Sports Medicine and Health Science 6 (2024) 89–93

Contents lists available at ScienceDirect

Sports Medicine and Health Science

journal homepage: www.keaipublishing.com/smhs

Case Report

Exertional calf pain at kilometer five – Finding the cause

Samuel A. Renier [a], Angela M. Voight [b], Emilee J. Trost [c], William O. Roberts [d,*]

[a] St. John's Hospital Family Medicine Residency, Department of Family Medicine and Community Health, University of Minnesota Medical School, Minneapolis, MN, USA
[b] Summit Orthopedics, Woodbury Clinic, 2090 Woodwinds Dr., Woodbury, MN, 55125, USA
[c] Minnesota Distance Elite, 4007Forest Rd, Minneapolis, MN, 55416, USA
[d] Department of Family Medicine and Community Health, University of Minnesota Medical School, 516 Delaware St. SE, 6-240 Phillips-Wangensteen Building, Minneapolis, MN, 55455, USA

ARTICLE INFO

Keywords:
Claudication
Lower leg pain
Vascular imaging
PAES
FPAES
Popliteal entrapment

ABSTRACT

A 23-year-old professional distance runner with several years of exertional calf pain was diagnosed with a unique mixed type III and functional popliteal artery entrapment syndrome (PAES). Surgical reduction of the obstructing tissue allowed her to return to professional running. This case highlights the importance of including PAES in the differential for chronic intermittent lower extremity claudication and outlines the work-up required to diagnose this vascular obstruction in younger athletes.

1. Introduction

Popliteal artery entrapment syndrome (PAES) is a rare condition that sports medicine physicians must recognize. The symptoms are caused by compression of the popliteal artery, popliteal vein, and/or tibial nerve by structures in the popliteal fossa. Intermittent claudication in the calves during intense exercise is the most common presenting symptom.[1] The claudication pain often starts a few minutes into activity and is relieved by rest. The vein is compressed at lower pressures than the artery, which may account for some variations in the presenting symptom complex.

PAES has six variants: types I through V - anatomical variants that each uniquely compress the neurovascular bundle, either due to an anomalous artery course or from compression by muscle, accessory ligament, or fibrous band, and type VI - normal anatomy with muscle hypertrophy compressing the neurovascular bundle.[1–3] Fig. 1 illustrates types 1–6.

Type VI entrapment is termed "functional" (FPAES) as there is normal anatomy, but the surrounding muscle tissue is hypertrophied.[4] Turnispeed describes FPAES as compression of the neurovascular structures at the soleal sling, causing soleus and gastrocnemius cramping and foot paraesthesias."[5] Others attribute the compression to hypertrophy of the medial gastrocnemius muscle.[4] FPAES occurs when the muscles passing through the popliteal fossa hypertrophy in response to exercise loading, compressing the neurovascular bundle. The condition has significant symptom overlap with chronic exertional compartment syndrome (CECS).[5] PAES, and especially FPAES with no structural abnormality, takes time and expertise to diagnose. If the office-based work-up is equivocal, the diagnostic imaging required to confirm the diagnosis is costly and invasive. A meta-analysis on 30 studies on PAES showed that the average patient with PAES undergoes three imaging evaluations to determine the cause of leg pain.[1] The condition requires either surgical correction of the obstructing structures or reduced training load to decrease the size of the obstructing the muscle mass.

2. Case

Consent for publication was obtained from the study participant, who is one of the authors. Institutional review and approval are waivered.

A 23-year-old female professional distance runner presented for evaluation with a 5-year history of worsening bilateral throbbing pain in her calves and paresthesia in both her feet with onset 3–5 km into races and intense workouts. Her symptoms began while in college and persisted into her post-collegiate professional career, often preventing her from competing in races and completing training sessions. She had several lower extremity overuse injuries diagnosed during this time including plantar fasciitis, peroneal tendinopathy, posterior tibialis tendinopathy, and medial tibial stress syndrome. Her symptoms were worsened by running, bicycling, and prolonged standing on hard surfaces; rapidly relieved by rest; and improved by massage, stretching,

* Corresponding author. Department of Family Medicine and Community Health, University of Minnesota Medical School, 516 Delaware St. SE, 6-240 Phillips-Wangensteen Building, Minneapolis, MN, 55455, USA
E-mail address: rober037@umn.edu (W.O. Roberts).

