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Surgical release of anterior tibial artery entrapment with associated popliteal artery entrapment

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ABSTRACT

Popliteal artery entrapment syndrome (PAES) is compression of the popliteal artery from embryologic myotendinous variation or calf muscle hypertrophy. PAES necessitates prompt diagnosis and complete release of the entrapped vasculature for symptom relief and to prevent chronic cumulative vascular damage. Our patient is a 27-year-old female referred for progressive bilateral claudication. Workup was consistent with bilateral PAES with preoperative imaging notable for an atypically proximal origin of the anterior tibial artery, which was also encased anterior to the popliteus muscle. Preoperative angiogram confirmed the diagnosis, and complete surgical release resolved symptoms by 4 months postoperatively. (J Vasc Surg Cases Innov Tech 2024;10:101395.)

Keywords: Popliteal artery entrapment syndrome; Popliteal artery entrapment; Artery entrapment; Popliteal; Entrapment; Popliteal artery; Popliteal artery entrapments

Popliteal artery entrapment syndrome (PAES) is symptomatic compression of the popliteal artery due to anatomical variation of myotendinous structures or calf muscle hypertrophy. First described in 1879, PAES incidence is estimated to be as high as 3.5% in the general population, with subtypes based on relative locations of the gastrocnemius and popliteus muscles and associated tendons, the popliteal artery and vein, and the tibial nerve.[1-4] We describe a case of a 27-year-old female with PAES and associated entrapment of the anterior tibial (AT) artery, which had a high bifurcation.

CASE REPORT

A 27-year-old female was referred for bilateral lower extremity claudication progressive over 6 months before presentation. Pain occurred with active plantarflexion at the ankle with the knee in full extension. Her right leg was more symptomatic than the left, and she denied any smoking history or family history of peripheral arterial disease. The differential diagnosis included PAES, exertional compartment syndrome, neurogenic claudication, atherosclerotic peripheral arterial disease, and adventitial cystic disease. A diagnostic workup by an outside provider included at rest bilateral computed tomography angiography that demonstrated widely patent popliteal arteries at rest but bilateral high AT artery origins that coursed anterior to the popliteus muscles (Fig 1).

On physical examination, pedal pulses were palpable at rest with multiphasic Doppler signals bilaterally. During active plantar flexion, however, the pulses were nonpalpable with complete loss of Doppler signals in the bilateral dorsalis pedis and posterior tibial arteries. While resting ankle-brachial indices were 1.02 bilaterally, treadmill exercise testing was terminated early due to pain. Duplex ultrasound examination revealed multiphasic waveforms in the bilateral popliteal arteries at rest with a near four-fold increase in the peak systolic velocity on the right (from 100 to 390 cm/s) during active plantar flexion (Fig 2). On the left, the popliteal artery displayed a preocclusive pattern during active plantar flexion with blunted and resistive waveforms. Diagnostic angiogram revealed enlarged sural collaterals bilaterally with a high AT artery bifurcation as previously demonstrated on computed tomography angiography (Fig 3, *A*). Bilaterally, during active plantar flexion, there was early filling of the large sural collaterals with no flow in the AT artery and significant compression of the popliteal arteries (Fig 3, *B*). These findings were consistent with concurrent AT and popliteal artery entrapment, and the patient was offered surgical release on her more symptomatic right leg.

Surgical exposure was via a posterior approach with a vertical incision created over the small saphenous vein. The small saphenous vein and sural nerve were protected and retracted laterally. Dissection commenced between the two heads of the gastrocnemius muscle, revealing the tibial nerve, which was protected with a vessel loop and retracted laterally (Fig 4, *A*). The popliteal vein was encountered with fibrous attachments that required additional dissection to allow lateral mobilization (Fig 4, *B*). After ligating several venous tributaries and mobilizing the vein, the popliteal artery was visualized deep to it (Fig 4, *C*). Extensive

