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Ultrasound evaluation of Achilles tendon among Diabetic Patients (type 2 Diabetes Milletus)

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Abstract:

The Achilles tendon (AT) is the strongest tendon in the human body, gaining its crucial importance with the upright gait of humans. The Achilles tendon (AT) is a well-studied structure that permits foot plantar flexion It connects the triceps surae muscle, consisting of the medial and lateral heads of the gastrocnemius muscle and the soleus muscle forming the dorsal shape of the calf with the calcaneus bone tuberosity. Tendinopathy is a chronic and often painful condition affecting both professional athletes and sedentary population. It is a multi-etiological disorder, the most evident causes of tendinopathy are related to tendon overuse and overload. Ultrasound (US) performed with high-resolution linear-array probes has become increasingly important in the assessment of ligaments and tendons around the ankle. US can provide a detailed description of normal anatomic structures, effective for evaluation of ligament integrity. In addition, it allows the performance of dynamic maneuvers, which may contribute to increase visibility of normal ligaments and improve detection of tears. It can facilitate accurate identification, localization and differentiation between synovial, tendinous and entheseal inflammation as well as joint, bursal and soft tissue fluid collection.

Keywords: *Radiological imaging, Achilles tendon, Diabetic Patients*

Introduction.

The Achilles tendon (AT) is the strongest tendon in the human body, gaining its crucial importance with the upright gait of humans. The Achilles tendon (AT) is a well-studied structure that permits foot plantar flexion It connects the triceps surae muscle, consisting of the medial and lateral heads of the gastrocnemius muscle and the soleus muscle forming the dorsal shape of the calf with the calcaneus bone tuberosity. In 65% of cases the tendon of the plantaris muscle associates laterally with the AT, the AT allows the transmission of forces generated by the triceps surae muscle, high enough to compensate a multitude of loading of the body weight as main actor at the ankle joint mediating plantar flexion [(1)].

The Achilles tendon (AT) is a common injury site with a wide range of overuse and acute presentations. These can include mid-portion tendinopathies, insertion tendinopathies, muscle injuries, and acute ruptures, there is Several factors contributes AT injury, including poor vascularization, especially in the midportion of the tendon, exposure to high loads during locomotion, and an altered tendon cell response [(2)].

Achilles tendon:

The average length of the AT is 15 cm and ranges from 11 to 26 cm. The mean width of the AT is 6.8 cm (4.5– 8.6 cm) at its origin, gradually becoming thinner at the midsection being 1.8 cm (1.2–2.6 cm). Then, it becomes more rounded until approximately 4 cm above the calcaneus before expanding. The mean width of the AT is 3.4 cm (2.0–4.8 cm) at its insertion to the midpoint of the posterior surface of the calcaneus [(3)].

The relative contribution of the soleus and gastrocnemius fibers to the AT is variable, and the exact

degree of contribution can be difficult to appreciate because of the changing orientation of the tendon fibers. From cadaver studies, in 52 % of the subjects 52 % of the Achilles tendon fibers come from the soleus and 48 % from the gastrocnemius, an equal contribution is provided in 35 %, and more than 60 % of contribution arises from the gastrocnemius in 13 % of cadavers [(3)].

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, The rotation is more evident in the terminal 5–6 cm ,it may cause constriction of the vascular networks, but also results in less fiber buckling when the tendon is lax and less deformation of individual strands when under tension. In this way, fiber distortion and inter fiber friction may decrease resulting in increase of tendon strength, This twisting occurs in a counterclockwise fashion for the right Achilles tendon and clockwise fashion for the left [(4)].

At its insertion site, the tendon flattens and broadens into a deltoid-type of attachment and develops an anterior concavity before inserting along the middle third of the posterior aspect of the calcaneus, The calcaneal insertion is a true enthesis with fibrocartilage intermeshing with the marrow of the calcaneus. This direct meshing of tendon fibrils into marrow provides significant strength at this site; thus, the site is a rare location of tendon failure [(5)].

