Original Article

Magnetic resonance imaging findings of plantar venous thrombosis: a comparative study with Doppler ultrasound

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Abstract

Objective: Analyze magnetic resonance imaging (MRI) findings, demographics and clinical data in patients with plantar venous thrombosis (PVT).

Methods: A retrospective study screening patients with PVT, diagnosed with color Doppler ultrasound, who also underwent MRI of the foot and ankle. In the post-contrast MRI sequences, the intravascular filling defect was analyzed, and in the non-contrast MRI sequences, the diameter of the foot vessels was analyzed, and perivascular edema was documented. MRI exams from a control group without PVT were also analyzed.

Results: Sixty-one cases and 204 controls were included. The lateral plantar veins were the most frequent location in the foot with PVT (63.9%). In all post-contrast sequences, an intravascular filling was seen. In the case group, veins had a significantly greater diameter compared to controls (p < 0.05), and perivascular edema was observed in all cases (100%). A history of trauma/mechanical overload of the foot was documented in 83% of the patients (p < 0.001).

Conclusion: Our results showed that the lateral plantar vein was the most frequent location of PVT. An intravascular filling defect on post-contrast MRI sequences was seen in all PVT cases. Compared to the control group, all vessels had a greater diameter in the case group, and perivascular edema was observed in all cases. Our results also suggest that a clinical history of trauma or mechanical overload (physical activities) in the foot and ankle may be one of the causal factors of this pathology.

Level of evidence III; Therapeutic studies - investigating the results of treatment; Retrospective comparative study.

Keywords: Magnetic Resonance Imaging; Venous thrombosis; Thrombosis; Foot and ankle.

Introduction

Plantar venous thrombosis (PVT) is an underdiagnosed cause of pain on the plantar surface of the foot. It is characterized by the formation of a thrombus within the deep plantar veins, and its clinical symptoms are nonspecific: usually pain, local edema, and tenderness to palpation, which may be associated with difficulty in gait and functional loss. This symptomatology may be confused with other more frequent causes of pain syndrome on the plantar surface of the foot, such as plantar fasciitis, musculotendinous injuries, stress fractures, plantar plate injuries, bursitis, and neuromas⁽¹⁾.

Drainage of the deep venous system of the foot starts from the digital plantar veins, which originate on the plantar surface of the toes, uniting to form the metatarsal veins located in

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the intermetatarsal spaces, which then form the deep plantar venous arch. They follow the plantar arterial arch and give rise to the medial and lateral plantar veins, which unite behind the medial malleolus to give rise to the posterior tibial veins⁽¹⁻⁴⁾.

The pathophysiology of PVT is still controversial. In addition to sharing common risk factors for thrombosis in any part of the body, such as recent surgery, trauma, infection, neoplasms, and oral contraceptives, mechanical stress on the plantar surface of the foot can cause microtrauma and damage to the endothelial wall, acting as a key factor in Virchow's triad (hypercoagulability, venous stasis, and endothelial injury) and activating the blood clotting cascade⁽⁵⁾.

Doppler ultrasound is the most used imaging modality to diagnose thrombosis and is an accessible and inexpensive method, therefore are some limitations to its use, the fact that this method is examiner-dependent, the access of veins that are deeply located, and frequently the study of the plantar veins is not part of the routine protocol for investigating foot pain⁽²⁾. On the other hand, magnetic resonance imaging (MRI) can assess all foot structures simultaneously and threedimensionally, with good differentiation between structures. In addition, intravenous contrast facilitates visualization of plantar thrombi through imaging of vascular filling defects. Computed tomography provides limited assessment of the soft tissues of the foot and may not be considered for this indication. PVT has been believed to be a relatively innocuous entity. However, significant complications are associated with this condition, notably including the extension of the thrombus to the proximal veins and the development of pulmonary thromboembolism (PTE), underscoring the potential severity and clinical significance of these complications, necessitating heightened attention and prompt intervention (6-8).

Considering the probable underdiagnosis of this condition and the fact that MRI is frequently used in the investigation of painful foot syndrome, the purpose of this study is to analyze MRI findings, demographical and clinical data in patients with PVT, confirmed with color Doppler ultrasound.

Methods

Study design and patient selection

A retrospective search was performed on exams from 2017 to 2022 using the institution's PACS system (VR12, Phillips) by screening patients who also underwent contrast-enhanced MRI of the foot and ankle within seven days of the Doppler ultrasound examination with the diagnosis of PVT.

