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Ramon Balius, Mireia Bossy, Carles Pedret, Carme Porcar, Xavier Valle, Hèctor Corominas

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HEEL FAT PAD SYNDROME BEYOND ACUTE PLANTAR FASCITIS.

Ramon Balius [1,2], Mireia Bossy [2,3], Carles Pedret [2,3] Carme Porcar [4,] Xavier Valle [5] and Hèctor Corominas [2,6]

[1] Consell Català de L´Esport. Generalitat de Catalunya. Barcelona. Catalonia Spain.

[2] Clínica Diagonal. Barcelona. Catalonia. Spain.

[3] Clínica Creu Blanca. Barcelona. Catalonia. Spain.

[4] Centre Alt rendiment. Sant Cugat del Vallès. Catalonia. Spain.

[5] Futbol Club Barcelona. Serveis Mèdics. Barcelona. Catalonia. Spain.

[6] Servei de Reumatologia. Hospital Universitari de Sant Pau, Barcelona, Catalonia. Spain

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***Corresponding Author:**

Hèctor Corominas MD, PhD

Servei de Reumatologia i malalties autoimmunes sistèmiques Hospital Universitari de la Santa Creu i Sant Pau, Barcelona Universitat Autònoma de Barcelona (UAB)

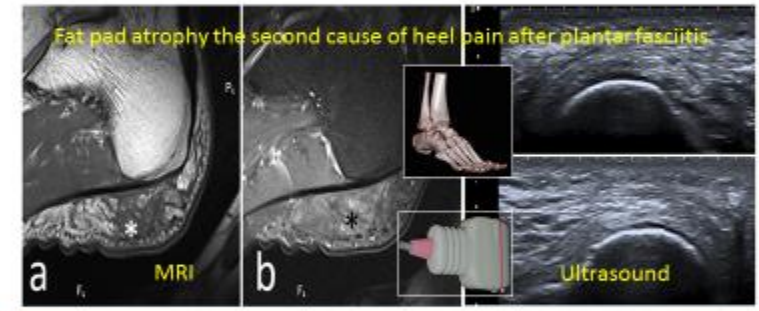
vancor@yahoo.com

Mas Casanovas, 90

Telèfon: 935565609

08025. Barcelona, Catalonia. Spain

Graphical abstract



Fibrosis and atrophy. MRI study in T1 sequence (a) and T2-STIR sequence (b) characterized by changes in signal intensity with low-signal intensity bands (*). Short-axis (c) and long-axis (d) of grey scale ultrasound where a thickness reduction of heel fat tissue is observed with overall normal echogenicity.

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HIGHLIGHTS

- Heel pain is a frequent cause of pain and disability in adult active population
- Pathological findings of the heel fat pad area using MRI and US can provide a differential diagnosis of heel pain beyond plantar fasciitis
- Observational case series study of patients with fat pad atrophy in the plantar aspect of the heel with a bed-side exam with US

ABSTRACT

Heel pain is a frequent cause of pain and disability in adult active population. In patients with this clinical presentation, several causes must be ruled out, among them plantar fasciitis the most common. Other etiologies of plantar heel pain are the entrapment of muscular branch of the lateral plantar nerve (Baxter nerve) or fat pad atrophy, being the last one the second cause of heel pain after plantar fasciitis.

We describe a case series of patients with pathological findings of the heel fat pad area using MRI and US to provide a differential diagnosis of heel pain.

Observational case series study. Nine patients visited presented with pain in the plantar aspect of the heel.

The plantar aspect of the heel was evaluated in detail with US and MRI. Main inclusion criteria were to present acute or chronic pain on the plantar aspect.

In five cases the right heel was affected, in three cases the left heel. One case presented bilateral complaints. All patients presented mechanical pain. Specifically, four of them also described a constant clunk during footstep. Heel fat pad lesion was confirmed with MRI and US in the medial aspect, observed in five patients. In four patients, the heel fat pad was globally affected respectively.

This case series tries to put some light on other heel conflicts beside plantar fasciitis that should be ruled out, being one of those, heel fat pad atrophy. Our presentation highlight the role that bed side ultrasound can play in the definition of a specific pattern confirmed with MRI after the US.

Keywords: heel fat pad, fat atrophy, ultrasound, magnetic resonance

INTRODUCTION

Heel pain is a frequent cause of pain and disability in active adult population [1]. When pain is presented in the plantar aspect of the heel, several causes should be ruled out, among them plantar fasciitis is the most common one. In 2010, the American College of Foot and Ankle Surgeons (ACFAS) guidelines recognized

fasciitis (or fasciosis) presenting with or without spur that was the outstanding cause of plantar heel pain [2].

Plantar fasciitis can present as a degenerative process with micro tears (fasciosis) similar to tendinosis (a degeneration of collagen in tendons), and fascial thickening over inflammatory changes. However, other etiologies of plantar heel pain such as the entrapment of muscular branch of the lateral plantar nerve (Baxter nerve), limited ankle dorsiflexion with swelling or atrophy of the heel fat pad that are mandatory to be considered in the differential diagnosis. Yi et al., described in 2011 that fat pad atrophy is the second most common cause of heel pain (14.8%) after plantar fasciitis (53.2%) [3].

The heel pad consists of adipose tissue enclosed within a structure of elastic fibrous septa extending from the skin to the calcaneus periosteum [4, 5, 6]. These septa form a structure of two micro-chambers (one rigid and superficial, the other deeper and deformable) when loading, force the heel pad to play a role of hydraulic shock absorber [1, 7, 8]. Heel fat pad anatomy can be evaluated either by MRI, US or both (Figure 1 and Figure 2).

