress to the fascia occurs during walking, especially at the heel elevation phase. It is more common in athletes because of overuse and repetitive impact to the heel, but in patients $>$ 40 years of age, it is more related to other factors, such as being overweight; long-standing periods, especially wearing work boots; and systemic disease such as diabetes and/or rheumatoid and other inflammatory types of arthritis. It is also related to AT and biomechanical problems.

The plantar fascia has three components: the medial, central, and lateral band. The most affected part in PF is the central band, a strong and thick aponeurosis attached to the calcaneus tuberosity and the main support of the plantar arch. The central band is divided at the level of the Lisfranc joint into five fascicles that cover the flexor digitorum brevis and more distally help stabilize the flexor tendons, ending at the base of the proximal phalanges.

The most frequent symptom in PF is pain at the inferomedial part of the heel that is at its peak in the morning and improves during the day. It worsens with load, sport activities, and long walks. At the physical examination, patients have selective pain at the calcaneus origin of the fascia that improves with dorsal flexion and increases with extension of the foot.

Overuse and biomechanical problems may increase excessive tensile strain within the plantar fascia, resulting in microscopic tears and chronic inflammation. Overuse rather than anatomy is thought to be the most common cause in athletes. Although PF presents with the typical clinical signs of inflammation, such as pain and swelling in acute cases, the histologic findings do not show inflammation but instead are characterized by tissue destruction and tissue repair, neovascularization, and fibrosis with infiltration of macrophages, lymphocytes, and plasma cells. The histologic findings in chronic PF therefore support a degenerative rather than an inflammatory process. Thus the term *fasciopathy* would be more accurate; fasciitis should be used when the condition becomes clinically significant.[29,41]

The diagnosis of PF is usually made with clinical findings. US findings are hypoechoic thickening ($>$ 4–5 mm) of the insertional or proximal part of the plantar fascia.

The first treatments are rest, avoiding flat shoes, physiotherapy with stretching of the fascia and the triceps surae muscle, and solving biomechanical problems if they are present with arch supports and orthotics. PF is normally regarded as a self-limiting condition and usually resolves within 6 to18 months.[41] But symptoms improve slowly, and there are many intractable cases that need another treatment, such as US-guided injections or extracorporeal shockwave therapy. When conservative management fails, US-guided CS injections are often the first option.[42]

Two types of imaging-guided interventional procedures are described in PF. In the first, a substance like CS is injected around the fascia (either superficial or deep layers or both). In the second, procedures are performed directly on the tendon, such as injection with PRP.

Regarding CS injection, no studies have compared the efficacy of the different approaches around the fascia (superficial or deep of both layers). Therefore the decision on technique remains a personalized one based on patient factors, imaging findings, and the radiologist's experience.

A Delphi-based consensus of experts from the European Society of Musculoskeletal Radiology (ESSR) recently reviewed the literature and concluded that US guidance is strongly recommended to improve the efficacy of interventional procedures for PF, particularly using PRP.[8]

There is evidence that US-guided CS injections are more effective than palpation-guided injections to treat PF, providing significant short-term pain relief, particularly when combined with strength training and stretching. Both can be recommended as the first-line treatment in patients with PF.[8,43,44] Often combined with local anesthetic, these injections are often diagnostic and therapeutic.

Adverse effects of steroid injection at the site are local infection, tendon rupture, skin depigmentation, and fat pad

atrophy.[26] Tendon rupture, the most serious complication, is relatively rare with a reported incidence of 2.5 to 6.7% in case of injection for PF and associated more with recurrent and blind palpation-guided injections. US guidance significantly decreases complication rates.[27]

For chronic PF, PRP injections provide significant pain relief with better outcome at mid- and long-term follow-up if compared with CS injections.[45,46] Although both PRP and CS can decrease inflammation, PRP may be advantageous over CS because it may modulate the plantar fascia degeneration due to its biological regenerative properties. PRP contains an abundance of growth factors and bioactive cytokines that are believed to influence healing by augmenting cellular migration, improving cellular proliferation, promoting angiogenesis, and increasing matrix deposition. This results in increasing fiber organization and tensile strength in soft tissue. PRP also releases vascular endothelial growth factor that promotes angiogenesis and may facilitate healing of a degenerative condition by improving neovascularization and repair. By contrast, CS has no such regenerative capacity, and consequently its effect is solely to reduce inflammation.[45]