The inclusion criteria were patients with a clinical history of acute foot pain and thrombosis, as confirmed by a color Doppler ultrasound study-considered the gold standard, defined by non-compressible venous segment and absent color flow.

Superficial phlebitis cases, patients with diabetes, vasculopathy, vascular malformations, infection, postsurgical cases, and unsatisfactory technical quality were excluded.

Therefore, 86 cases were obtained. Twenty-five cases were excluded: Fifteen cases with superficial phlebitis, five cases

with vascular malformations, four cases with unsatisfactory technical quality, and one case of osteomyelitis.

The final case cohort included 61 patients that met all inclusion and exclusion criteria for PVT. All patients had a clinical history of acute-recent foot and/or ankle pain.

Control group

A control group was also selected, matched for age, sex, and body mass index (BMI), who underwent contrast-enhanced MRI of both feet and ankles without thrombosis on imaging studies during the same period, excluding postsurgical cases, diabetic feet, cases with artifacts and/or cases with vasculopathy.

For the control group, 242 cases were initially selected: all without any imaging signs of thrombosis and no clinical findings suggestive of thrombosis. After a detailed review, seven cases with vasculopathy, six cases with infection impairing the review of all vascular segments, and five cases with partial amputation of the foot/ankle were excluded. Finally, 224 patients were included in the control group.

Demographics and clinical data

Clinical information was collected from questionnaires completed by the patients before the exams. These questionnaires included information on sex (male and female), age (years), weight (kg), height (m), and clinical symptoms (pain, pain and swelling, pain and paresthesia, nodulation, paresthesia) at the time of the examination and duration of symptoms (days). The questionnaires also documented the use of oral contraceptives (yes vs. no), comorbidities (diabetes, hypertension, cancer, cholesterol, neurological disease, thrombophilia, and postoperative), history of recent physical exercise, and recent foot trauma (yes vs. no).

Ultrasound acquisition

A HD11 XE ultrasound machine (Philips, Andover, MA, USA) with a high-resolution 7.5-12MHz transducer was used. All the main foot and ankle veins were imaged with high-resolution multi-linear array transducers, and the location and extent of thrombosis were recorded in detail. The Doppler ultrasound was the gold standard: In ultrasonography, thrombosis was defined by absent flow on color Doppler study with no vessel compressibility (Figure 1) as previously described⁽⁹⁾.

MRI acquisition

All included patients underwent a complete contrastenhanced MRI of the foot or ankle. The exams were performed using a 1.5 Tesla MRI system (GEM, GE Healthcare) with a dedicated foot and ankle coil (GE Healthcare). In the ankle exams, the patient was in a supine position with the foot perpendicular to the table axis. In the foot exams, the patient was a prone position with the foot in plantar flexion using the same coil. The routine protocol of the ankle included



Figure 1. MRI sequences in a 47-year-old woman with pain for seven days. (A) coronal fs T2-w sequence of the ankle shows an enlarged lateral plantar vein and perivascular edema (open arrow). (B) coronal postcontrast fs T1-w sequence of the ankle shows a filling defect lateral plantar vein (open arrow) with enhancement of the perivascular planes (arrow). (C) ankle ultrasound showing echogenic material inside the bifurcated lateral plantar vein with no flow on the Doppler study, without the compression maneuver on the left, related to lateral plantar vein thrombosis. (D) coronal fs T2-w sequence of the midfoot shows plantar arch enlargement and perivascular edema (arrow). (E) coronal T1-w sequence of the midfoot shows material with a high signal within the plantar arch (arrow) associated with obliteration of the perivascular planes. (F) coronal postcontrast fs T1-w sequence of the midfoot shows a filling defect by contrast of the plantar arch with material with an intermediate signal inside that corresponds to the thrombus (arrow) associated with obliteration and enhancement of the perivascular planes.

T1- and fat-saturated (fs) T2-weighted (-w) fast spin echo (FSE) sequences in the axial and sagittal planes as well as a coronal fs T2-w FSE sequence. The foot protocol included T1- and fs T2-w FSE sequences in the coronal and axial planes and sagittal fs T2-w sequences. In addition to this basic protocol, intravenous paramagnetic contrast (gadoteric acid - Dotarem[®], Guerbet) was used in all cases. The MRI sequence parameters are described in Tables 1 and 2.