Every single footstep produces a smashing impact of about 110% on body weight when walking, reaching up to a 250% when running. In those scenarios, the fat pad can only absorb an 80% of this energy [9]. During working, sport activities or using continuously defective footwear, heel fat pad overloads and acute injuries may occur to the fibro-adipous septa. Clinical exam is somehow not enough to confirm the final diagnosis, thus, radiological techniques such as ultrasonography (US) and magnetic

resonance imaging (MRI), beside the clinical examination, are essential to evaluate the soft tissue complications.

The main goal of this case series is to describe the pathological findings of the heel fat pad area using US and MRI to provide better imaging information on differential diagnosis of heel pain to, therefore, prescribe the appropriate treatment.

MATERIALS AND METHODS

We present a case series manuscript of nine patients prospectively visited in our outpatient clinic of different sports medicine and rheumatology health care facilities that presented with pain in the plantar aspect of the heel and were evaluated in detail with US and MRI.

The main inclusion criteria for this study were to present acute or chronic pain on the plantar area, specifically in the calcaneal region. Patients were excluded if they had received previously any corticosteroid injection in the talus region. Among the group of patients, two were tennis players, two taekwondo (TKD) athletes and five track and field athletes (4 long-distance runners and one triathlete). One of the cases complained about bilateral pain. First examination was made with US, thereafter if some evidence of heel fat pad injury was observed, an MRI was performed as well. Ultrasound study was performed using a Toshiba Medical Systems model SSA-770, "Aplio 80 XV" (Nasu, Japan), with a 5-18 MHz probe model PLT 805AT with a transducer length of 67 mm and a viewing width of 58 mm that rendered unnecessary any kind of electronic view widening (Panoramic or Trapezoid View). The most common frequency used was a pulse subtraction harmonic frequency of 9-12 MHz with a gain range between 85 and 95, a dynamic range of 70 and a depth of 2 to 3 cm focused on a single focus at 0.5-1 cm. The ultrasound was performed by two ultrasound experts (sports medicine and rheumatologist specialist), with up to 25-years of experience in musculoskeletal ultrasound.

Magnetic resonance imaging was conducted using a high-resolution 3.0-T MRI scanner (Magnetom VERIO, Siemens Medical Solutions) with a maximum gradient strength of 45 mT/m, a rise time of 225 μs and 32 receiver channels. Image acquisition was performed using a 3-surface coil with 1 element per coil, 7 cm in diameter. Coronal and axial turbo spin echo (TSE) T1-weighted sequences and axial, sagittal and coronal TSE fat-saturated T2-weighted sequences were performed and reviewed by a radiologist (XA) with more than 20 years of experience in musculoskeletal MRI.

US and MRI were used to evaluate heel fat pad atrophy and fibrosis as well as to detect septa defects with fluid in the surrounding tissues. The fibrosis and atrophy were classified using a numerical scale where 0 means no atrophy and/or fibrosis, 1 mild, 2 moderate, and 3 severe atrophy or fibrosis. The presence of edema and the integrity of the septa were also evaluated. Every single case was reviewed independently by the experts who had extensive experience in MRI (XA, RB) and US (CP, RB, HC). A consensus agreement was previously made (Fig. 3).

Finally, to show differences, a control group of 24 track and field healthy athletes (17 men and 7 women with a mean age: 29,3-year-old) were also evaluated using ultrasound measurements. The inclusion criteria were that they had never suffered from heel and/or plantar pain and that they had trained more than 5 years continuously in the track and field mode.

The unloaded heel-pad thickness (UHPT) and loaded heel-pad thickness (LHPT) measurements were made by US in accordance with the Uzel criteria in 7 of the 9 patients [10, 11] and in the control group. The heel pad compressibility index (IC) was also US-evaluated using the definition by Prichasuk: ratio between heel pad

thickness in loaded and unloaded positions [12]. All patients included have mentioned their informed consent for participation in the clinical and ultrasonography & MRI research study.

RESULTS

In five cases, the right heel was affected, in three the left heel was affected, and one case bilaterally. All patients complained about mechanical pain, specifically four of them also described a constant clunk during footstep (snapping). Physical evaluation showed heel fat pad hypermobility in 7 of the 9 patients. The group description data are shown in Table 1.

The fibrosis and atrophy categorization are shown in Table 2. Heel fat pad lesion was confirmed with MRI and US in the medial aspect. In five patients the heel fat pad was globally affected. In two cases the heel fat pad injury was associated with plantar fasciitis. The UHPT, LHPT and IC data are shown in Table 3. The cases of heel fat pad injury and plantar fasciitis were treated with ultrasound-guided injection with Pure Platelet-Rich Plasma (P-PRP). P-PRP was obtained by using the PRGF-Endoret technique (BTI Biotechnology Institute, Vitoria, Spain). Only one patient required surgical treatment, the free tissue was removed, and the virtual spacewas closed by suturing. The rest of patients were treated with rest, physiotherapy and silicone orthesis (against vibrations). The average returning to play (RTP) was 2.8 (1,5-8) months.