Just anterior to the Achilles tendon, proximal to the point of insertion, lies the retrocalcaneal bursa (RCB) also known as the pre-Achilles bursa and retro-Achilles bursa (RAB)or superficial calcaneal bursa which is located between the skin and the Achilles tendon insertion, also lying Intra-articular and joint-associated fat pads including the Kager fat pad (KFP) ,The two bursae support AT and protect it from wear and tear and attached via the fibrocartilaginous enthesis at the calcaneal tuberosity, These structures are prone to overuse , the bursae and Kager fat pads might contribute to

inflammatory responses and healing [6].

Kager fat pad located anteroinferior to the distal soleus muscle, and posterior (superficial) to the distal flexor hallucis longus (FHL) muscle and tendon, the posterior ankle joint, talus and calcaneus, It is a triangular mass of adipose tissue lying anterior to the tendon, having multiple functions: they are paracrine-active, modulating inflammation, but also exert biomechanical functions by distribution of loading and protection of neighboring structures In addition, they contain sensory nerve endings (mediating proprioception and sensation of pain), and hence can protect tissues from overload during the healing process [7].

There is no true synovial sheath surrounding the Achilles tendon. Instead, a paratenon forms an elastic sleeve around the tendon to permit gliding. It is composed of sheets of dense connective tissue that separate the tendon from the deep fascia of the leg. The paratenon is not a true tendon sheath, it has no synovium. It is richly vascularized and it can become inflamed and enlarged, usually in runners [8]. The peritendinous structure and the abundance of mucopolysaccharides within the sheath permits sliding of the tendon along the adjacent tissues. Proximally, the paratenon is continuous with the muscle fascia and distally it blends with the periosteum of the calcaneus [9].

The nomenclature of tendon conditions is confusing. Tendonitis relates to acute conditions and involves inflammation of the tendon, from injury or overuse. Tendinopathy, however, is a degenerative process, with little or no inflammation and occurs over weeks or months [10].

Tendinopathy is a chronic and often painful condition affecting both professional athletes and sedentary population. It is a multi-etiological disorder, the most evident causes of tendinopathy are related to tendon overuse and overload, but may also be associated with certain sports (especially explosive power sports, e.g., jumping, sprinting, etc.), training errors). (Related to intensity,

frequency and technique), inadequate training area, adverse weather conditions and inappropriate footwear or equipment [11]. However, constitutional and systemic factors including sex, ageing, obesity, smoking, corticosteroid and statin use seem to play a role in the development of such disorder [12].

Chronic exposure to these factors may then result in an alteration of tendon structure and function, with a consequent increased risk of lesions, tears and rupture [11]. Among these, chronic hyperglycemia, as occurring in diabetes mellitus (DM), has shown to significantly increase the risk of tendinopathy [13].

Tendinopathy is the end result of a chronic imbalance between opposing processes of degeneration and repair in the tendon, usually from overuse injuries. In diabetic tendinopathy, however, the degeneration arises from advanced glycation end-products accumulating in load-bearing collagen. Vascular integrity and healing are often impaired, Collagen and matrix production abnormal. [14]. Obesity, hypertension, ageing, alcohol, and smoking often coexist, and are independent and exacerbating factors. The pathological processes accumulate over years preceding clinical symptoms [13].

DM is a metabolic disease affecting nearly 425 million individuals between 20 and 79 years of age worldwide in 2017, with an estimated global prevalence of 9% amongst the adult population. DM is characterized by persistent hyperglycemia as a consequence of a deficiency in insulin secretion and/or insulin resistance. DM can be distinguished in two major types: type 1 diabetes, an autoimmune disease characterized by the loss of ß-cells, hence resulting in a total insulin deficit; and type 2 diabetes, which is accompanied by insulin resistance and loss of insulin secretion at more advanced stages [12].

Type 2 diabetes mellitus accounts for around 90% of all cases of diabetes [15], The most important risk factor for this condition is represented by an increased body mass index (BMI), The classic phenotype of the patient with type 2 diabetes is accompanied by overweight or Obesity. These conditions cause the onset of insulin resistance. Other several factors, not less important, include age, race and lack of physical activity. [12]

Type 2 Diabetes has been associated with several musculoskeletal alterations, including fractures, spinal stenosis, ossification of the posterior longitudinal ligament, intervertebral disc degeneration, osteoarthritis, and a decrease of bone mineral density (BMD) [16].