The latest meta-analysis showed significantly lower VAS scores in the PRP group in the intermediate term (6 months) and long term (12 months) compared with CS injection. However, in the short term (1 and 3 months), no differences between the two groups were observed.[47]

In conclusion, PRP injection for PF is an effective and safe treatment option. In comparison with CS, PRP showed significantly better pain control at intermediate- and long-term follow-up, so for chronic PF cases, the current clinical evidence suggests it may lead to a greater improvement in pain and functional outcome. Therefore PRP could be recommended as a first-line treatment for those patients.[8,47]

According to the recent ESSR consensus, the effectiveness of US-guided injections with ozone, hyaluronic acid, or botulinum toxin type A has not yet been sufficiently proven to be recommended for PF because no comparative studies are available.[8]

Recommended Clinical Indications

When conservative management is not effective, we recommend CS injections combined with strength training and stretching and PRP injections for chronic PF.

Pretreatment US Tendon Evaluation

The Normal Plantar Fascia

Plantar fascia is a thick and strong aponeurosis that supports the plantar arch (►**Fig. 14**). It is a dense collection of collagen fibers on the sole of the foot. These fibers are mostly longitudinal but also transverse, and the fascia is firmly attached to the calcaneus tuberosity.

Plantar fat pad is a group of specialized fat pads separated by collagen septa and elastic fibers that extend from the skin to the calcaneus periosteum. It absorbs heel impact forces when walking or running.

US demonstrates the normal dimensions and morphology of the fascia and can scan the insertional and proximal zones but also the thin heads behind the metatarsals. The fibrillary anatomical architecture can be appreciated as tightly packed thin echogenic lines on longitudinal scanning, with no vascularity on color Doppler examination (►**Fig. 15**).

Plantar Fascia Injuries

PF injuries can be separated into insertional fasciitis and nonproximal/noninsertional fasciitis (►**Fig. 16**). On US, PF is seen as focal hypoechoic thickening and loss of normal echogenicity, often with fluid around the fascia and calcaneus cortical irregularity or bone spur (►**Fig. 16**). It can demonstrate hyperemia on Doppler examination that should be evaluated in plantar flexion (relaxation of the fascia).

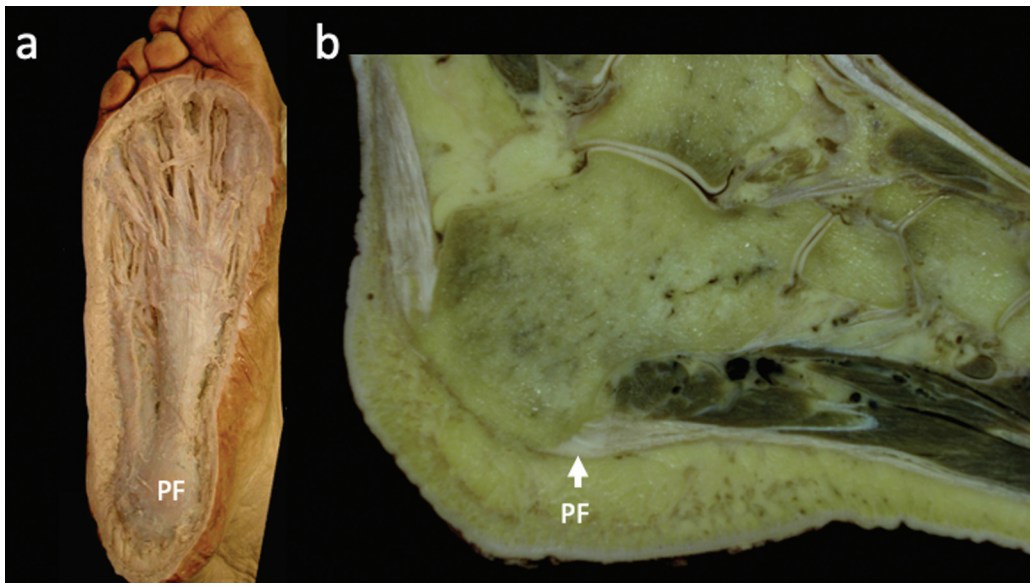

Fig. 14 Gross anatomy of the plantar fascia (PF). (**a**) Plantar view of the PF. (**b**) Sagittal view of the PF shows the normal insertion of the fascia and the fat pad (figures courtesy of Dr. Alfonso Rodriguez, University of the Balearic Islands).