DISCUSSION

Heel fat pain is a common and recurring complaint in sports medicine, rehabilitation and rheumatology clinics. Several factors have been considered to be the reason why patients present with such rather acute or chronic heel pain. Actually, the variability present in heel fat pad thickness may exist due to differences in race, gender, age or method (X-ray and US) [10, 11]. In a study on patients with plantar heel pain syndrome Uzel et al., in 2006 reported beside the clinical aspects of the disease, the following US measurements: UHPT 19.8± 2.9 mm, LHPT 12.3 ± 2.9 mm and IC 0.6 ± 0.09 mm. Interestingly, they also described a strong correlation between the US and X-ray measurements ($r = 0.84$, $P < 0.001$) [10, 11], leading to summarize that US is the ideal non-invasive method for evaluating heel fat pad thickness. Our data are similar to those from Uzel et al., especially regarding the IC (IC=0.6) [10, 11], and slightly higher than the data reported for a normal adult population between 20 and 34 years of age [13]. An increase in the heel fat pad compressibility index indicates a loss of elasticity [12]. Our group due to continuous sport activity may probably present an increase in IC which is because of this high physical activity as a major factor to suffer from acute heel injury. To date, it is clearly recognized that heel fat pad observed in sports men is thicker than in men [12, 13, 14, 15], presenting a progressive decrease in thickness with age and body weight due to the loss of water, collagen and elastin from the tissue. All these factors in conjunction lead to a reduction of the absorption of an impact and weakness and loss of calcaneus protection [1, 3, 13, 16]. Heel fat pad atrophy may be due to other aetiologies whose nature is neurological (tarsal tunnel syndrome, radiculopathy), rheumatological (rheumatoid arthritis) [17] or traumatic (stress fracture) and others

such as diabetes, tumors or infections [6]. Interestingly, heel fat pad atrophy has also been described as a side effect of corticosteroid infiltrations, a common accepted practice in cases of acute plantar fasciitis [3, 18, 19].

The influence of sports activity on heel fat pad thickness has not been widely studied. In a US study, Challis et al., found differences between runners and bikers in terms of heel fat pad thickness (1.49/1.36cm), deformation (4.8/4.3mm) and rigidity (20.7/17.5 $N \cdot mm^{-1}$) [20]. To this extent, physical activity does influence heel fat pad properties and may cause atrophy in young athletes [21]. On the other hand, Uzel et al, found no differences between sedentary people and athletes with an average physical activity of eleven hours per week, either in heel fat pad thickness (1,86/1.82cm) or IC (0.61/0.6) [10, 11]. Higher levels of physical activity may be necessary for premature reduction in heel fat pad thickness to occur. Data from the control group are very similar to those from Uzel et al., [11] and also the patient group, probably due to the high sports activity of the athletes of the control group.

Accordingly, very high and repeated levels of physical activity over long periods of time may affect the heel fat pad, causing atrophy, mostly asymptomatic. Sometimes a direct trauma on the altered fat pad produces an acute injury requiring medical assistance.

The dynamic exam by ultrasound showed clinical hypermobility detected in patients was related to the movement between both microchambers secondary and shearing forces.

Fibrosis was observed by MRI in 5 of the 10 cases, described as “changes in signal intensity, with low-signal-intensity bands” (Fig. 4). Although both MRI and US can evaluate the heel fat pad injuries [3], fibrosis is more difficult to detect by US (2 out

of 5 cases of them), probably due to difficultness to differentiate the echogenicity of the fibrosis itself from the echogenicity resulting from the loss of thickness in the heel fat pad (Figure 4).

In our series, atrophy in heel fat pad thickness was detected in 8 of the 10 cases. The atrophy was detected by both MRI and US (Figure 4) described as a bilateral reduction in heel fat pad thickness values [10, 11, 22, 23].

Both the MRI and US studies showed edema and defects in the fat pad septa.

In four cases, US and MRI detected septa affected by edema and small pseudocystic liquid collections (Figure 5). In three cases US detected free and mobile tissue with fluid surrounding (Figure 6). On the other hand, MRI is unable to detect this dynamic behavior of injured tissue. The most remarkable point in our series is that in 6 out of 10 cases there was a clear history of injury to a slightly atrophic heel fat pad. For instance, patient 7 was a triathlete who complained about hitting his naked heel directly against a stone during the transition from swimming to cycling; this injury caused the pain and septum rupture subsequently was observed by US and MRI (Figure 6).

In addition, septum rupture may often occur in old or obese patients due to the repetitive load of their own bodyweight and poor stress absorption as observed in rheumatologic and patients with chronic inflammatory diseases [1, 3, 13]. In such cases, inflammation of the heel fat pad may occur, causing pain and, to a lesser extent, fat pad degeneration and fibrous substitution [5]. In two of the cases, there were no clear previous injuries, but clunky heel landings were felt (cases 2 and 8); and in a single case (Figure 4), bilateral heel fat pad atrophy without previous

trauma was described. That patient was a long-distance runner with severe heel fat pad atrophy who complained about longstanding pain.

A common finding in most of the cases studied by MRI was heel fat pad edema, with poorly-defined areas of reduced signal intensity in T1 weighted images rising in signal intensity in T2 weighted images. In such cases, a hypoechoic area with altered structure is detected with US. Heel fat pad septum disruption in circumscribed areas was detected by both MRI and US, corresponding with pseudocystic areas in four of the cases (Figure 7). Yamakado et al., reported a similar case, describing it as subtalar bursitis commonly associated with heel fat pad atrophy and subsequent loss of cushioning effect that may lead to plantar fasciitis [9, 24].

In our four other cases, high density imaging of free septa tissue was detected. The dynamic US study of these cases using direct probe pressure showed how this dense material bulged sharply and painfully in anterior and medial direction to the calcaneus (Figure 8). In all cases the injury was located in the deep microchamber; even so, the new dissection plane was located between both microchambers. During the PRP infiltration, the liquid could clearly be observed spreading out between both fat layers.

The treatment of this heel fat pat syndrome, according to our experience and the existing literature, is based on the prescription of NSAIDs, the use of proper shoes, silicone heels and avoiding high impact activities. The cup-shaped silicone heel gave the best results; with that the calcaneus is blocked in its posterior, lateral and medial aspects, decreasing the heel fat pad displacements more than in any other heel models [1, 3, 21].

As we described above, a P-PRP treatment was prescribed in two cases, as a non-evidence-based treatment. The P-PRP action mechanism and preparation protocol are controversial but in both patients the symptoms and signs improved [25].