Type 2 Diabetes is thought to be an important factor favoring the development of tendinopathy. Indeed, the risk of tendinopathy seems to increase in patients undergoing insulin treatment and with diabetes duration (mean 5.3 years) [13]. Ultrasound screening of older patients with Type 2 Diabetes but without neuropathy found that 36.8% showed signs of asymptomatic Achilles tendinopathy [17]. Other soft-tissue conditions such as bursitis, carpal tunnel syndrome, shoulder capsulitis and plantar fasciitis are not primarily disorders of tendons but share pathological similarities and are also more common in diabetes [12].

Epidemiology of Tendinopathy in Diabetic Patients

There is higher incidence of tendinopathy in patients with type 2 diabetes. The epidemiological relationship between tendinopathy and type 2 diabetes was confirmed by different meta-analysis showing the actual existence of a greater risk of developing tendinopathy in individuals with type 2 diabetes [14]. However, the strength of such an association may be even stronger considering age, ethnic group, duration of type 2 diabetes, glycated hemoglobin levels and body mass index (BMI) in individuals affected [18].

Overall, patients with type 2 diabetes tend to show more severe signs of tendinopathy with increased tendon pain [13]. Compared to subjects without type 2 diabetes and inherently present a greater risk of tendon rupture requiring hospitalization [19]. The most represented forms of tendinopathy in individuals with type 2 diabetes involve Achilles tendinopathy (AT), rotator cuff disease and hand finger flexor tendinopathy [12].

Clinical features:

The onset of tendinopathy is usually gradual with no history of injury, although it can be precipitated by a seemingly trivial activity. Thereafter, tendon movement causes disproportionate pain and stiffness that the patient assumes will self-resolve. Symptoms may fluctuate and patients often present weeks or months after failing to improve. Symptoms lasting longer than 2 months are suggestive of tendinopathy. On examination, there is tenderness on palpation of the tendon and pain on active or passive movements, but absence of erythema or warmth to touch. There is often an associated reduction in muscle strength [10].

Patients with DM with poor glycemic control often suffer from chronic pain, limited range of motion (ROM) of Ankle joint, and have a higher risk of tendon tears [20]. The prevalence of connective tissue diseases, such as Dupuytren's disease, trigger finger, carpal tunnel syndrome, rotator cuff tears and shoulder adhesive capsulitis (frozen shoulder), were increased in both patients with type 1 and type 2 diabetes [21].

Symptomatic rotator cuff tears and acute Achilles tendon ruptures are common in patients with DM [22]. After surgical repair, these patients showed a limited ROM and a higher incidence of re-tears, For the ruptured Achilles tendons, patients with DM have higher rate of postoperative infection and poorer tendon healing [22], Clinical research revealed that patients with diabetes with poor glycemic

control easily develop severe lower limb infection and even diabetic foot, and are at a higher risk of lower extremity amputation, There is increasing evidence that reduced ankle joint ROM, static stiffness of the Achilles tendon and the diabetic neuropathy may be responsible for the development of diabetic foot [23].

However, not all the patients with DM are symptomatic, some of them are asymptomatic, even with an increased thickness or structural abnormalities in supraspinatus and biceps tendons and Achilles tendons found with imaging examinations [24]. It is suggested that the insensitive neuropathy may reduce or even block the transmission of pain signal, thus leading to asymptomatic conditions in some patients with DM [23].

Asymptomatic structural alterations of AT in patients with type 2 diabetes have been documented in numerous studies. Ultrasound abnormalities with defects focused on the enthesis, including disorganized fibrillar pattern, thickening, accumulation of fluid and amorphous tissue, hypo- and hyperechoic areas, and reduced neurotendinous vascularization [17].