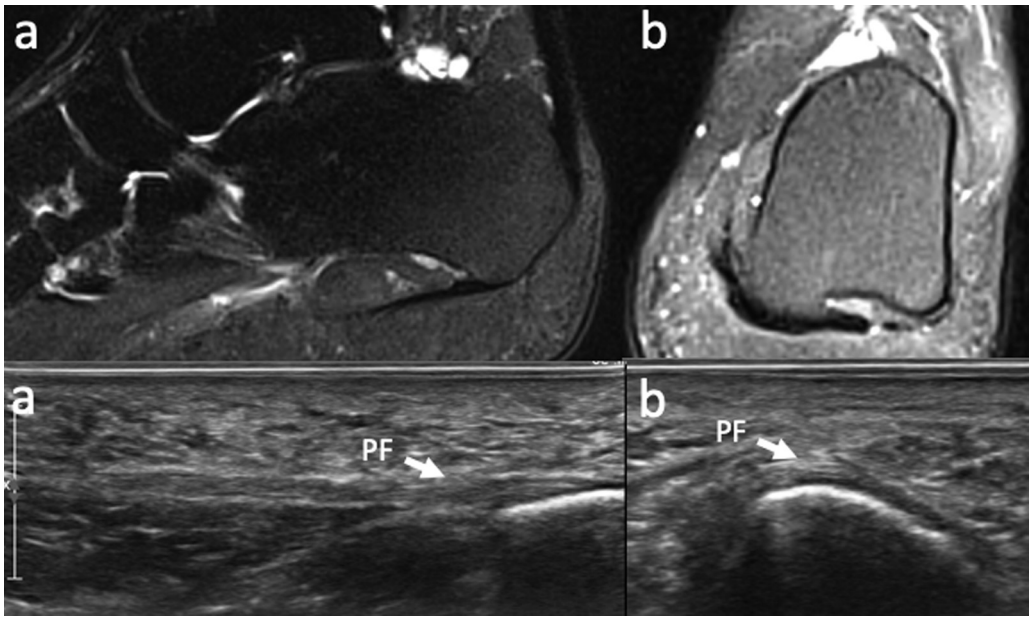

Fig. 15 Magnetic resonance imaging and high-resolution ultrasonography (US) demonstrate the dimensions and morphology of the fascia. (**a**) Longitudinal and (**b**) transversal US view of the normal plantar fascia (PF).