Surgical treatment is also controversial; some authors advise against it [5], while others recommend it [6, 24]. Surgical treatment was done in only one case because of its long outcome (3 months) and the clunk sensation. As a result of that, the patient got better although with some discomfort due to the atrophic residual tissue.

Our present report has some limitations not only due to the small sample of patients included, but also because this is only an observational case-control US exam. It is important to highlight the need of a high degree of expertise of the sonographers, Seeking this expert was hardly difficult. Nonetheless, once having the experts, our project eased the study and allowed us to order the MRI to confirm our US suspicion.

To summarize, we present this observational case series to put some light on a certainly common issue in clinics of different specialties. We want to emphasize that, not all cases of heel pain are due to plantar fasciitis, being the major and principal cause though. Other heel conflicts should be ruled out such as fat pad atrophy. Our presentation highlights the role that bed-side ultrasound can play in the definition of a specific pattern which we later confirmed with MRI. We consider that sports medicine physicians, rehabilitation, orthopedists and rheumatology specialists should be aware of this syndrome and reinforce the role of US to be implemented in their clinical daily practice.

Conflict of interest

Do not have any conflicts of interest.

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Figure Legends

Figure 1. MRI of the normal heel fat. Sagittal (a) and coronal (b) MRI T1 sagittal (c) and coronal (d) STIR. The arrowheads indicate the rigid superficial micro-chamber. *Asterisks highlight the deep macro-chamber deforming when weight bearing.

Figure 2. Grey scale ultrasonography of the normal heel fat pad. (a): long-axis view without pressure to evaluate the thickness. (b): long-axis view with probe pressure on the heel to evaluate decompression of the fat pad. The superficial micro-chamber (arrowheads*) and the deep micro-chamber (*) not as clear as MR observed in figure 1.

Figure 3. Different degrees of atrophy and / or fibrosis based on a numerical scale. A: No atrophy (A=0), mild fibrosis (F=1) and talar edema. B: Moderate atrophy (A=2) and moderate fibrosis (F=2). C: No atrophy (A=0), moderate fibrosis (F=2) and mild edema. D: Moderate atrophy (A=2) and severe fibrosis (F=3). E: severe atrophy (A=3) and mild fibrosis (F=1)

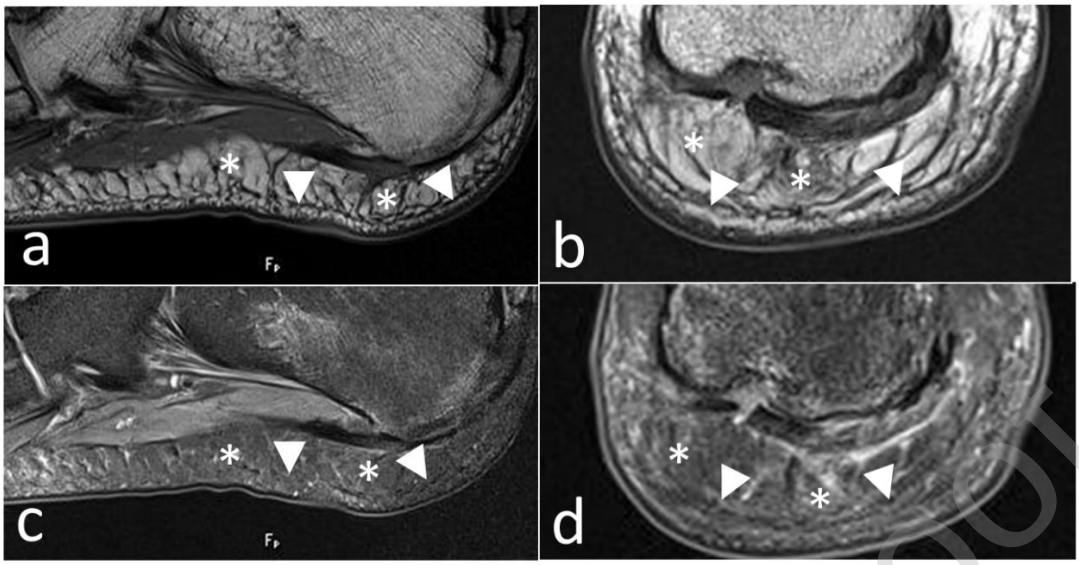
Figure 4. Fibrosis and atrophy (case 4). MRI study in T1 sequence (a) and T2-STIR sequence (b) characterized by changes in signal intensity with low-signal intensity bands (*). Short-axis (c) and long-axis (d) of grey scale ultrasound where a thickness reduction of heel fat tissue is observed with overall echogenicity.

Figure 5. long-axis (a) and short-axis (b) ultrasound study (case 3). Echoic septal free tissue (*) with hypoechoic fluid.

Figure 6. Heel fat with septal defect injury (arrows) and pseudocystic fluid (*) (case 7). Coronal T1 (a) and coronal T2 (b) MRI. Long-axis US without (c) and with probe pressure showing fluid disappearance (d) similarly to the defect observed when wandering.

Figure 7. Heel fat with defect (arrows) and septal free tissue (*) (case 1). Sagittal MRI T2 (a). Long-axis (b) ultrasonography without probe pressure with dense material observed.

Figure 8. Heel fat with dense material (*) (case 2). US study on long axis without probe pressure (a) and with probe pressure (b) where the previous highlight of septal free tissue (arrow) is shown.