Image analysis

All MRI imaging studies were analyzed separately by two radiologists specialized in musculoskeletal radiology with ten and seven years of experience (MDM and FOZ, respectively).

Post-contrast MRI

In the post-contrast MRI sequences, the intravascular filling defect was analyzed and defined as an intravascular filling defect with absent signal intensity surrounded by high-signal intensity from flowing blood (Figures 1-4).

Non-contrast MRI sequences, the diameter of the foot vessels was analyzed. Posterior tibial, fibular, anterior tibial, medial plantar, lateral plantar, plantar arch, and intermetatarsal veins (Figure 5) were measured in the largest transverse diameter of the vessels in fs T2-w sequences using the Philips Carestream VR 12 PACS system measurement tool. The diameter of the anterior and posterior tibial and fibular veins was measured in the axial sequence of the ankle in the

Table 1. MRI	sequence	parameters	of the ankle
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Ankle	FOV	Thickness (mm)	Spacing (mm)	Slices	RT (ms)	ET (ms)	Matrix pixels	NEX	Bandwidth
Sagittal T1	15	4	0.4	14	485	13	348 x 256	2	41.67
Sagittal T2 Fat sat	15	4	0.4	14	2604	70	256 x 224	4	41.67
Axial T1	16	4	0.4	22	600	14	351 x 192	1	31.25
Axial T2 Fat sat	15	4	0.4	14	3823	60	256 x 224	4	41.67
Coronal T2 Fat sat	16	4	0.8	20	3627	60	256 x 224	4	31.25
Coronal OBL Fat sat	16	3	1	24	2554	45	252 x 252	2	35.71

FOV: Field of view; RT: Repetition time; ET: Echo time; mm: millimeters; sat: saturation.

Table 2. MRI sequence parameters of foot

Foot	FOV	Slice thickness (mm)	Spacing (mm)	Slices	RT (ms)	ET (ms)	Matrix pixels	NEX	Bandwidth
Sagittal T2 Fat sat	15	3.5	0.5	20	3202	60	256 x 256	4	31.25
Coronal T2 Fat sat	11	3	0.5	24	4114	60	256 x 224	4	25
Coronal T1	11	3	0.5	12	418	9	288 x 224	2	31.25
Axial T2 Fat sat	12	3	0.3	12	2062	70	256 x 224	4	19.23
Axial T1	12	3	0.3	12	394	9	288 x 256	2	31.25

FOV: Field of view; RT: Repetition time; ET: Echo time; mm: millimeters; sat: saturation.

bimalleolar plane (where we visualize the two malleoli in the axial sequences). The diameter of the medial and lateral plantar veins was measured in the coronal plane of the ankle at the midpoint of the distance between the bimalleolar plane; the plantar arch was measured in the coronal plane of the midfoot in the plane of its longest longitudinal axis, and the intermetatarsal veins were measured in the coronal plane of the forefoot at the midpoint between the plantar arch and the transition of the intermetatarsal veins with the interdigital veins. All measurements of vascular diameter were made in millimeters (mm). The analyses were performed separately by two radiologists and inter- and intraobserver correlation was also evaluated.

Also, in the non-contrast MRI fs T2-w sequences, the presence or absence of perivascular edema (defined as a halo of high signal in the fs T2-w sequences around the vessel) was documented in all vascular segments of both groups (Figures 2-4).

Statistical analysis

Data analysis was performed using descriptive statistics (mean, standard deviation, median, minimum, maximum, frequency, and percentage) using R version 4.2.0 (copyright © 2022, The R Foundation for Statistical Computing). Case and control groups were compared using the chi-square test (categorical variables) or the Mann-Whitney test (numerical variables). The level of significance considered in the analyses was 5%. Interobserver agreement was assessed using the intraclass correlation coefficient (ICC).

Results Demographics and clinical findings

Of the 61 patients in the case group, 26 were women, and 35 were men. The mean age was 49.2 years (36-63), and the mean BMI was 26.3 (24.5-30). Thirty-one patients had thrombosis on the right side, and 30 had thrombosis on the left side.

In the control group, there were 131 women and 93 men, with a mean age of 47.4 years (36-57.2 years) and a mean BMI of 27.3 (24-29.8). No significant differences were found between the two groups regarding age, sex, and BMI (p > 0.05), as shown in Table 3. The right side was used for comparison in 107 of them, and the left side was used for comparison in 117.