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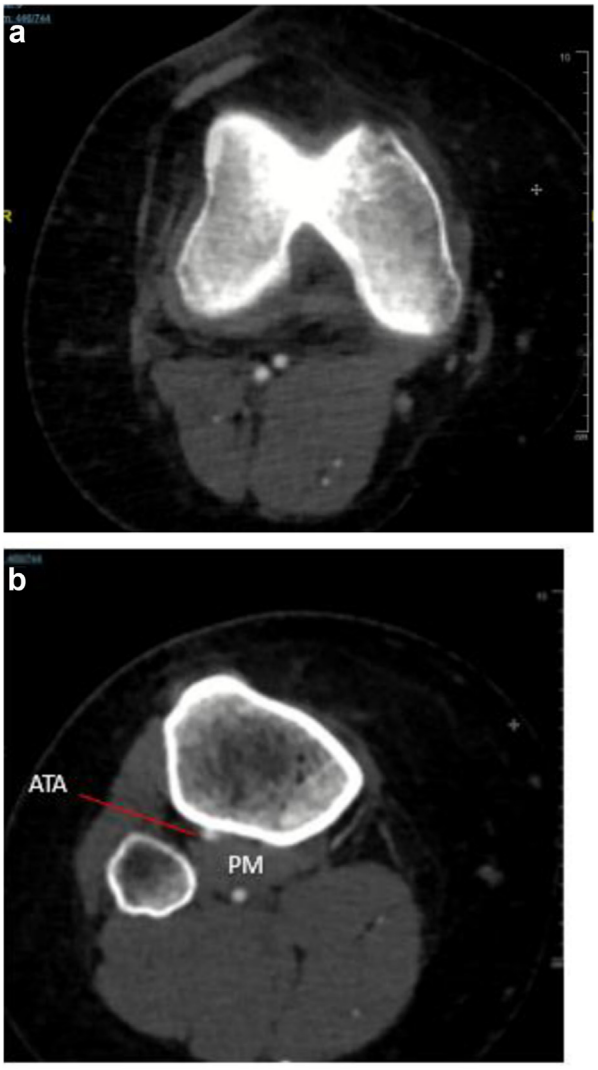

Fig 1. Right lower extremity computed tomography angiography (CTA). **(A)** Proximal origin of anterior tibial artery (ATA). **(B)** ATA anterior to the popliteus muscle (PM).

fibrous bands encasing the artery consistent with type III PAES were sharply dissected circumferentially from the popliteal artery. More proximal dissection revealed the popliteus muscle with the AT artery coursing below it (Fig 4, *D*). A popliteus myomectomy was performed with electrocautery and curved Mayo scissors to completely resect the popliteus muscle from the underlying AT artery (Fig 4, *E* and *F*). Intraoperative ultrasound examination confirmed no compression of the popliteal and AT arteries with passive maneuvers (Fig 5). We did not note any external evidence of arterial injury, and the duplex ultrasound examination was normal as well; therefore, no arterial reconstruction was required. The patient discharged home on postoperative day 1. At her 4-month follow-up visit, she endorsed complete resolution of her right-sided symptoms. She has since undergone release of her left-sided PAES and is awaiting follow-up evaluation.

DISCUSSION

A high index of suspicion for PAES is necessary in young patients without atherosclerotic risk factors who present with claudication symptoms.[5-7] Standard workup includes history, physical examination, treadmill exercise testing, and duplex ultrasound examination with provocative maneuvers. A diagnostic angiogram is the ideal imaging modality to confirm PAES, supported by enlarged sural collaterals and dynamic loss of flow in the popliteal artery with provocative maneuvers as seen in this patient.[6,8,9] Cross-sectional imaging was performed for this patient by an outside provider, and although it revealed that the patient had a high AT artery bifurcation, this was also visualized on a diagnostic arteriogram and did not change the surgical approach. According to a previously published algorithm, all types of popliteal entrapment and anatomical derangements can be treated from a posterior exploration of the popliteal fossa.[8,9] Axial imaging is, therefore, not necessary for diagnosis or surgical planning.

Given the risk of cumulative vascular damage from repetitive popliteal artery trauma, complete surgical release of an entrapped popliteal artery should be performed in all confirmed cases.[6,7,10-12] An intraoperative ultrasound examination was performed in this case to confirm complete surgical release of the popliteal and AT arteries, and it can even aid in the clinical resection of the gastrocnemius muscle in cases of type VI PAES.[13] Intraoperative ultrasound examination can be performed with the aid of a vascular laboratory technician and a sterile hockey-stick probe that is placed on the popliteal artery within the surgical wound. Resting and passive ankle flexion maneuvers by the surgeon can be performed to visualize any significant changes in waveform morphology or velocities that would indicate more arterial release is needed.