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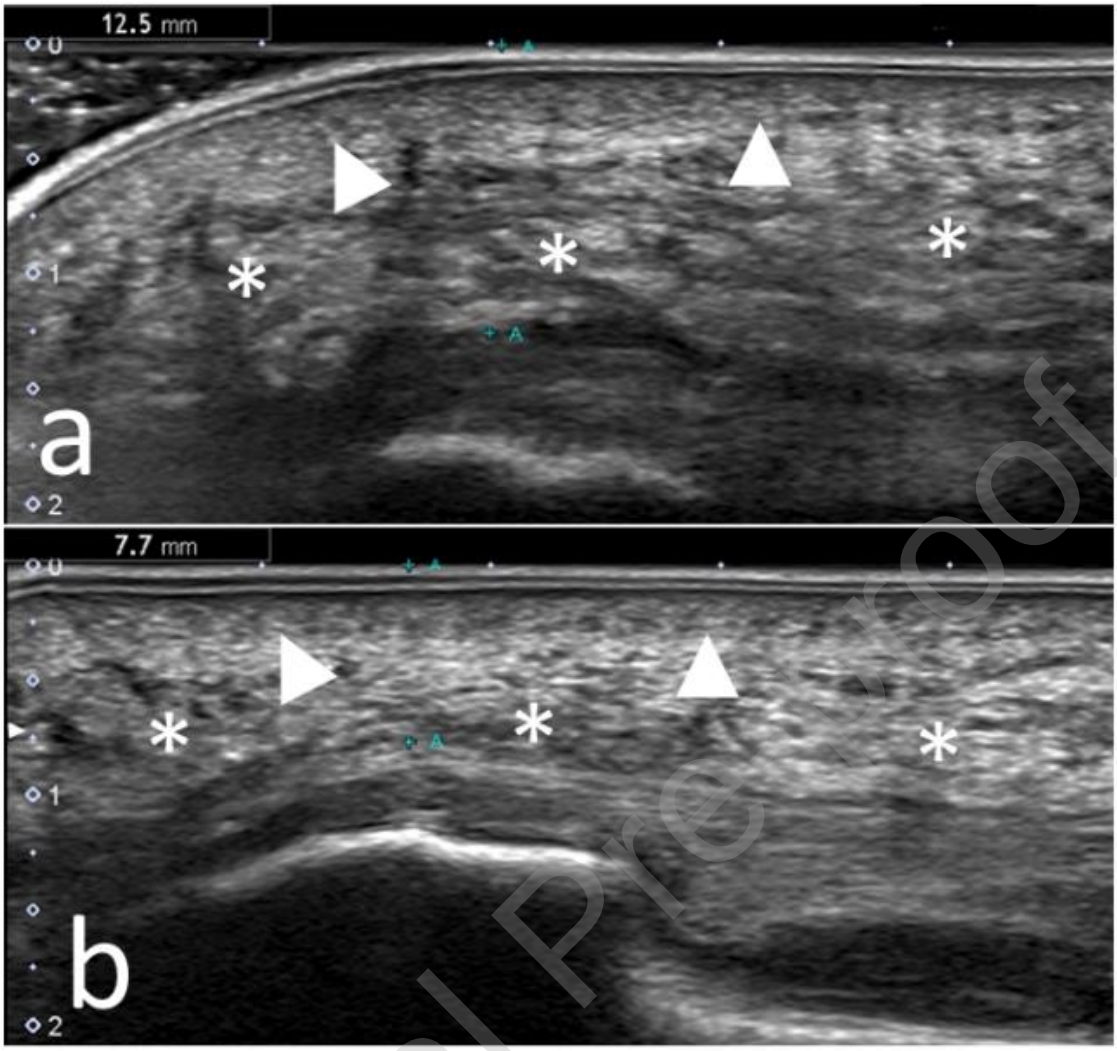

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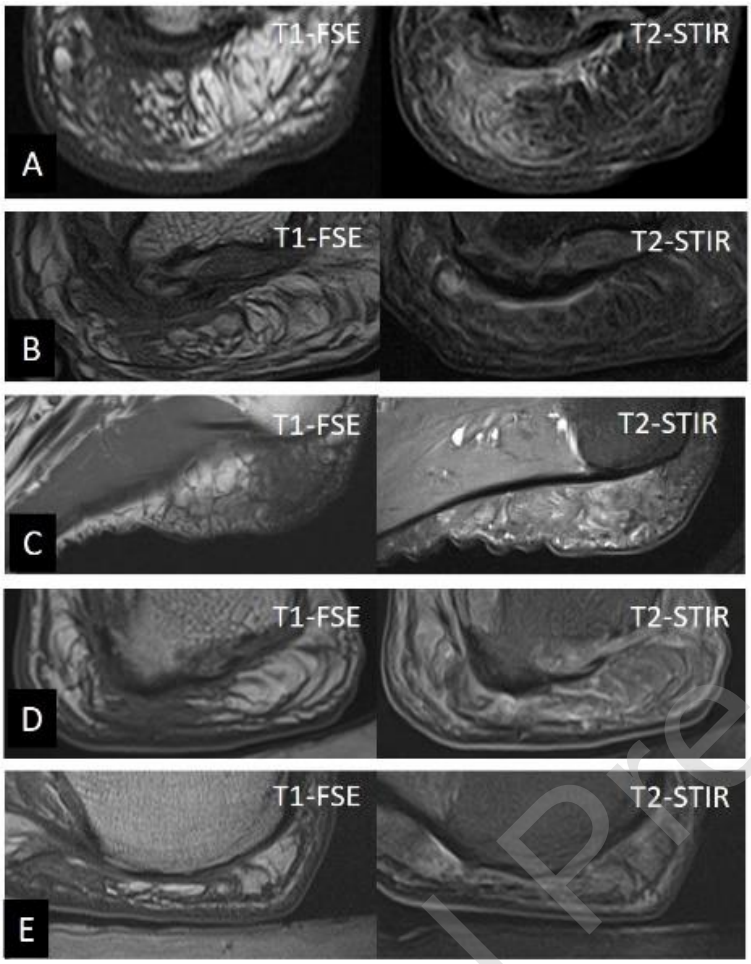