Radiological imaging of Achilles tendon

Ultrasound

Ultrasound (US) performed with high-resolution linear-array probes has become increasingly important in the assessment of ligaments and tendons around the ankle. US can provide a detailed description of normal anatomic structures , effective for evaluation of ligament integrity. In addition, it allows the performance of dynamic maneuvers, which may contribute to increase visibility of normal ligaments and improve detection of tears. It can facilitate accurate identification, localization and differentiation between synovial, tendinous and entheseal inflammation as well as joint, bursal and soft tissue fluid collection [25].

Ultrasonography (US) is a reliable imaging method to evaluate soft tissue structures, such as muscles, tendons, aponeurosis, and ligaments. it allows in vivo visualization of the biological integrity of the tendon. It is a safe, rapid, non-invasive, free of ionizing radiation, relatively inexpensive and popular method used in the assessment of Achilles tendinopathy. The technique also allows easy contralateral comparison. Furthermore, ultrasound is increasingly being used to direct therapeutic interventions for Achilles tendinopathy refractory to conservative treatment regimes, thus offering an alternative to surgical management. Ultrasonography carries a high positive predictive value for Achilles tendinopathy [26].

For tendinopathy, US reveal structural abnormalities, such as tendon thickening, textural heterogenicity with intra-tendinous focal hypoechoic areas, and altered vascularity [27]. Ultrasound is as good as MRI in the diagnosis of tendinopathy and full thickness tear, however MRI is more superior in the diagnosis of partial thickness tear, and in the differentiation of the different types of tendinopathy [28].

The advantages of ultrasound over MRI are low cost and the possibility to perform dynamic examination, Dynamic evaluation of the tendon and surrounding structures in long axis is achieved by actively or passively plantar and dorsiflexion of the foot, The morphology and the dimensions of the tendon can be assessed with a 7.5 MHZ or higher probe. The disadvantage of ultrasound is operator dependence, as its reliability appears to correlate with the experience of the examiner [29].

Sonographic assessment of Achilles tendon is performed with the patient prone position, and feet hanging over the end of the examination bed , as this allows plantar and dorsiflexion of the foot to be performed while conducting imaging. Comparison to the asymptomatic contralateral side is easy to

perform, The ultrasound probe should be aligned perpendicular to the tendon fibers to avoid artifacts from acoustic anisotropy, which can simulate the appearance of tendinopathy [28], The Achilles tendon should be scanned from its myotendinous junction to its calcaneal insertion in transverse and longitudinal planes. The Achilles tendon thickness should be measured on transverse images, because longitudinal images may yield measurement errors if the probe is tangential to the tendon [5].

The normal tendon is composed of fascicles of collagen fibers running in parallel, which appear as fibrillar hyperechoic (brighter) bands, and a closely adherent paratenon on the longitudinal plane. The normal tendon shows no vascularity on color Doppler [29], on the transverse plane AT shows oval or elliptical tendon echoes, and the boundary between the AT and the surrounding soft tissues is clear ; there is no fluid accumulation in the posterior calcaneal bursa, and no calcification at the calcaneal attachment [30].

Conversely, in people with AT tendinopathy, presence of dark (hypoechoic) intratendinous regions and sometimes irregular contours of the tendon on Ultrasound, the fibrillar striation pattern is often altered as a result of a disorganization of the collagen fibers and a thickened and hypoechoic portion of the AT reflects an increase in the quantity of extracellular matrix and [30], This will typically translate to focal thickening along the AT, Tendon thickening greater than 6 mm has been commonly used as a diagnostic criterion for Achilles tendinopathy[27], as well as the presence of dark (hypoechoic) intratendinous regions and sometimes irregular contours of the tendon on Ultrasound, with loss of the normal anterior concavity of the tendon on transverse images[31], Most tears and tendinopathy lesions occur approximately two to six centimeters from the insertion of the Achilles tendon on the calcaneus (the pre-insertional area) [32].

Aside from the thickening of the AT, other features, like abnormal morphology (twisted, curved, overlapping, and highly disorganized fibers), and calcific degeneration have been identified as

degenerative changes affecting the AT [33].