Almost all cases of PAES involve only the popliteal artery with or without the popliteal vein. There are only a few case reports of PAES with concurrent AT entrapment. In most cases of AT entrapment, the AT is compressed distally by ligamentous structures such as the interosseous membrane and ankle extensor retinaculum, but can be compressed proximally by myotendinous structures such as fibrous bands from the plantaris muscle.[14-18] Surgical release of a high proximal origin of the AT artery with entrapment symptoms has been described in a couple of cases, both with resection of a portion of the medial head of the gastrocnemius—one via an unspecified approach including resection of the popliteus and in another case via a posterior approach including the plantaris rather than the popliteus.[14,15] Our patient's case of a high AT bifurcation with entrapment by the popliteus muscle released via popliteus without gastrocnemius resection describes successful treatment through a posterior exploratory approach

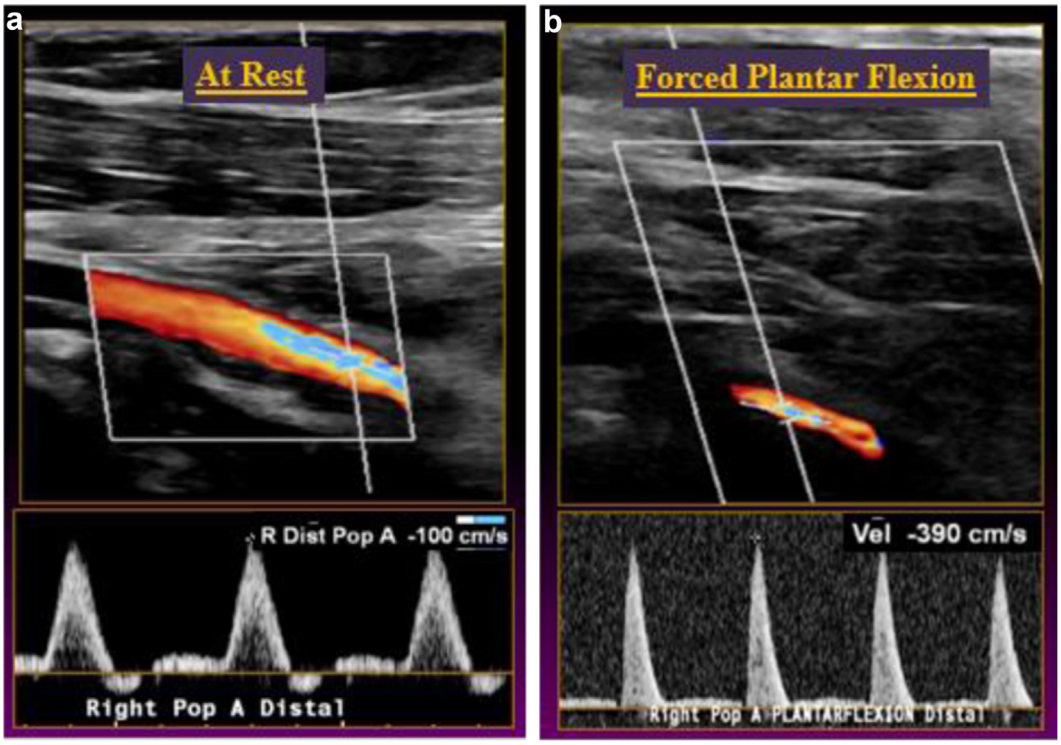

Fig 2. Right leg preoperative duplex ultrasound examination. **(A)** At rest. **(B)** Active plantarflexion.