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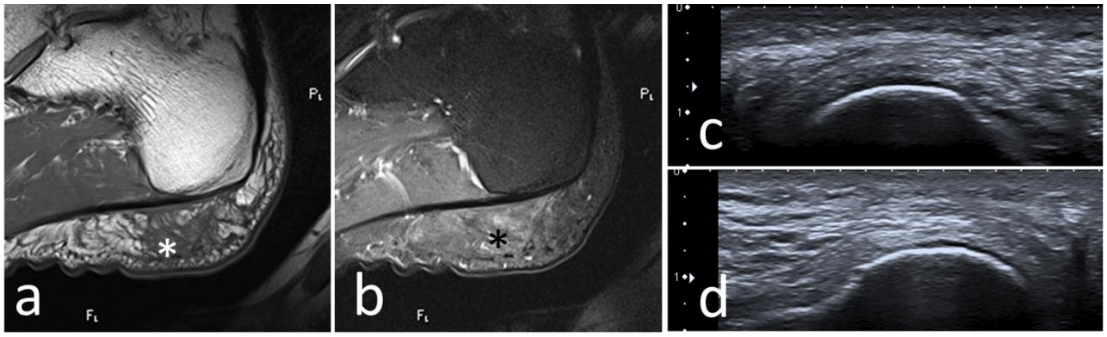

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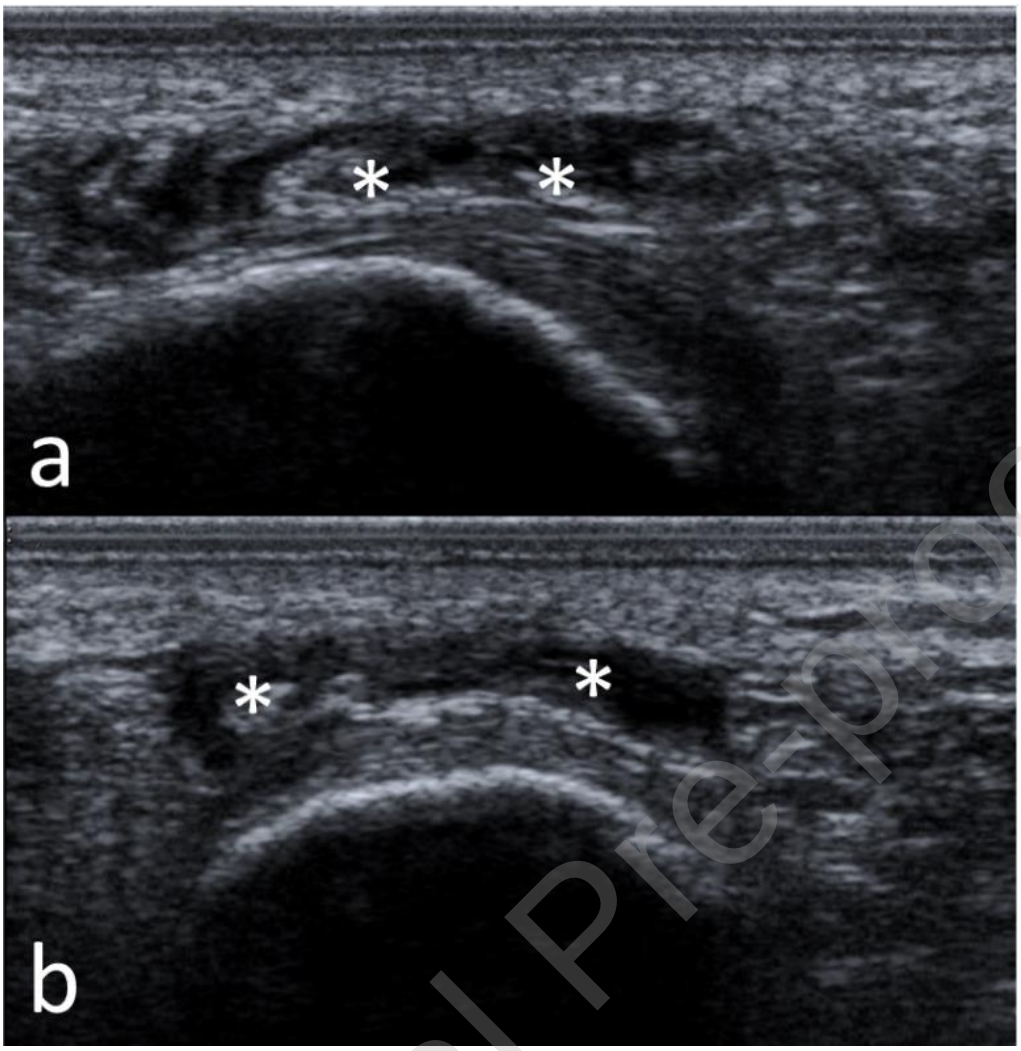