All patients in the case group presented with a clinical history of acute foot and ankle pain. The mean duration of symptoms in patients with thrombosis until the Doppler ultrasound was performed was 8.5 days, with an interquartile range between 6 and 10 days and a median of seven days. The mean duration until the MRI was performed after the ultrasound was five days, with an interquartile range between 1-7 days.

Of the case group, 83.6% (52/61) reported a history of recent physical exercise or recent foot trauma; most patients reported pain as the main clinical symptom (86.9%, 52/61). An association with swelling was seen in 6.5% of cases. Table 4 shows the distribution of categorical variables between the case and control groups. Trauma and recent physical exercise in the foot demonstrated a statistically significant association



Figure 2. Lateral plantar vein thrombosis in a 65-year-old woman with plantar pain and tingling sensation for two weeks. Frequent Muay Thai classes three times per week. Hindfoot coronal (A) and axial (B) fs T2-w sequences show proximal lateral plantar enlargement and perivascular edema (arrow). Hindfoot coronal (C) and axial (D) fs T1-w postcontrast showing filling defect of the proximal lateral plantar vein, related to thrombosis (arrow).

with thrombosis (p < 0.001). Also, the relevant comorbidities, such as diabetes mellitus, arterial hypertension, metabolic syndrome, hypercholesterolemia, smoking, thrombophilia, neoplasia under treatment, and chronic kidney disease, were documented, but no statistical difference between the groups was found (p = 0.178).

MRI findings

Of the vessels affected by thrombosis, 39/61 (63.4%) were part of the lateral plantar veins, 21/61 (34.4%) of the intermetatarsal veins, 9/61 (14.8%) of the plantar arch and five or less were other vessels affected (fibular, interdigital, medial plantar, and posterior tibial). In 14 patients, one or



Figure 3. MRI sequences in a 51-year-old woman with pain in the plantar surface of the forefoot for ten days after a marathon. (A) Coronal fs T2-w of the forefoot shows enlargement and perivascular edema in the second intermetatarsal vein in the plane of the metatarsal neck (arrow), with obliteration of the adjacent fat planes. (B) Coronal T1-w sequence of the forefoot shows obliteration of the fat planes of the second interspace in the plane of the metatarsal neck (arrow). (C) Coronal postcontrast fs T1-w shows a filling defect in the contrast study (arrow) of the intermetatarsal vein of the second associated space and edema with contrast enhancement of the perivascular planes.

more vessels were affected. In 18 cases (22.5%), proximal extension of the thrombus to the calf veins was shown.

On MRI, the veins with thrombosis observed on Doppler ultrasound presented with intravascular filling defect in all cases (100%) in the post-contrast enhanced sequences.

The perivascular edema was observed in all cases (100%) on non-enhanced sequences. Also, compared to the control group, all vessels had a greater diameter in the case group (Table 5); differences were most pronounced in the lateral

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Figure 4. MRI sequences in a 47-year-old woman who practices regular walks with plantar pain which is worse while walking. Coronal (A) and axial (B) fs T2-w sequences show enlargement and perivascular edema in the second intermetatarsal vein (arrow). Coronal (C) and sagittal (D) fs T1-w postcontrast also show perivascular edema and a filling defect in the intermetatarsal veins. Note that edema follows the neurovascular plexus and is not in the plantar fat, differing from bursitis.

plantar veins, and veins were overall largest in the plantar arch location.

Perivascular edema was not identified in any case in the control group. Also, the intravascular filling defect was not identified in the post-contrast MRI sequences in the control group.

Agreement between readers

Table 6 and Figure 6 shows the agreement of vascular diameter measurements between the two readers. The highest agreements were observed for the measurements of the calibers of the plantar arch (ARCH) and posterior tibial (PT), second space intermetatarsal (MET2), and medial plantar (MP) veins. However, all vessels had an ICC greater than 0.7, indicating good agreement between the two readers. The graphs with the lowest agreement between the readers, corresponded to the diameters of the intermetatarsal veins of the first and fourth interspaces (MET1 and MET4), probably because the vessels are smaller and more curved.

Discussion

Our study showed that the lateral plantar vein was the most frequent location of PVT. An intravascular filling defect was seen in all cases using post-contrast MRI sequences. Compared to the control group, all vessels had a greater diameter in the case group, and perivascular edema was observed in all cases. Our results also suggest that a clinical history of trauma or mechanical overload (physical activities) in the foot and ankle may be one of the causal factors of this pathology.