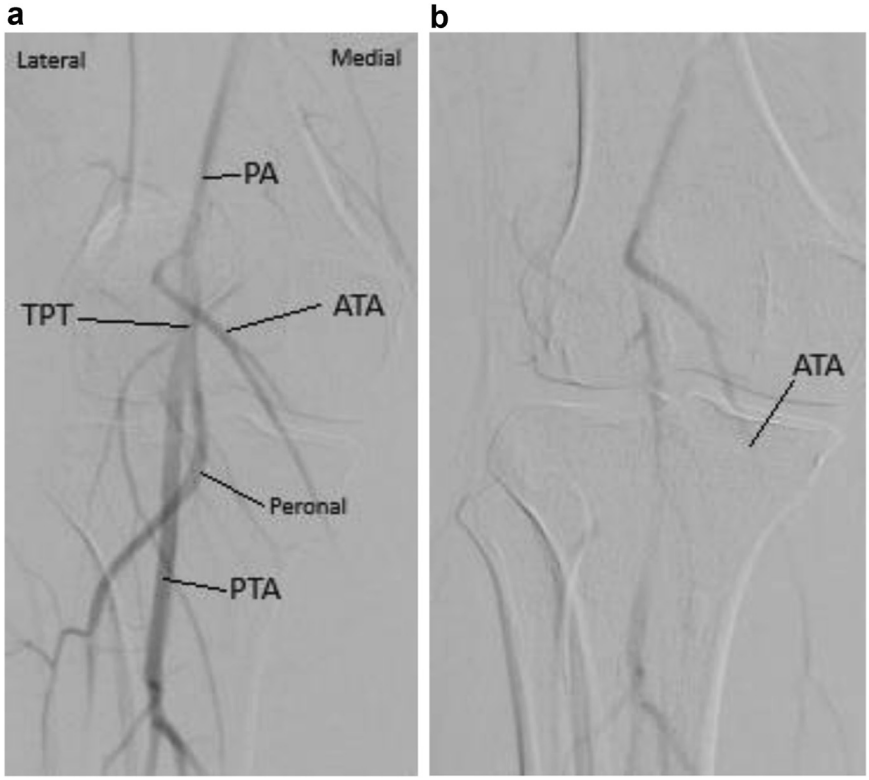

Fig 3. Right leg diagnostic angiogram. **(A)** Enlarged right sural collaterals at rest and high origin of the anterior tibial artery (ATA). *PA*, popliteal artery; *PTA*, posterior tibial artery; *TPT*, tibioperoneal trunk. **(B)** Right popliteal and anterior tibial artery (ATA) effacement with active plantarflexion.

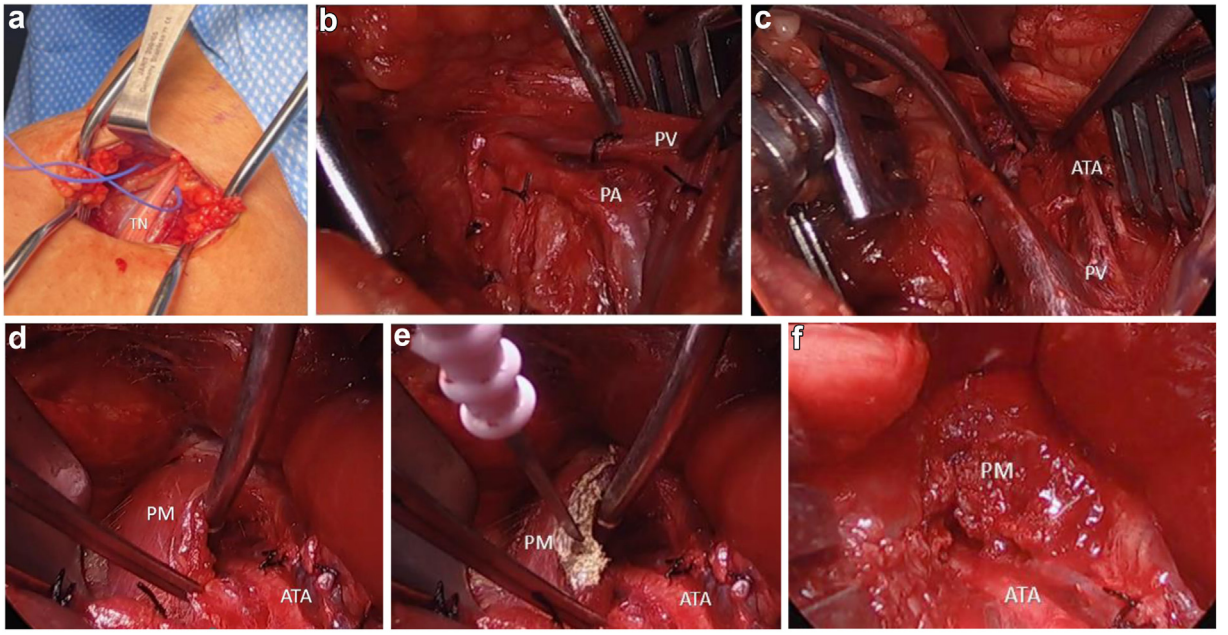

Fig 4. Operative exposure. **(A)** Protection of the right tibial nerve (*TN*). **(B)** Lateral retraction of the popliteal vein (PV). **(C)** Proximal exposure of the anterior tibial artery (*ATA*) deep to the PV. **(D)** Popliteus muscle (*PM*) encasing the ATA. **(E)** Popliteus muscle (*PM*) myomectomy. **(F)** Complete release of the ATA after PM myomectomy.