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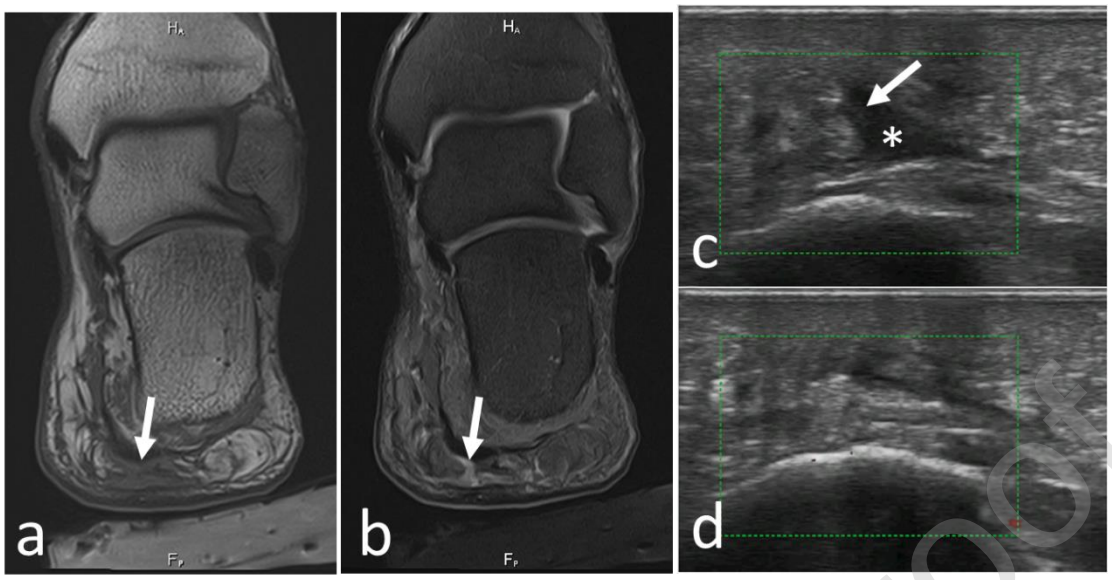

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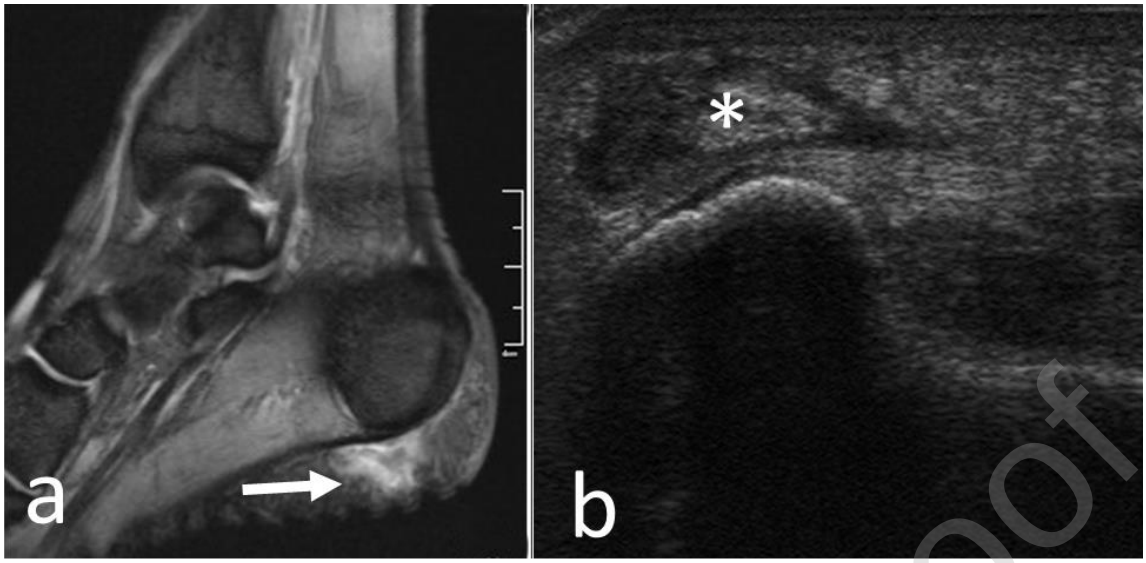

Figure 7. Heel fat with defect (arrows) and septal free tissue (*) (case 1). Sagittal MRI T2 (a). Long-axis (b) ultrasonography without probe pressure with dense material observed.