https://doi.org/10.1016/j.smhs.2023.10.001
Received 20 March 2023; Received in revised form 26 September 2023; Accepted 7 October 2023
Available online 11 November 2023

Abbreviations

PAES	Popliteal artery entrapment syndrome
FPAES	Functional popliteal artery entrapment syndrome
CECS	Chronic exertional compartment syndrome
MRI	Magnetic resonance imaging
ABI	Ankle-brachial index
km/h	Kilometers per hour
cm/s	Centimeters per second
PSV	Peak systolic velocity
MRA	Magnetic resonance angiography
DSA	Digital subtraction arteriography
CTA	Computed tomography angiography

taping, compression sleeves, dry needling, and extracorporeal shockwave therapy. Her menstrual cycles were irregular, and she had 2-3 per year. She estimates her daily caloric intake was 1 800–2 000 kCal during hard training.

On physical exam, she had a normal gait and muscle tone. Her active range of motion and strength in the hip, knee, and ankle joints were normal, with normal inversion and eversion in both ankles. Her popliteal, dorsalis pedis, and posterior tibial pulses were normal, with no decrease in pulses or provocation of symptoms with forced passive plantar flexion for 30 seconds (s). She had normal sensation in all lower extremity dermatomes. The retrocalcaneal bursa, the medial calcaneus, and medial mid-calf were tender on the left side. The tibia and fibula were non-tender and the Achilles, peroneal, and anterior/posterior tibialis tendons were not tender or swollen. There was no ecchymosis, erythema, or rash on either lower leg.

Right anterior compartment pressure testing completed in November 2017 following 30 minutes (min) of treadmill running to the point of intolerable pain was normal (8 mmHg). She was referred to a vascular surgeon about one year later for work up of possible PAES. Magnetic resonance imaging (MRI) showed left-sided posterior tibialis tendinosis and retrocalcaneal bursitis. Her ankle-brachial index (ABI) doppler ultrasound was normal both at rest and with exercise (measured 1 min after cessation of a 15 min treadmill run at 5.76 km/h, which reproduced bilateral medial calf pain with a 4 of 10 rating). Provocative testing of the lower extremity using arterial ultrasound showed compression of the popliteal artery below the left knee with the foot in forced passive plantar flexion and below the right knee with the foot in forced passive plantar flexion and dorsiflexion (Fig. 2). This was associated with an increase in peak systolic velocity (PSV), from 166 cm/s at rest to 309 cm/s with plantarflexion and 442 cm/s with dorsiflexion. Venous ultrasound showed nearly complete compression of the left popliteal vein below the knee with the foot in plantar flexion, causing a decrease in venous diameter from 0.34 cm at rest to 0.197 cm with plantar flexion (Fig. 3). There was also nearly complete compression of the right popliteal vein at the knee and below the knee with the foot in both plantar flexion and dorsiflexion.

Based on these ultrasound findings, her vascular surgeon diagnosed PAES. She had surgery to mobilize the popliteal neurovascular structures. Intraoperatively, she had a type III entrapment in her left leg with an anomalous tendinous/vascular band originating from the proximal medial gastrocnemius muscle and attaching to the soleus (Fig. 4), which was surgically divided. This may have been compressing the popliteal vein in addition to the artery (Fig. 3), implying an additional type V entrapment, though this could also simply have been artifact. She was also noted to have hypertrophied soleus muscles bilaterally, characterized by apparent impingement of the neurovascular bundle by the soleal sling (Fig. 4), which was relieved by vertically dividing both soleus muscles. Both plantaris muscles were also excised to create more space in the popliteal fossa (Fig. 5).