The mechanism related to PTE and PVT is still not fully understood. It is speculated that in patients with PVT who are not anti-coagulated, there may be thrombus fragmentation due to repeated compression of the foot determined by the muscular and venous gait action. Because of this significant biomechanical compression, the pressure transmitted to the plantar venous plexus can override the blood column of the calf's deep venous system, causing thrombi in this region to spread to other parts of the body^(10,11). Thus, the correct diagnosis of deep vein thrombosis is important, and improving the diagnosis of PVT represents a crucial factor in preventing PTE.



Figure 5. Measurements of vascular diameters (A to D) in a 38-year-old man presenting with heel pain for approximately three months, with progressive worsening. There were no signs of thrombosis in this study (control group). (A) Axial fs T2-w sequence in the bimalleolar plane. Caliber of the posterior tibial vein (arrow): 2.46 mm; fibular vein (arrowhead): 1.73 mm; anterior tibial vein (curved arrow): 2.48 mm. (B) Coronal fs T2-w sequence in the plane of the longest longitudinal axis of the plantar arch (arrow): 2.43 mm. (C) Coronal fs T2-w sequence at the midpoint between the bimalleolar plane and the plantar arch. Medial plantar vein caliber (arrow): 1.29 mm; lateral plantar vein (arrowhead): 2.71 mm. (D) Coronal fs T2-w sequence of the forefoot at the midpoint between the plantar arch and the transition of the intermetatarsal veins with the beginning of the interdigital veins. First space intermetatarsal vein caliber (arrow): 2.51 mm; second space intermetatarsal vein (arrowhead): 1.44 mm; third space intermetatarsal vein (curved arrow): 3.05 mm and fourth space intermetatarsal vein (larger arrow): 1.91 mm.

Table 3. Comp	barison between	groups in	numerical	variables
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Variable	Cases	s (N = 61)	Contro	Controls (N = 224)			
	Mean (SD)	Median [Q1;Q3]	Mean (SD)	Median [Q1;Q3]			
Age	49.2 (15.2)	47.0 [36.8;63.0]	47.4 (14.9)	47.0 [36.0;57.2]	0.461		
BMI	27.5 (5.03)	26.3 [24.5;30.0]	27.3 (5.03)	26.6 [24.0;29.8]	0.890		
Time (days)	8.5 (3.9)	7.0 [6.00;10.0]					

SD: Standard deviation; Q1: Lower quartile; Q3: Upper quartile; BMI: Body mass index; Time: time in days from the onset of symptoms to the time of performing the MRI.

Inflammatory cytokines play a crucial role in the development, differentiation, and reabsorption of the thrombus, and they also act in the coordination of immune system cells⁽¹²⁻¹⁴⁾. The interaction between circulating leukocytes and injured endothelium is the major event that promotes thrombus formation, and they act together with proinflammatory cytokines such as interferon-gamma, interleukin-6, and tumor necrosis factor-alpha, increasing capillary permeability and favoring extravasation of fluid into the extravascular

Table	4.	Com	parison	between	aroups	categorical	variabl	es
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Variable	Case (N = 61)	Control (N = 224)	p-value
Side			0.486
Right	31 (50.8%)	107 (47.8%)	
Left	30 (49.2%)	117 (52.2%)	
Sex			0.079
Female	26 (42.6%)	131 (58.5%)	
Male	35 (57.4%)	93 (41.5%)	
Comorbidities			0.178
No	22 (36.0%)	78 (47.9%)	
Yes	39 (64.0%)	85 (52.1%)	
Complaint			0.821
Pain	53 (86.9%)	175 (88.8%)	#Considering
Pain and swelling	4 (6.5%)	20 (10.2%)	only the categories pain
Pain and paresthesia	0 (0%)	1 (0.5%)	and pain and
Nodule	2 (3.3%)	0 (0%)	swelling
Paresthesia	2 (3.3%)	1 (0.5%)	
Trauma/ Recent exercise			<0.001
No	10 (16.4%)	77 (52.4%)	
Yes	51 (83.6%)	70 (47.6%)	

environment, which on MRI can be translated as perivascular edema⁽¹²⁾. On the other hand, leukocytes play important roles in the thrombus reabsorption process. Neutrophils and monocytes invade the thrombus to modulate collagen and fibrin degradation through the secretion of matrix metalloproteinases (MMPs) and plasminogen activators.