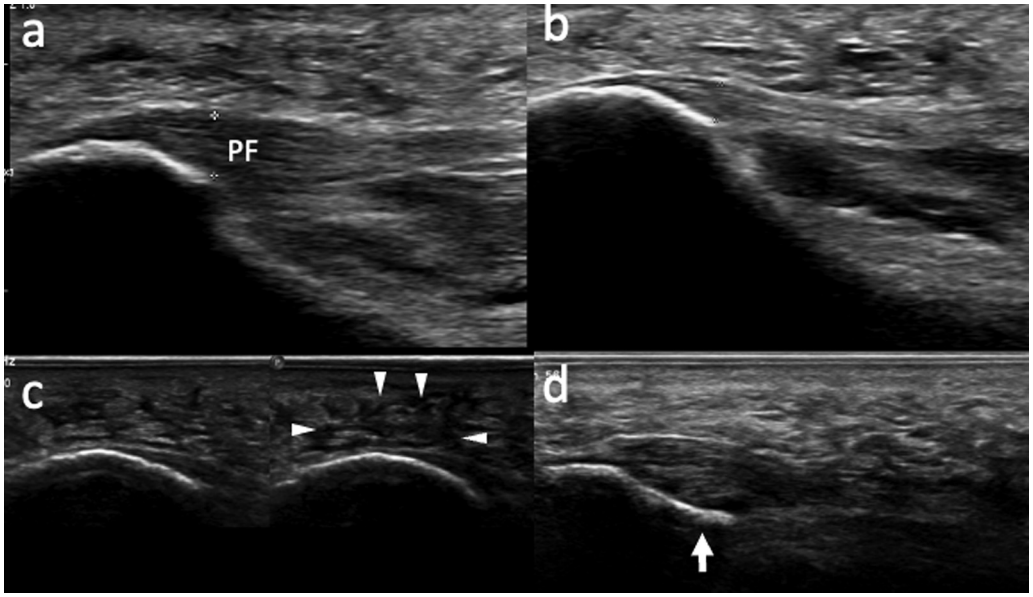

Fig. 16 High-resolution ultrasonography of the plantar fascia (PF) in the sagittal and axial planes showing fasciitis. A thickened hypoechoic fascia is better appreciated in the sagittal/longitudinal view and is useful to compare with the contralateral fascia. (**a**) Plantar fasciitis. (**b**) Contralateral normal fascia. (**c**) Note edema and fluid in the fat pad (arrowhead) and (**d**) bone spur (white arrow), findings often associated with fasciitis.

US Treatment Procedure

Patient Positioning

The patient is placed in a supine position, with the leg fully extended and external rotation of the foot (►**Fig. 5**).

Skin Antisepsis and US Probe Disinfection

Ordinary antisepsis is generally sufficient to guarantee a safe procedure for both the patient and operator. Preliminary disinfection of instruments is mandatory before starting the procedure.[34] For skin cleaning, we use uncolored disinfectant (►**Fig. 7**).

We use a sterile probe cover. The use of a sterile lubricating gel is advisable to optimize the contact between probe and skin.

Corticosteroid Injection

Equipment Needed

A. Transducers: Linear, 6–15 MHz; hockey stick, 8–18 MHz
B. Nonsterile materials (►**Fig. 7**)
- Nonsterile gloves
- Bandages

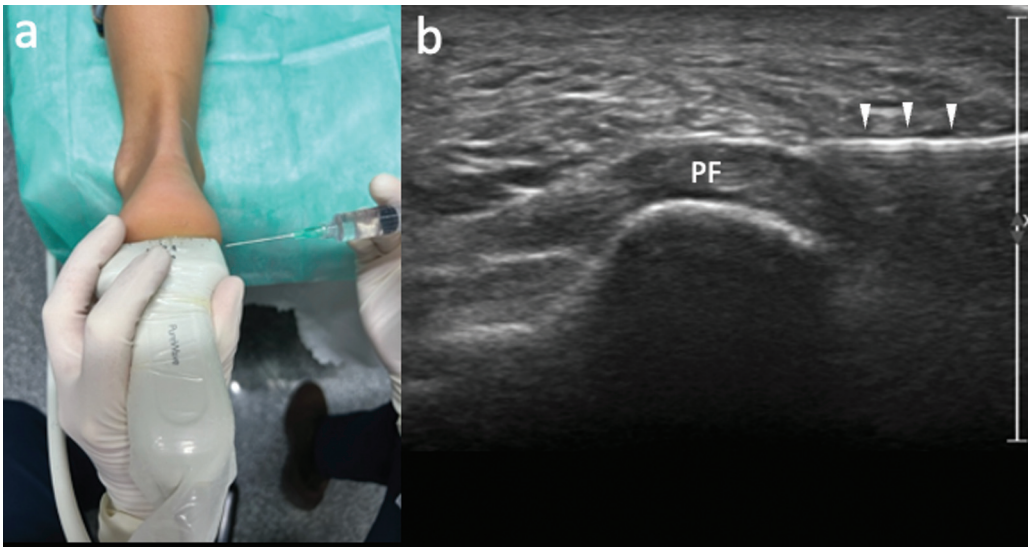

Fig. 17 Ultrasonography-guided corticosteroid injection. (**a**) In the axial orientation, the needle is introduced at 15 to 20 degrees, using an in-plane technique from the medial aspect of the heel. (**b**) The needle is inserted close to the superficial layer of the plantar fascia (PF; arrowheads) (arrowheads). A mixture of 2 cc betamethasone and 2 cc 1% or 2% lidocaine or 1 cc triamcinolone acetonide and 2 cc 1% or 2% lidocaine is introduced around the fascia.