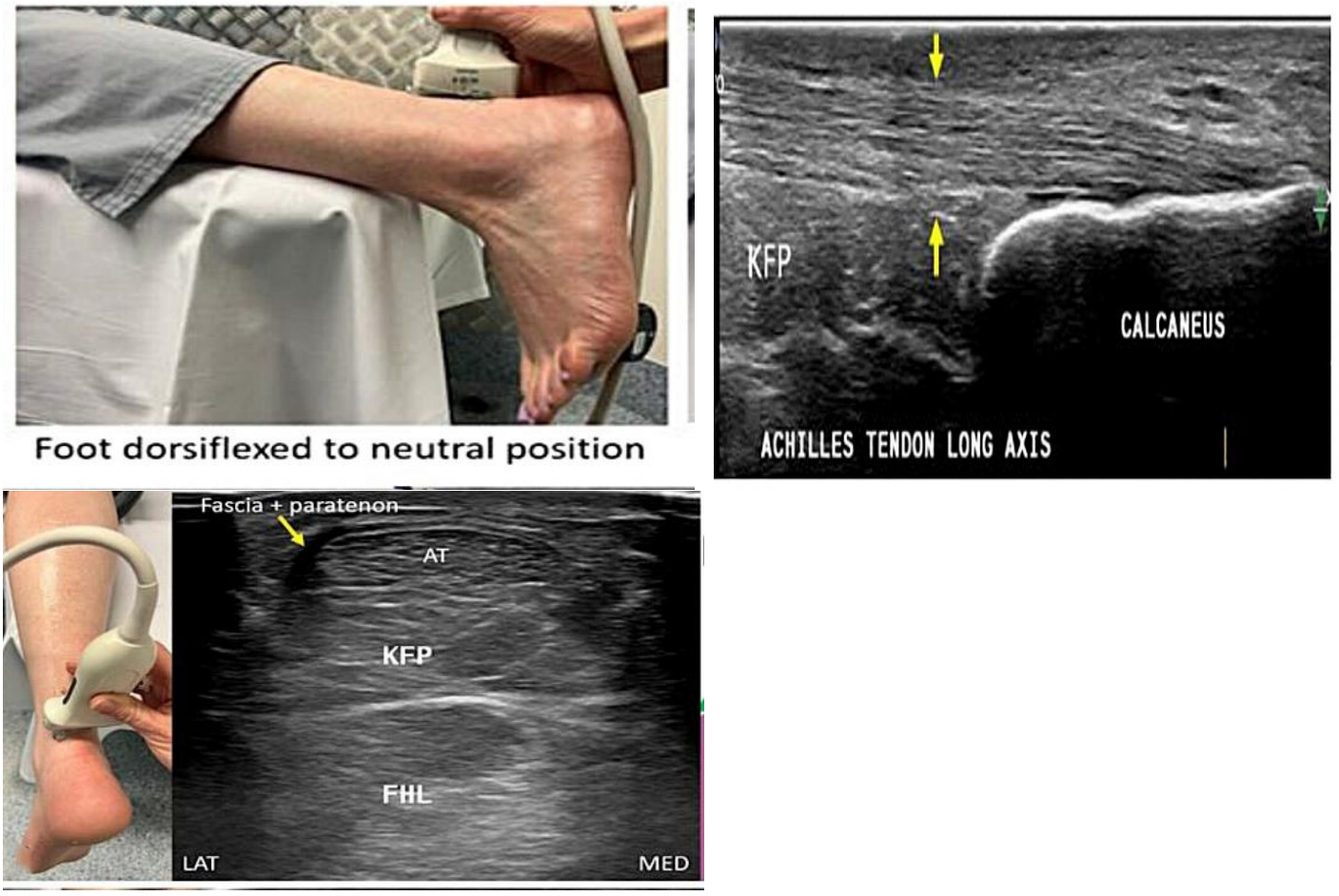

Fig. (1) Longitudinal and transverse images of normal Achilles tendon and enthesis. Achilles tendon, yellow arrows; Calc, Calcaneus, FHL, Flexor Hallucis Longus muscle; KFP, Kager's fat pad. LAT, lateral aspect of image; MED, medial aspect of image **[34].**

Ultrasound is useful in defining the difference between a complete and partial Achilles tendon tear because of the ability to image dynamically during ankle plantar flexion and dorsiflexion to better define the tendon anatomy, In full-thickness tears, the tendon usually exhibits a degree of retraction, with a gap present between the tendon ends The gap may increase during dorsiflexion, aiding evaluation [5].Partial tears are defined as the partial discontinuation of the Achilles tendon; They may be located at the surface of the tendon or intratendinous [35].