Our results showed that vein enlargement and perivascular edema were present in all plantar thrombosis cases and were not identified in the control group. Our results highlight that this finding could be used as an imaging biomarker of vascular abnormalities, and whenever it is present, the possibility of vascular thrombosis must be considered. This becomes even more important as, currently, most of the foot and ankle MRI studies are performed without the paramagnetic contrast agents, and these findings were identified in nonenhanced sequences. In cases of suspicion, the diagnosis can be confirmed with post-contrast MRI imaging, as all studied cases with thrombosis on Doppler ultrasound presented with an intravascular filling defect on MRI. Note that this is the first study to describe the non-contrast MR imaging aspects of PVT, correlating with Doppler ultrasound.

Most patients reported a history of recent physical exercise or foot trauma, suggesting that the trauma/mechanical overload could be related to endothelial wall injury activating the blood clotting cascade. This injury mechanism may also explain why the most affected was the lateral plantar vein, which has a more superficial path along the lateral segment of the plantar arch and usually bears more load in the biomechanics of gait⁽¹⁰⁾. There was no case in which the medial plantar vein was affected alone, probably due to its deeper intermuscular course in the medial segment of the plantar arch^(3,15).

The caliber of vessels in cases of thrombosis was greater in all segments analyzed in relation to the examinations of controls. This difference can be explained by the presence of the thrombus inside the vessels impairing venous return,

Table 5.	Vascular	diameter	in millimeters	comparison	hetween	arouns in n	umerical	variables
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Variable	Cases	(N = 61)	Control	n voluo	
Diameter	Mean in mm (SD)	Median in mm [Q1;Q3]	Mean in mm (SD)	Median in mm [Q1;Q3]	h-vaine
PT	2.78 (0.82)	2.60 [2.20;3.00]	2.57 (0.52)	2.50 [2.20;2.90]	0.332
FIB	1.94 (0.48)	1.85 [1.60;2.20]	1.75 (0.49)	1.70 [1.40;2.00]	0.013
AT	1.92 (0.41)	1.90 [1.60;2.10]	1.75 (0.39)	1.70 [1.50;2.00]	0.007
LP	3.78 (0.79)	3.80 [3.23;4.20]	3.19 (0.90)	3.10 [2.40;3.80]	<0.001
MP	2.45 (0.80)	2.30 [1.95;2.85]	2.10 (0.62)	2.00 [1.70;2.40]	0.001
ARCH	2.56 (0.93)	2.50 [2.00;3.10]	2.14 (0.72)	2.10 [1.60;2.50]	0.001
MET1	1.51 (0.46)	1.40 [1.15;1.90]	1.33 (0.41)	1.30 [1.00;1.50]	0.005
MET2	1.69 (0.70)	1.50 [1.25;2.00]	1.25 (0.37)	1.20 [1.00;1.50]	<0.001
MET3	1.52 (0.50)	1.40 [1.15;1.85]	1.17 (0.35)	1.10 [0.90;1.40]	<0.001
MET4	1.10 (0.35)	1.00 [0.90;1.20]	0.99 (0.27)	0.90 [0.80;1.10]	0.009

PT: Posterior tibial vein; FIB: Fibular vein; AT: Anterior tibial vein; LP: Lateral plantar vein; MP: medial plantar vein; ARCH: Plantar arch; MET 1: First space intermetatarsal vein; MET 2: Second space intermetatarsal vein; MET 3: Third space intermetatarsal vein; MET 4: Fourth space intermetatarsal vein; SD: Standard deviation.