- Anesthetic spray
- Lubricating gel
- Uncolored disinfectant

C. Medications (►**Fig. 12**)
- Betamethasone 12 mg/ 2 mL or triamcinolone acetonide 40 mg/1mL
- Lidocaine 1% or 2% (10 mL)

D. Expendable materials (►**Fig. 12**)
- One 21G needle
- One 18G needle
- One Cono Luer Lock (5 mL) syringe
- Sterile gel
- Sterile gloves
- Gauzes
- Sterile probe cover
- Sterile dressing pack

US-guided Corticosteroid Procedure

In general, an anterior and medial oblique to lateral approach and perifascial injection is preferred, avoiding intrafascial or fat pad injection. Under US control, we identify the injection point.

Using an aseptic technique and with a transverse needle approach, a 21G needle is located close to the fascia (►**Fig. 17**). A mixture of 2 cc betamethasone and 2 cc 1% lidocaine or 1 cc triamcinolone acetonide and 2 cc 1% lidocaine is introduced into the perifascial tissue (superficial or deep injection or both).

Platelet-Rich Plasma Injection

Equipment Needed

A. Transducers: Linear, 6–15 MHz; hockey stick, 8–18 MHz (►**Fig. 6**)

B. Nonsterile materials (►**Fig. 7**)
- Nonsterile gloves
- Bandages
- Anesthetic spray
- Lubricating gel
- Uncolored disinfectant

C. Medications (►**Fig. 10**)
- PRP (1–3 mL)
- Lidocaine 1% (10 mL)

D. Expendable materials (►**Fig. 10**)
- One 21G needle
- One 18G needle
- Two Cono Luer Lock (10 mL) syringes
- Sterile gel
- Sterile gloves
- Gauzes
- Sterile probe cover
- Sterile dressing pack

US-guided PRP Procedure

We use nonactivated L-PRP prepared manually by the Hematology Department. Under US control we identify the point where we want to perform the injection and mark the skin with a needle cap and marker.

We can optionally use anesthetic spray. In our experience, the procedure is painful, so we inject anesthesia into the perifascial level but not into the fascia.

For PRP injections, the approach is the same as the CS injection, but the needle should be introduced directly into the fascia into the most hypoechoic/degenerative area and can be delivered using a fenestration technique (►**Fig. 18**).

Using an aseptic technique and under real-time US guidance, < 5 mL 1% lidocaine is injected into the perifascial level. Then with US we localize the lesion and infiltrate it with the 2