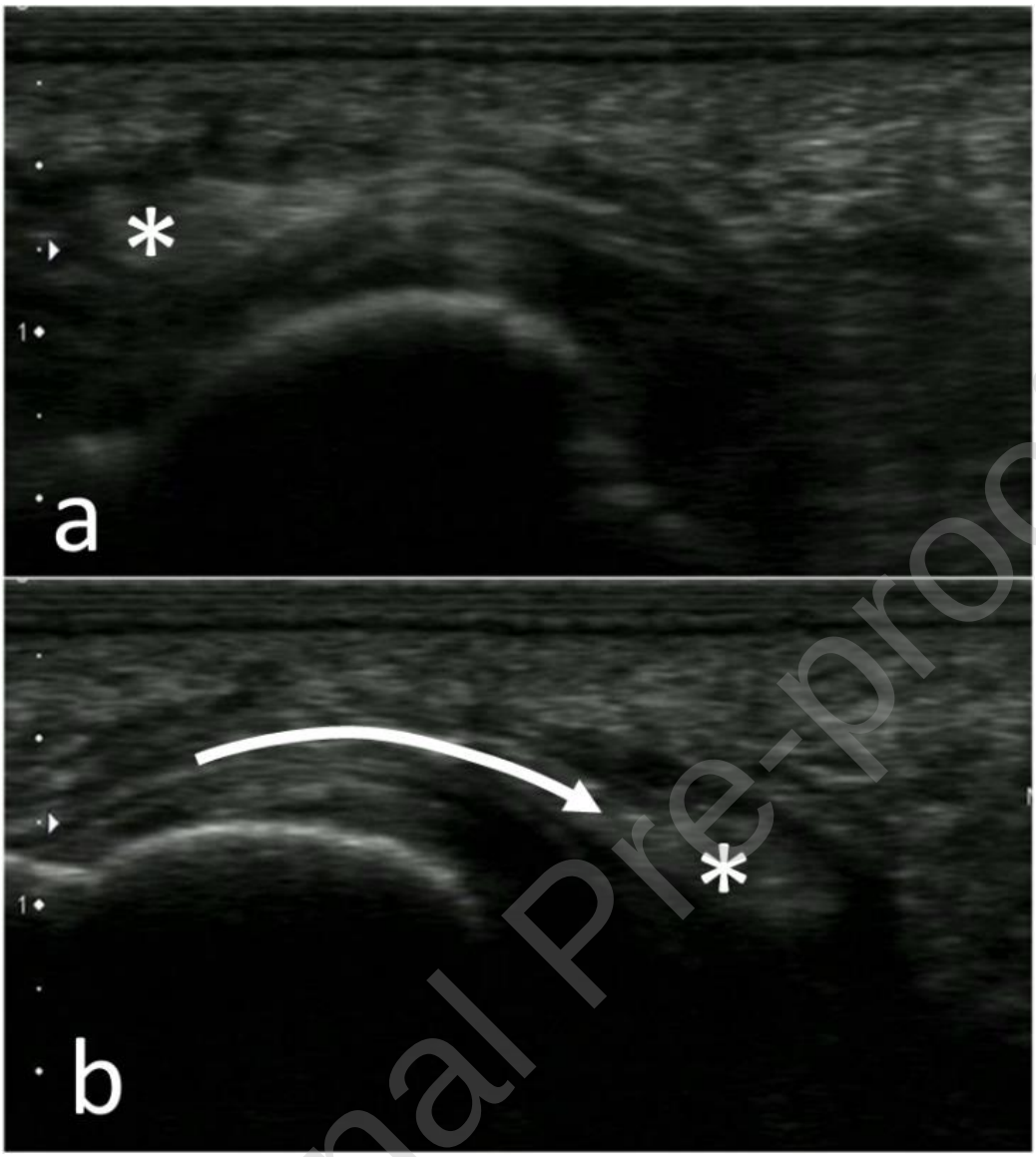

Figure 8. Heel fat with dense material (*) (case 2). US study on long axis without probe pressure (a) and with probe pressure (b) where the previous highlight of septal free tissue (arrow) is shown.

Table 1. Group description: sport, heel side, trigger event, clinical findings & treatment

	Age	Sports	Heel	Delay in consultation (days)	Traumatic background	Clinical findings	Hipermobility	RM	US	Injury associated	RTP (months)	Treatment
1	18	Tennis	Right	15	Acute pain after a jump on the heel.	Mechanical Pain	no	+	+	no	2	Orthopedic
2	35	TKD	Right	90	no	Mechanical Pain and snap	yes	+	+	no	8	Surgery
3	30	Runner	Right	30	Acute pain after a jump on the heel.	Mechanical pain and snap	yes	+	+	no	2	Orthopedic
4	28	Runner	Bilateral	365	no	Mechanical Pain	yes	+	+	no	4	Orthopedic
5	49	Runner	Right	30	Acute pain after a jump on the heel.	Mechanical Pain	yes	+	+	plantar fasciitis	2,5	Orthopedic (PRP)
6	24	Tennis	Left	15	Acute pain after a jump on the heel.	Mechanical Pain	no	+	+	no	1,5	Orthopedic
7	30	Triatlon	Right	60	Acute pain after a jump on the heel.	Mechanical Pain	yes	+	+	no	2,5	Orthopedic (PRP)
8	34	Runner	Left	30	no	Mechanical Pain and snap	yes	+	+	Plantar fasciitis	1,5	Orthopedic
9	31	TKD	Left	30	Acute pain after a jump on the heel.	Mechanical Pain and snap	yes		+	no	1,5	Orthopedic
	31			73,8							2,8	

Table 2. MRI and US findings: fibrosis and atrophy categorization**

	MRI Edema	US Edema	MRI Septa	US Septa	MRI Atrophy	US Atrophy	MRI Fibrosis	US Fibrosis	Retinacula Injured	Content retinácula
1	yes	yes	rupture	rupture	+	+	-	-	Medial	Pseudocyst
2	non	non	rupture	rupture	++	++	++	+	Medial	Free septal tissue
3	yes	yes	rupture	rupture	++	++	++	-	Medial	Free septal tissue
4 (right)	non	non	normal	normal	+++	++	++	+	Global	non
4 (left)	non	non	normal	normal	++	+++	++	+	Global	non
5	yes	non	normal	rupture*	-	-	-	-	Medial	non
6	yes	yes	rupture	rupture	+	+	-	-	Medial	Pseudocyst
7	yes	non	rupture	rupture	-	-	-	-	Medial	Pseudocyst
8	yes	yes	rupture	rupture	+++	++	+	-	Global	Free septal tissue

* Observed when PRP infiltration ** All observations were assessed by two experts with previous inter-reader reproducibility evaluation.
accordingly to a previous consensus.

Table 3. Unloaded heel-pad thickness (UHPT), Loaded heel-pad thickness (LHPT), Heel-pad compressibility index (HPCI). Nd: not done

	Heel injured	Right UHPT (mm)	Right LHPT (mm)	Right HPCI	Left UHPT (mm)	Left LHPT (mm)	Left HPCI
1	Right	11	7	0,6	14	9	0,6
2	Right	nd	nd		nd	nd	
3	Right	12,1	8,1	0,7	15	10	0,7
4	Bilateral	10,3	5,2	0,5	10	5	0,5
5	Right	10,2	7,9	0,8	12,5	5,4	0,4
6	Left	18,5	12,6	0,7	19	13	0,7
7	Right	nd	nd		nd	nd	
8	Left	18,4	10,3	0,6	15	10	0,7
9	Left	19,8	12,4	0,6	18,2	12,4	0,7
				0,6			0,6

UHPT: Unloaded heel-pad thickness: 19.8 ± 2.9 mm
LHPT: Loaded heel-pad thickness: 12.3 ± 2.9 mm
HPCI: Heel-pad compressibility index: 0.6 ± 0.09 mm
Nd: not done