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Vein	Reader	N	Mean	SD	Min	Median	Max	ICC	95% CI (ICC)
ARCH	1	261	2.2	0.8	0.8	2.1	6.0	0.948	0.934; 0.959
	2	261	2.2	0.8	0.2	2.0	5.5		
FIB	1	253	1.7	0.5	0.3	1.7	3.9	0.815	0.769; 0.852
	2	253	1.8	0.5	1.0	1.8	4.0		
MET1	1	263	1.3	0.4	0.3	1.2	2.9	0.707	0.642; 0.763
	2	263	1.4	0.5	0.7	1.3	5.0		
MET2	1	263	1.3	0.5	0.2	1.2	3.9	0.911	0.888; 0.929
	2	263	1.3	0.5	0.6	1.2	3.5		
MET3	1	263	1.2	0.5	0.2	1.1	3.0	0.849	0.812; 0.88
	2	263	1.2	0.4	0.5	1.1	2.8		
MET4	1	261	1.0	0.4	0.5	0.9	3.1	0.777	0.725; 0.821
	2	261	1.0	0.3	0.7	0.9	2.5		
LP	1	262	3.3	1.0	1.2	3.2	6.8	0.895	0.868; 0.917
	2	262	3.3	0.9	1.4	3.2	5.5		
MP	1	259	2.2	0.7	0.9	2.0	5.6	0.906	0.882; 0.926
	2	259	2.2	0.7	1.0	2.0	5.2		
AT	1	253	1.8	0.4	0.9	1.7	3.4	0.845	0.805; 0.877
	2	253	1.8	0.4	0.9	1.8	3.4		
PT	1	253	2.6	0.6	1.1	2.6	6.1	0.930	0.911; 0.945
	2	253	2.6	0.6	1.0	2.5	5.5		

Table 6. Agreement between readers: Measurements of position and dispersion per reader and intraclass correlation coefficient to assess agreement between readers

PT: Posterior tibial vein; FIB: Fibular vein; AT: Anterior tibial vein; LP: Lateral plantar vein; MP: medial plantar vein; ARCH: Plantar arch; MET 1: First space intermetatarsal vein; MET 2: Second space intermetatarsal vein; MET 3: Third space intermetatarsal vein; MET 4: Fourth space intermetatarsal vein; SD: Standard deviation; ICC: Interclass correlation coefficient; CI: Confidence interval.



Figure 6. Scatter plots of the measurements of the two readers.

PT: Posterior tibial; FIB: Fibular; LP: Lateral plantar; ARCH: Plantar arch; MET 2: Second intermetatarsal vein.

causing vascular congestion, and increasing intravascular hydrostatic pressure and vascular caliber; this also contributes to the extravasation of fluid into the extravascular environment even in more prolonged cases of thrombosis, which also present perivascular edema⁽¹⁶⁾.

In pathologies such as vasculitis, there may also be perivascular edema; however, these conditions tend to affect more vascular segments, often with bilateral involvement and a more systemic and prolonged clinical scenario⁽¹⁷⁾.

Although the exact cause of PVT is unknown, it is usually associated with coagulopathies, paraneoplastic syndromes, trauma, contraceptive medication, and postoperative conditions. Recent studies report an idiopathic cause in up to 50% of cases^(13,15). It is worth mentioning that, in the absence of a defined causal factor, it is recommended to continue the diagnostic investigation in search of thrombophilia and paraneoplastic syndromes, and any respiratory symptoms that may be related to PTE should be considered^(2,11,15,18).

The mean duration of symptoms in patients with thrombosis until the MRI was performed was 8.5 days, consistent with the literature^(19,20). The clinical scenario of PVT is nonspecific, usually including pain, local edema. and tenderness on palpation, and it is often confused with other more frequent causes of pain on the plantar surface of the foot, such as plantar fasciitis, musculotendinous injuries, stress fractures, plantar plate, bursitis, and neuromas. Thus, the diagnosis of PVT is usually first made through imaging exams, such as MRI or ultrasonography, and our data support that the presence of perivascular edema on foot MRI studies should raise this diagnosis.

Ultrasonography is an accessible and inexpensive method that allows the evaluation of the entire compromised vascular segment and the performance of dynamic maneuvers such as compression of the affected vascular segment. However, there are limitations to its use, which include challenges with the clinical diagnosis, the fact that the evaluation of the plantar veins is not part of the routine protocol for investigating foot pain syndrome, the difficult evaluation of vascular segments with smaller caliber, and impaired evaluation in obese patients and patients with very thick skin.

Due to its increasingly frequent use, MRI is often the first method for detecting PVT. MRI can assess all foot structures simultaneously and three-dimensionally, with good differentiation between structures, and demonstrating vascular filling defects using contrast-enhanced imaging. Furthermore, perivascular edema and an increase in vascular caliber were identified in most cases of thrombosis already in MRI sequences without contrast.