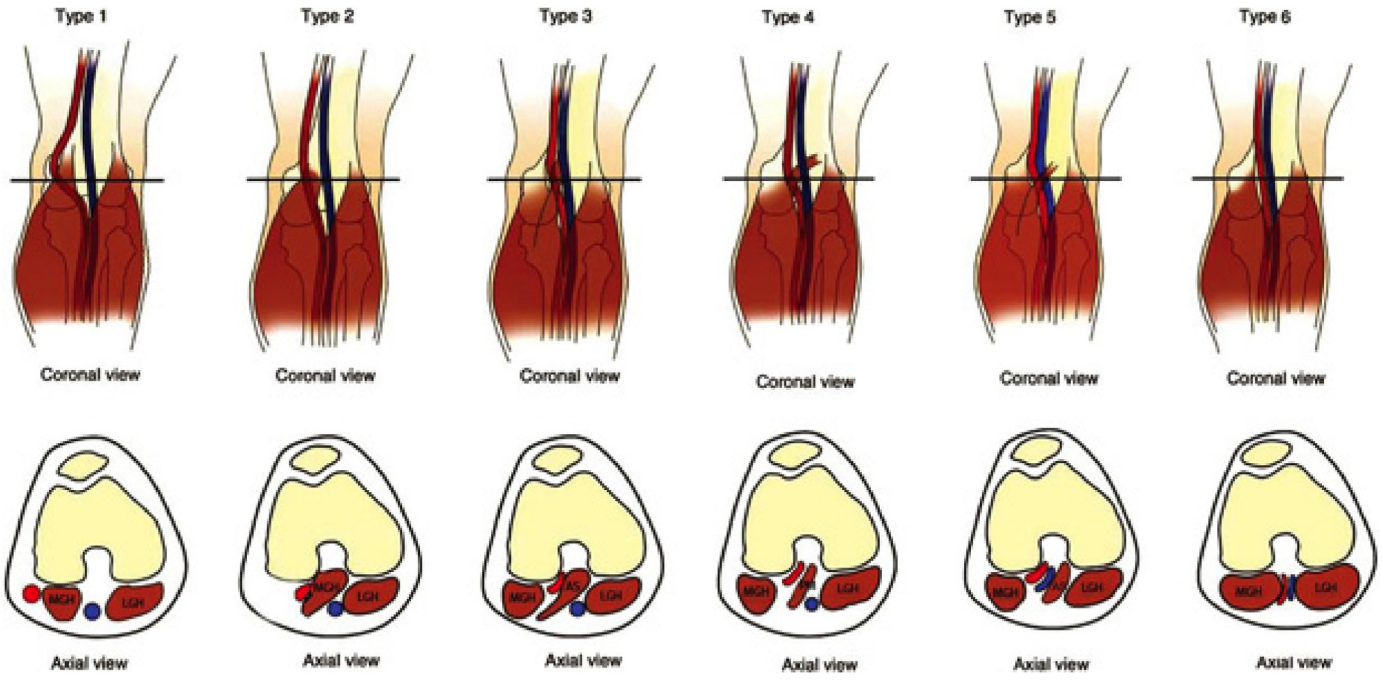

Fig. 1. PAES types 1–6. Type I, where the popliteal artery runs medial to the medial head of the gastrocnemius; type II, where the medial head of the gastrocnemius attaches lateral to the artery and medial to the popliteal vein; type III, where an accessory fibrous band arising from the medial head of the gastrocnemius compresses the artery; type IV, where the artery is located deeply and is compressed by the popliteus muscle or by a fibrous band; type V, in which the popliteal vein is also entrapped in addition to artery entrapment due to any of types I through IV anatomy; and type VI, where the artery and surrounding tissues are positioned normally, but the artery is entrapped by a hypertrophied gastrocnemius or soleus muscle.[1–3]
From Popliteal artery entrapment syndrome: morphological classification utilizing MR imaging, Springer Link (https://link.springer.com/article/10.1007/s00256-006-0158-5).

She returned to running four weeks post-surgery and by six months she was training with her professional team without calf pain. Three years after her surgical procedure she reported "I'm in a nice routine of running, cross-training, lifting, and learning Pilates. This is the part where they tell you to 'enjoy the process' and while I agree that training hard day in and day out is great, the reward of racing is calling my name. In a few days it will be the three-year anniversary of my popliteal artery entrapment surgery. Looking back, it is amazing how far I've come and how wonderful it is to run without constant calf pain." She has been setting personal bests in races during the current (2023) season.

3. Discussion

Intermittent claudication is relatively common in older adults, especially adults with risk factors for peripheral vascular disease. However, exercise-related claudication is not common in adolescents or young adults and should not be ignored. This young athlete had symptoms with heavy exertion suggesting PAES, but the condition was not suspected for five years. Athletes moving from high school to college to professional training programs experience a greater training load at each level and increasing muscle hypertrophy, which may be part of the reason it took time to find the cause in this case with a functional component.

In a retrospective study conducted by Turnispeed, 100 % of the patients eventually diagnosed with PAES presented with symptoms of claudication in the calves and about 40 % of patients also had paresthesia in one or both feet.[5] The differential diagnosis for exercise-related calf pain also includes CECS, and many PAES patients have a compartment release surgery performed at some point in their clinical course.[5] The symptom overlap with other causes of lower extremity pain can be confusing and steer providers down the wrong clinical pathway. Other causes of lower leg pain can occur concurrently, also potentially slowing the diagnosis of PAES.