When an acute rupture of the Achilles tendon is present, the proximal and distal ends of the tendon will not appear continuous with long-axis sonographic imaging. Additionally, the fibrillar tendon echo pattern will be disrupted, and a focal defect extending between the superficial and deep borders of the tendon and between proximal and distal torn tendon edges will be observed, hematoma may be present at the site of injury and may be hyperechoic [34].

The plantaris tendon is not necessarily involved in an Achilles tendon tear and may remain intact On ultrasound, an intact plantaris tendon should not be mistaken for intact residual Achilles tendon fibers [5].

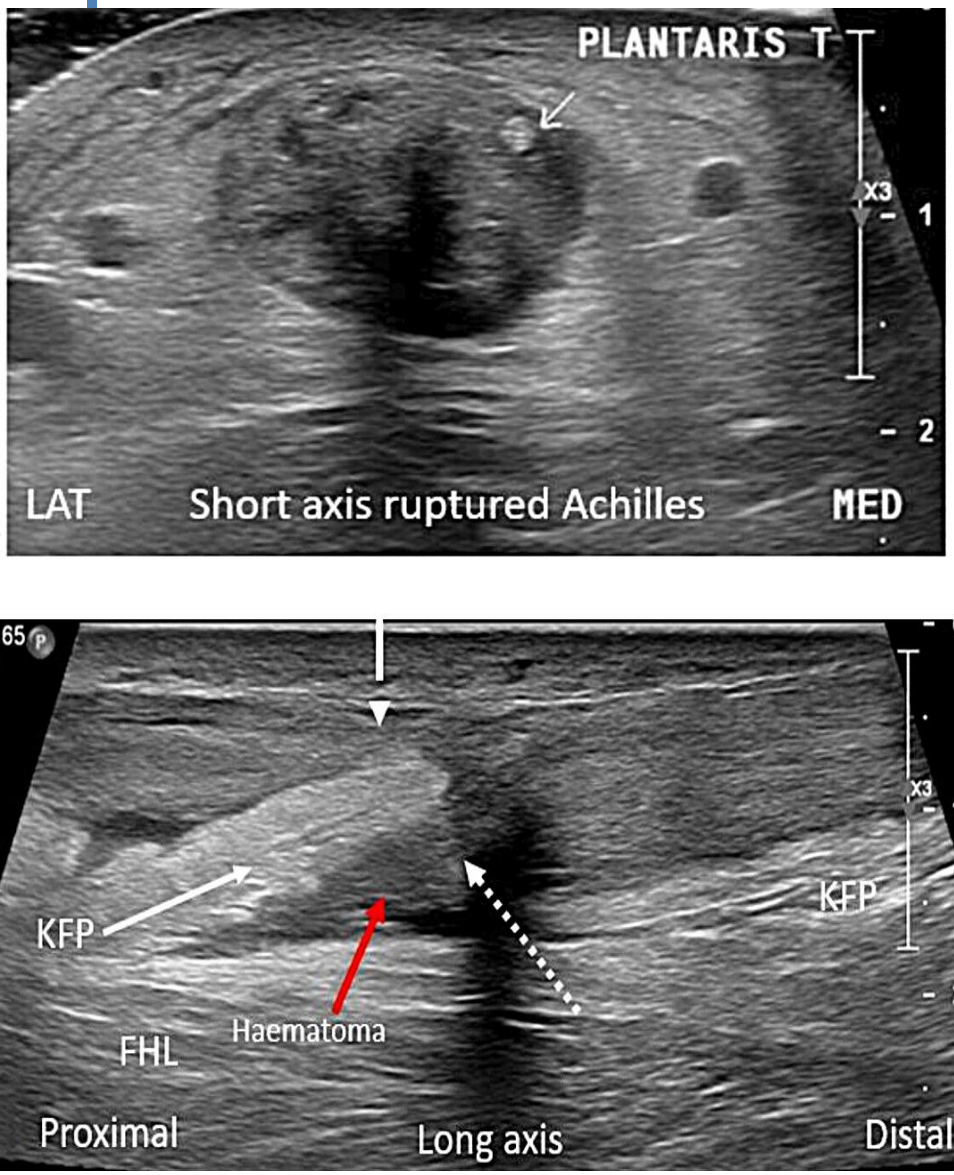

Fig. (2) Short-axis imaging of ruptured Achilles tendons and associated findings, Intact plantaris tendon. ((Long-axis sonographic imaging of acutely ruptured Achilles tendon (mid tendon)., Kager's fat pad (KFP) can be seen herniating superficially and inferiorly through the gap between the proximal (dashed arrow) and distal (dotted arrow) tendon ends. FHL, Flexor hallucis longus muscle; KFP, Kager's fat pad; LAT, lateral aspect of a short axis image; MED, medial aspect of a short axis image [(34)]**.**