Our study had limitations, including the retrospective study design. However, we could review 61 patients with PVT, and we compared the case cohort with a control group, one of the largest cohorts to investigate this abnormality and correlate imaging and clinical data. Another limitation is the lack of longitudinal follow-up, and cases were not reevaluated after treatment and resolution of symptoms.

Conclusion

Plantar venous thrombosis is an underdiagnosed cause of acute painful foot syndrome, and due to nonspecific clinical symptoms, the diagnosis is challenging. Our study demonstrated that the most frequent location of the PVT was the lateral plantar vein. Also, it demonstrated that postcontrast MRI of PVT revealed intravascular filling defects in all cases and highlighted that vein enlargement and perivascular edema were identified in non-enhanced MRI sequences in most cases.

Additionally, it was demonstrated that one of the contributing causes to this pathology is the clinical history of trauma or mechanical overload in the foot and ankle.

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References

- Kubincova M, Vanhoenacker F. Plantar Vein Thrombosis Mimicking Tendinopathy. J Belg Soc Radiol. 2022;106(1):7.
- 2. Karam L, Tabet G, Nakad J, Gerard JL. Spontaneous plantar vein thrombosis: state of the art. Phlebology. 2013;28(8):432-7.
- Uhl JF, Gillot C. Anatomy of the foot venous pump: physiology and influence on chronic venous disease. Phlebology. 2012;27(5):219-30.
- Gray H. Anatomy of the human body. 20th edition. Philadelphia: Lea and Febiger; 1918. pp. 1-1364.
- Vansevenant M, Vanhoenacker FM. Plantar Vein Thrombosis: An Unusual Cause of Plantar Pain. J Belg Soc Radiol. 2015;99(2): 98-101.
- White RH. The epidemiology of venous thromboembolism. Circulation. 2003;107(23 Suppl 1):I4-8.
- Spritzer CE, Norconk JJ Jr, Sostman HD, Coleman RE. Detection of deep venous thrombosis by magnetic resonance imaging. Chest. 1993;104(1):54-60.

- Fraser DG, Moody AR, Morgan PS, Martel AL, Davidson I. Diagnosis of lower-limb deep venous thrombosis: a prospective blinded study of magnetic resonance direct thrombus imaging. Ann Intern Med. 2002;136(2):89-98.
- Needleman L, Cronan JJ, Lilly MP, Merli GJ, Adhikari S, Hertzberg BS, et al. Ultrasound for lower extremity deep venous thrombosis. Circulation. 2018;137(14):1505-15.
- Miranda FC, Carneiro RD, Longo CH, Fernandes TD, Rosemberg LA, de Gusmão Funari MB. Plantar thrombophlebitis: magnetic resonance imaging findings. Rev Bras Ortop. 2015;47(6):765-9.
- 11. Barros MV, Labropoulos N. Plantar vein thrombosis--evaluation by ultrasound and clinical outcome. Angiology. 2010;61(1):82-5.
- Najem MY, Couturaud F, Lemarié CA. Cytokine and chemokine regulation of venous thromboembolism. J Thromb Haemost. 2020;18(5):1009-19.
- ISTH Steering Committee for World Thrombosis Day. Thrombosis: a major contributor to the global disease burden. J Thromb Haemost. 2014;12(10):1580-90.

- 14. Mackman N. New insights into the mechanisms of venous thrombosis. J Clin Invest. 2012;122(9):3368.
- Swellengrebel HJC, Backus T, Zijta FM, van der Zwaal P. Plantar vein thrombosis provoked by mechanical strain to the foot: a rare cause of plantar heel pain. BMJ Case Rep. 2019;12(11):e230054.
- Peterson LH. Physical factors which influence vascular caliber and blood flow. Circ Res. 1966;18:I-3-13
- Shavit E, Alavi A, Sibbald RG. Vasculitis-What Do We Have to Know? A Review of Literature. Int J Low Extrem Wounds. 2018;17(4):218-226.
- Barros M, Nascimento I, Barros T, Labropoulos N. Plantar vein thrombosis and pulmonary embolism. Phlebology. 2015;30(1):66-9.
- Rastel D. Four new cases of isolated foot vein thrombosis: Is the first metatarsal interspace perforator responsible? J Med Vasc. 2021;46(3):114-122.
- Czihal M, Röling J, Rademacher A, Schröttle A, Kuhlencordt P, Hoffmann U. Clinical characteristics and course of plantar vein thrombosis: a series of 22 cases. Phlebology. 2015;30(10):714-8.