PAES is difficult to diagnose, and having a framework to guide the investigation is useful.

The work-up starts with a thorough history to elicit symptoms consistent with PAES like a predictable onset of leg pain with prolonged or intense exercise and rapid resolution of symptoms with cessation of exercise. It is important to differentiate PAES symptoms from those more specific to CECS like the "second-day" phenomenon, where pain occurs earlier in the exercise bout on consecutive days of exercise due to residually elevated compartment pressures from the first day of exercise; foot drop; or inability to run through the pain. The physical exam begins with putting the athlete in maximal passive ankle dorsiflexion with the knee in full extension and maximal active plantarflexion against resistance for at least 90 s. If these provocative maneuvers are positive, ABIs should be performed, both before and promptly after an exercise load that reproduces the symptom. This athlete developed symptoms at a relatively low workload on the treadmill for a professional runner, the same pain that prevented her from competing and training.

If the index of suspicion for PAES remains high based on findings from the history and office-based examination, imaging to confirm the diagnosis should be pursued. Duplex doppler ultrasound is often the first investigation performed, but it has a high false positive rate with many asymptomatic individuals having popliteal artery obstruction with provocative maneuvers.[4] Sinha et al. suggest all patients with suspected PAES be evaluated with ABIs before and after exercise, noninvasive compartment pressure testing, magnetic resonance angiography (MRA), and catheter-directed digital subtraction arteriography (DSA).[1] DSA is invasive and exposes patients to significant radiation, but has historically been considered the "gold standard" for diagnosing PAES and may be required to confirm the diagnosis.[1,4] alternative, some providers use computed tomography angiography (CTA), as it is less invasive than DSA,[4] and allows visualization of surrounding soft tissues that may be contributing to obstruction, which DSA does not.[6] MRA has a similar estimated sensitivity to CTA in diagnosing PAES, and provides even greater surrounding soft tissue resolution than CTA.[1,6] The limitations of MRA include the tendency to underestimate the degree of stenosis and the motion artifacts, which are common as the patient holds the affected