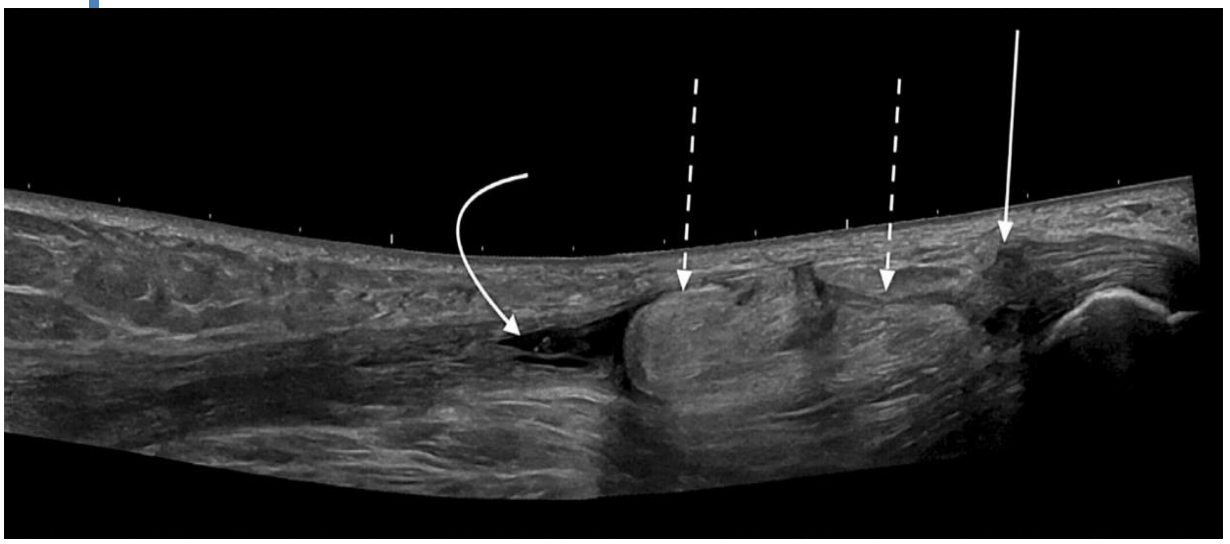

Fig. (3) Longitudinal ultrasound image shows chronic full-thickness Achilles tendon tear. Distal end of tendon (*straight solid arrow*) is in normal position. Proximal end (*curved arrow*) is retracted with adjacent fluid. Fat from Kager fat pad (*dashed arrows*) is interposed in gap **[5].**

Ultrasonography evaluation is usually the first-line imaging examination for patients with suspected Achilles tendinopathy since it is widely available, has a relatively low cost and provides a real-time assessment [36], though, conventional ultrasound images can only provide information about the anatomical and morphological features of the AT and cannot assess tendon mechanical properties. More recently, AT stiffness and elasticity have been evaluated with different ultrasound-based technologies, in particular shear wave elastography (SWE) and strain elastography (SE) [37].

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