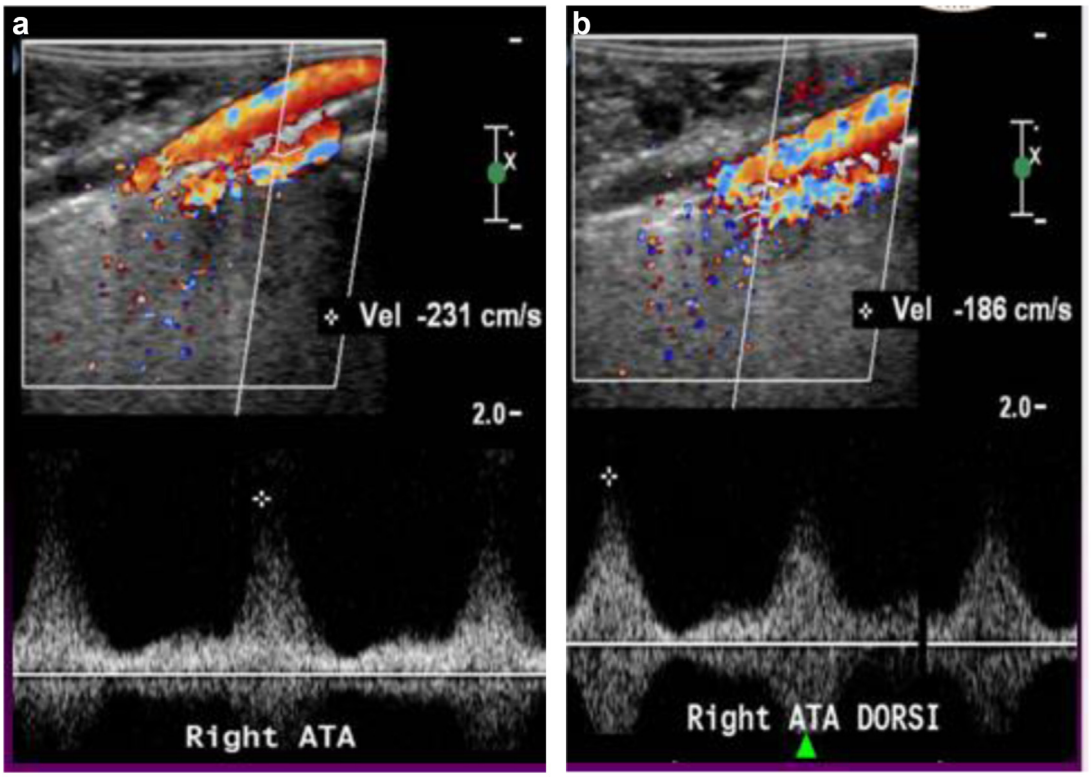

Fig 5. Intraoperative duplex ultrasound examination of the right anterior tibial artery (*ATA*) after surgical release. **(A)** At rest. **(B)** Passive dorsiflexion.

and targeted complete release, which we use for all cases of PAES at our institution. Release of more distal entrapment at the level of the interosseous membrane has been described via an anterolateral longitudinal incision between the tibialis anterior and extensor hallucis longus muscles where the interosseous membrane is then partially resected to enlarge the hiatus. Alternatively, nonsurgical release has been described with ultrasound-guided needle hydrodissection of the interosseous membrane, albeit without complete relief of symptoms.[16,17] Even more distally, entrapment of the AT artery at the extensor retinaculum is managed via a pretibial incision two-thirds of the way down the lower leg with periarterial dissection for ascending superficial fascial release and partial opening of the extensor retinaculum over the AT artery followed by angioplasty.[18] Arterial reconstruction is not described in any of these cases.

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DISCLOSURES

None.

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