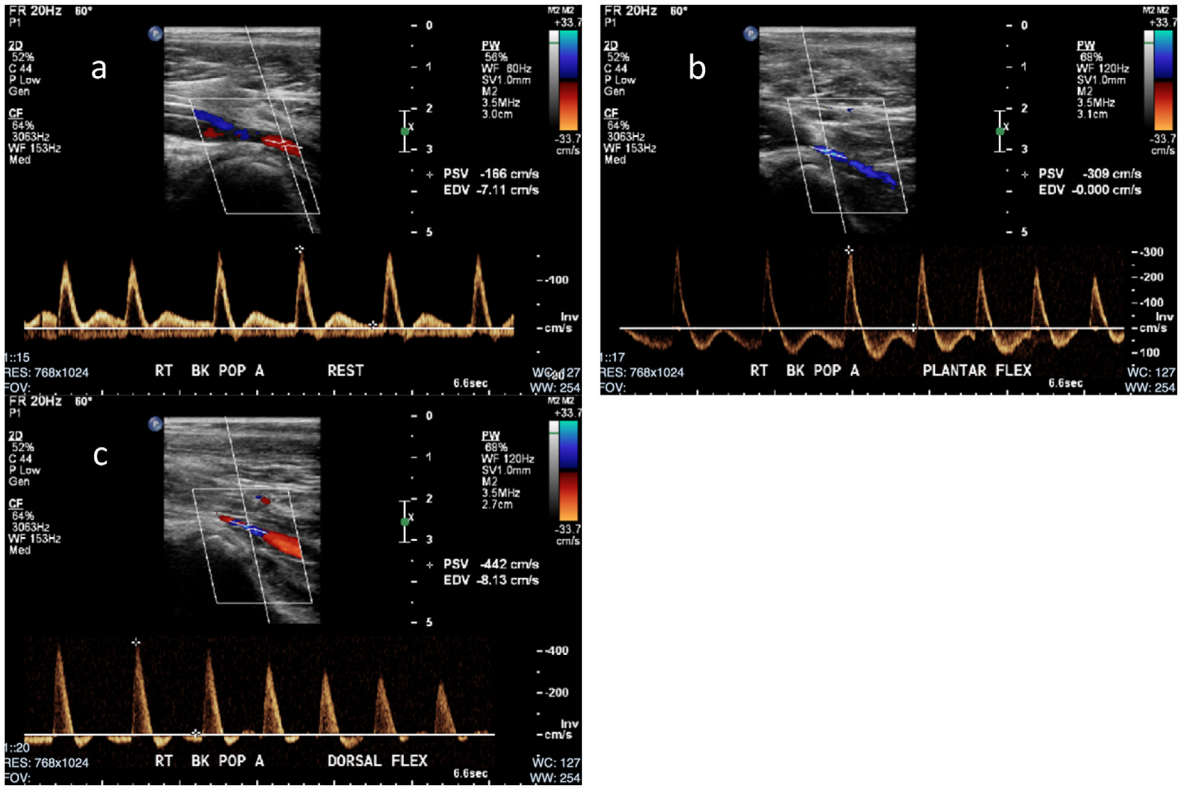

Fig. 2. Duplex Doppler Ultrasound images of the right below-knee popliteal artery at rest (image a) and with plantar (image b) and dorsiflexion (image c). Compression leading to elevated peak systolic velocity (PSV) is seen in plantar and dorsiflexion with associated disturbed flow.

limb in provocative positions for an extended time.[4,6] As shown in this case, standard imaging modalities that do not specifically evaluate the vasculature (like MRI) were not helpful in diagnosing this runner's PAES and have the potential to show clinically insignificant findings that may mislead providers.

ABIs and CT angiography, both with provocative maximal active plantar flexion and maximal passive dorsiflexion maneuvers, are the recommended imaging studies. If there are significant contraindications to CT imaging, MRA can be used. Given the high prevalence of co-morbid CECS, noninvasive compartment pressure testing is indicated in the evaluation of PAES. Ultrasound may be used as a screening modality and can support a diagnosis, but it should not be relied upon for confirmation.

Conservative management of this condition involves detraining the hypertrophied muscles to relieve the neurovascular compression. Botulinum toxin injection have also been used as both a diagnostic procedure and a therapeutic intervention, with symptom improvement in 66 % of patients at 10-months of follow-up.[7] These conservative measures should be reserved for FPAES, as surgical treatment is recommended for all patients with anatomical PAES to avoid serious long-term complications such as arterial intimal damage, thrombosis, distal embolization, post-stenotic dilatation, and aneurysm formation.[8] Definitive treatment is surgical decompression of the neurovascular bundle by removing and/or dividing surrounding structures like the soleus, gastrocnemius, and plantaris muscles. If arterial damage is present, vessel bypass or repair may be necessary.[8] The 2019 meta-analysis from Shahi et al. shows a success rate of surgical myotomy/myomectomy for correcting the blood flow restriction and resolving symptoms of 97 % at a mean follow-up of 25 months with 4 % of patients experiencing post-operative complications of seroma and/or hematoma and 2 % experiencing infection.[7]

Patients with suspected PAES requiring surgery may be best served at vascular centers with experience in the diagnostic protocol and treatment of this complex condition. Ideally, these centers are also collecting data to improve the evaluation and management of this rare pathology. The duration of symptoms has not been associated with irreversible arterial damage.[1] However, given that the pain is typically long-standing and prevents athletes from competing, early diagnosis is important.

4. Conclusion

PAES is a relatively rare condition that sports medicine providers will likely encounter over the course of a career. This case describes the clinical course of a female professional runner who was ultimately found to have bilateral PAES, with compression by an anomalous accessory slip off the medial gastrocnemius muscle on one side (type III), and by a functional hypertrophy of her soleus muscle bilaterally (type VI, or FPAES). The case highlights the importance of maintaining a strong suspicion of PAES in patients with exercise-related leg pain. Claudication with intense exertion is almost uniformly present in patients with PAES. This symptom should prompt investigation with provocative testing in the office, as outlined above. If this testing reproduces symptoms, ABIs should be performed both before and promptly after exercise – ensuring that the exercise intensity is sufficient to reproduce the patient's