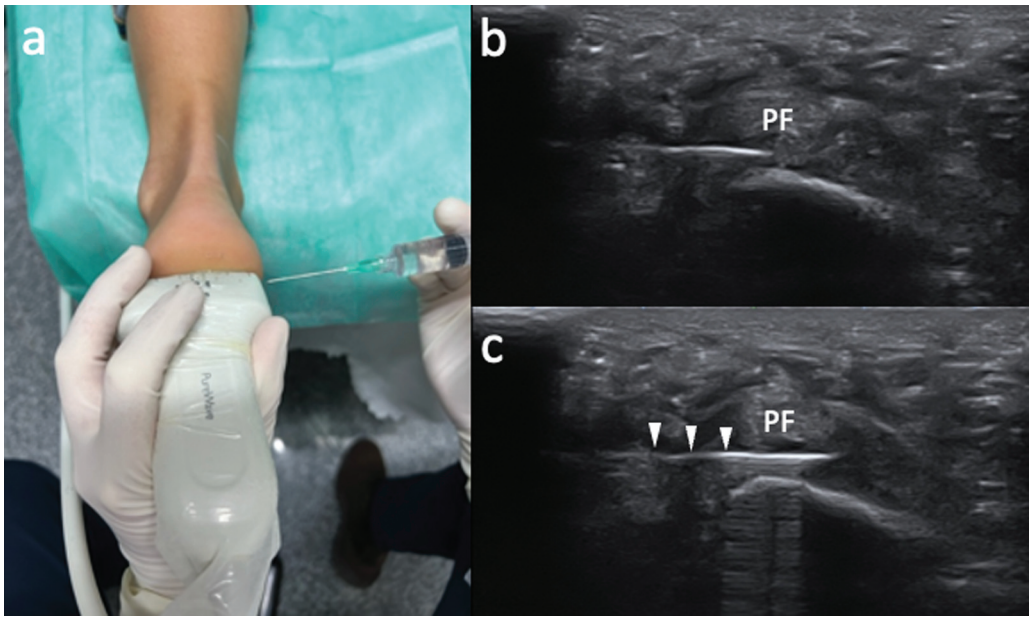

Fig. 18 Ultrasonography (US)-guided platelet-rich plasma (PRP) injection. (**a**) In the transversal orientation, the needle is introduced from the medial aspect, using an in-plane technique. (**b**) High-resolution US axial view. Under real-time US guidance, < 5 mL 1% or 2% lidocaine is injected into the soft tissues around the plantar fascia (PF) (arrowheads). (**c**) Then 4 to 5 minutes later, using the needle with US as a guide, PRP is injected into the fascia itself. The PRP can be delivered using a fenestration technique (arrowheads) (figures courtesy of Dr. Javier Fernández-Jara, Hospital Fundación Jiménez Díaz/Sanitas, La Zarzuela, Madrid, Spain).

to 3 mL PRP. We can repeat the procedure 12 weeks later, if necessary, but there is no consensus about the optimal timing and number of PRP injections.

Postprocedural Treatment

There is wide heterogeneity in how patients are managed after treatment. After a CS injection, patients are asked to avoid weight-bearing for 48 hours and to continue their baseline medications including NSAIDs.

After treatment with a PRP injection, we recommend the patient rest for 72 hours. It is recommended not to apply ice. The patient is advised not to take NSAIDs for 1 week before and 2 weeks after the procedure. Finally, a written recommendation for oral non-NSAID painkillers is given to the patients in case of pain.

Clinical Effectiveness

Two scales are often used to evaluate the effectiveness treatment of PF injuries in the literature. The VAS is a simple technique for measuring subjective experience of pain, as described earlier.

The American Orthopaedic Foot and Ankle Society (AOFAS) is a standardized evaluation of the clinical status of the ankle and hindfoot. It incorporates both subjective and objective information. Patients report their pain, and physicians assess alignment. The patient and physician work together to complete the functional portion. Scores range from 0 to 100, with healthy ankles receiving 100 points.[48]

With CS injection, imaging is not routinely used during follow-up. After treatment, patients are sent to their referring physician, and a short treatment of physiotherapy is recommended. Patients are instructed to call our department in case of fever or if pain persists at 2 months post-treatment. In this case, we perform US to detect the presence of complications, and in some patients, a second US-guided injection is performed.

After a PRP procedure, we perform a clinical control (VAS and AOFAS questionnaire) and US 12 weeks after treatment. In some cases, a second intrafascial infiltration of PRP is indicated.

Conclusion

US-guided injections in patients with Achilles tendon injuries has been shown to be a safe and feasible. These injections are quick, low cost, and minimally invasive. To date, no study has compared the efficacy of the different treatments, and therefore no definitive superior method has been established.

HVIGI seems to be effective to treat AT. The goal of PRP injected into areas of tendinopathy is to induce healing via cellular and humoral mediators. It seems to be effective for degenerative tendon disease with intrasubstance tearing. Local US-guided CS injection of the retrocalcaneal bursa improves bursitis symptoms.

US-guided injections in patients with PF injuries has been shown to be a safe and effective treatment. US-guided CS injections are quick, low cost, and minimally invasive. US-guided PRP injection showed significantly better pain control at intermediate- and long-term follow-up compared with CS. We can recommend PRP for chronic PF following the current clinical evidence because it has a regenerative capacity, facilitating healing of a degenerative condition by improving neovascularization and repair.

Conflict of Interest
None declared.

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