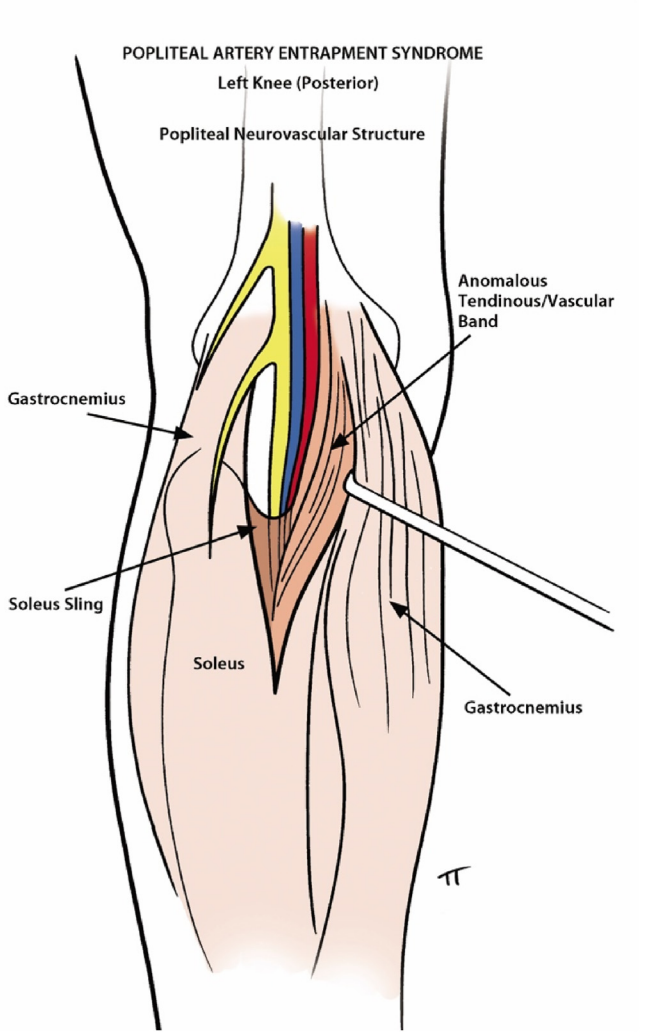

Fig. 4. Diagram of the patient's posterior left knee illustrating an anomalous tendinous/vascular band originating from the proximal medial gastrocnemius muscle and attaching to the soleus (type III entrapment), and the soleal sling (site of compression in functional entrapment). Illustration by Tim Trost.

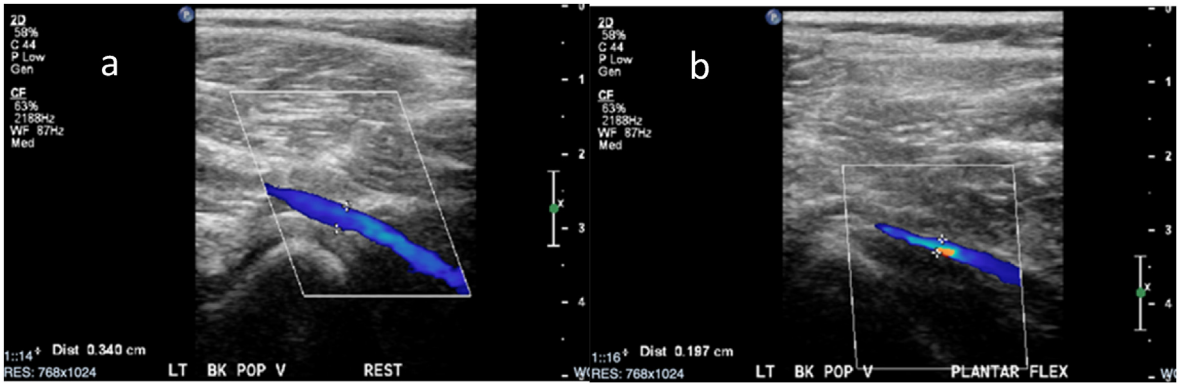

Fig. 3. Duplex Doppler Ultrasound images of the left below-knee popliteal vein at rest (image a) and with plantarflexion (image b), demonstrating decreased venous diameter and turbulent flow with plantarflexion.

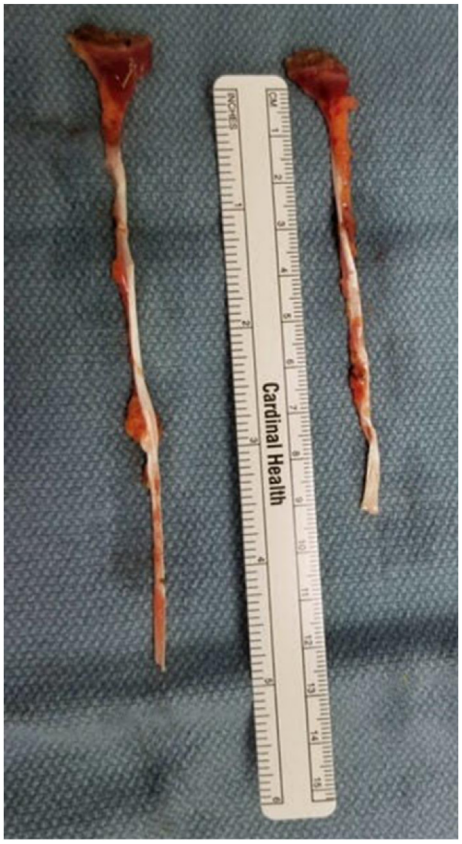

Fig. 5. Excised plantaris muscles to increase space for the neurovascular bundle in the popliteal fossa.

symptoms. If negative, one may consider noninvasive compartment pressure testing in screening for CECS, especially if the patient reports "second day" phenomenon, foot drop, or inability to run through the pain. If ABIs suggest PAES, CT angiography with provocative maneuvers should be used to confirm the diagnosis. Conservative management with reduced training volume or botulinum toxin injection of the hypertrophied muscle may be effective for symptom relief in FPAES. Ultimately, patients with PAES should be referred to a vascular surgeon with experience in the surgical management of this condition.

Submission statement

All authors have read and agree with manuscript content. The manuscript has not been published previously and is not under consideration for publishing elsewhere. It's publication in this journal is agreed to by all authors. If accepted, it will not be published elsewhere including electronically in the same form, in English or in any other language, without the written consent of the copyright-holder.

Ethical approval statement and consent for publication

Consent for publication was obtained from the study participant, who is one of the authors. Institutional review and approval are waivered.

Authors' contributions

SAR wrote the manuscript. WOR edited the manuscript. EJT provided specifics of her training and a chronologic history of her injuries from her training log (referenced in the "Case" section of the manuscript). All authors reviewed the final manuscript.

Conflict of interest

William O. Roberts is an Editorial Board Member for Sports Medicine and Health Science and was not involved in the editorial review or the decision to publish this article. The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

1. Sinha S, Houghton J, Holt PJ, Thompson MM, Loftus IM, Hinchliffe RJ. Popliteal entrapment syndrome. *J Vasc Surg*. 2012;55(1):252–262.e30. https://doi.org/10.1016/j.jvs.2011.08.050.
2. Hai Z, Guangrui S, Yuan Z, et al. CT angiography and MRI in patients with popliteal artery entrapment syndrome. *AJR Am J Roentgenol*. 2008;191(6):1760–1766. https://doi.org/10.2214/AJR.07.4012.
3. Gokkus K, Sagtas E, Bakalim T, Taskaya E, Aydin AT. Popliteal entrapment syndrome. A systematic review of the literature and case presentation. *Muscles, Ligaments, Tendons J*. 2014;4(2):141–148.
4. Lovelock T, Claydon M, Dean A. Functional popliteal artery entrapment syndrome: an approach to diagnosis and management. *Int J Sports Med*. 2021;42(13):1159–1166. https://doi.org/10.1055/a-1524-1703.
5. Turnipseed WD. Functional popliteal artery entrapment syndrome: a poorly understood and often missed diagnosis that is frequently mistreated. *J Vasc Surg*. 2009;49(5):1189–1195. https://doi.org/10.1016/j.jvs.2008.12.005.
6. Eliahou R, Sosna J, Bloom AI. Between a rock and a hard place: clinical and imaging features of vascular compression syndromes. *Radiographics*. 2012;32(1):E33–E49. https://doi.org/10.1148/rg.321115011.
7. Shahi N, Arosemena M, Kwon J, Abai B, Salvatore D, DiMuzio P. Functional popliteal artery entrapment syndrome: a review of diagnosis and management. *Ann Vasc Surg*. 2019;59:259–267. https://doi.org/10.1016/j.avsg.2018.12.105.
8. Noorani A, Walsh SR, Cooper DG, Varty K. Entrapment syndromes. *Eur J Vasc Endovasc Surg*. 2009;37(2):213–220. https://doi.org/10.1016/j.ejvs.2008.10.019.