Intravascular ultrasound as a novel tool for the diagnosis and targeted treatment of functional popliteal artery entrapment syndrome

Anna E. Boniakowski, MD,^a Frank Davis, MD,^a Dani Campbell, MD,^b Minhajuddin Khaja, MD,^c and Katherine A. Gallagher, MD,^a Ann Arbor, Mich; and Indianapolis, Ind

ABSTRACT

Functional popliteal artery entrapment syndrome can be difficult to diagnose, as the imaging modalities presently employed are designed to detect anatomic entrapment. We describe a novel imaging technique to aid in diagnosis in this cohort. A 22-year-old cyclist presented with exercise-limiting claudication. Magnetic resonance angiography with provocative maneuvers was nondiagnostic. Digital subtraction angiography revealed long-segment occlusion of the popliteal artery with plantar flexion; however, the specific site of compression remained unclear. Intravascular ultrasound allowed specific localization of compression and further confirmed the diagnosis. Thus, we report this as an adjunctive imaging modality to definitively diagnose functional popliteal artery entrapment syndrome and to assist in operative planning. (J Vasc Surg Cases and Innovative Techniques 2017;3:74-8.)

Popliteal artery entrapment syndrome (PAES) is an uncommon cause of lower extremity claudication. It usually affects the young to middle-aged athletic population without atherosclerotic risk factors.¹ The pathologic process is a result of compression of the popliteal artery by adjacent muscles and tendons, leading to vascular claudication.²

Given its transient and positional nature, diagnosis of PAES is not always straightforward. Whereas multiple diagnostic imaging modalities are available to evaluate this syndrome, the results are often nondiagnostic, leading to delay in diagnosis and subsequent risk of vascular injury.^{3,4} Furthermore, the specific site of compression is frequently not apparent on magnetic resonance angiography (MRA). Thus, we present for the first time the use of intravascular ultrasound (IVUS) as an adjunctive imaging modality to definitively diagnose PAES, particularly in cases of functional entrapment, and to localize the specific site of popliteal artery compression. The patient consented to this publication.

CASE REPORT

A 22-year-old male cyclist presented with recently worsening exercise-limiting claudication bilaterally (right worse than left).

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His past medical history and surgical history were unremarkable, family history was negative for any vascular disease, and he was a nonsmoker without other risk factors for atherosclerotic vascular disease. On physical examination, he had palpable bilateral femoral, popliteal, dorsalis pedis, and posterior tibial pulses.

Although his resting ankle-brachial indices (ABIs) were normal, his exercise ABIs dropped to 0.6 on the right and 0.7 on the left. Computed tomography angiography did not reveal a thickened iliac artery concerning for endofibrosis. MRA with provocative maneuvers did not show any findings suggestive of cystic adventitial disease or popliteal entrapment (Fig 1).

Given his symptoms and reduction in exercise ABIs, we had continued suspicion for popliteal entrapment. Thus, we performed angiography with provocative maneuvers and vasodilator administration. Angiography revealed no abnormalities bilaterally in neutral position (Fig 2, A), but on both passive dorsiflexion and active plantar flexion, the right midpopliteal artery demonstrated a complete occlusion that was more pronounced after administration of a vasodilator (Fig 2, B, arrow). Because these images revealed a long-segment occlusion of the popliteal artery, the exact site of compression was unclear. IVUS was used to better delineate the specific area of compression. With the patient in active plantar flexion, we noted on IVUS a complete compression of the midpopliteal artery region (Fig 2, C, arrow) and subsequent release of compression and resolution of vessel patency on release of flexion (Fig 2, D). Furthermore, the contralateral popliteal artery also revealed significant narrowing on active plantar flexion.

Given these findings, he underwent operative release through a posterior approach. Curvilinear incision was made in the popliteal fossa. There were no anatomic abnormalities present, but the medial head of the gastrocnemius muscle appeared hypertrophied, suggesting functional entrapment. The artery did not appear fibrotic or damaged on ultrasound examination and visual inspection, and thus no arterial repair was necessary. The gastrocnemius muscle was released from the medial head

From the Department of Surgery, Section of Vascular Surgery,^a and Department of Interventional Radiology,^c University of Michigan, Ann Arbor; and the Vascular Surgery, St. Vincent Medical Group, Indianapolis.^b

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Correspondence: Anna E. Boniakowski, MD, 1500 E Medical Center Dr SPC 5867, Ann Arbor, MI 48109 (e-mail: annaeli@med.umich.edu).

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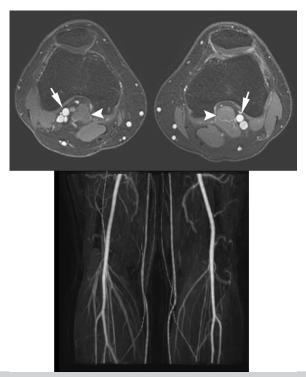


Fig 1. Magnetic resonance imaging (MRI) TI-weighted echo image after contrast enhancement revealing patent popliteal arteries bilaterally (*arrows*) and normal anatomic location of the medial head of the gastrocnemius muscle (*arrowheads*).

and debulked significantly. Because this was functional entrapment, the medial attachments of the soleus and the plantaris tendon were excised. In addition, under direct vision, the rigid band of anterior soleus fascia was excised from its tibial attachment laterally to the fibula. After completion of the procedure, the patient was extubated and flipped into a supine position; an angiogram was obtained, revealing resolution of the popliteal compression on plantar flexion (Fig 3). In addition, the patient reported cessation of symptoms on aggressive plantar flexion during angiography.

DISCUSSION

PAES is a cause of nonatherosclerotic peripheral arterial disease usually affecting the young to middle-aged population. It is caused by repetitive but transient positional compression of the popliteal artery by an abnormal relationship to its surrounding musculotendinous structures. It is considered a progressive disease, as repetitive compression can result in progressive arterial damage and ultimately arterial occlusion.^{5,6}There are six types of PAES. Types 1 and 2 result in deviation of the popliteal artery medially to the medial head of the gastrocnemius muscle. Types 3 and 4 result from normal anatomic location of the popliteal artery but slips of the gastrocnemius or fibrous bands from the popliteus muscle constricting the artery. Type 5 refers to the aberrant location of the popliteal vein, and type 6 is functional

entrapment, without anatomic abnormalities. Although types 1 and 2 could be ruled out by magnetic resonance imaging (MRI), given the anatomic location of the popliteal artery in our patient, it is possible that small muscle fibers were present that were not visible on MRI (type 3 or 4) or, more likely, that he was experiencing functional entrapment (type 6).

There is no consensus as to the optimal means of evaluating a patient with symptomatic PAES. Early on, patients usually complain of intermittent claudication that resolves with rest. Most often, resting ABIs are normal, although occasionally there may be a reduction in the exercise ABIs. However, in advanced stages, patients may present with acute limb ischemia due to a severely stenotic or occluded popliteal artery.^{1.7}

Several diagnostic imaging modalities have been proposed to aid in diagnosis; however, many are often nondiagnostic. Duplex ultrasound is unreliable, as the criteria suggested in several reports were not found to be sensitive or specific.^{1,6} In fact, more than half of patients with normal popliteal arteries had a functional occlusion on duplex ultrasound in both neutral and active plantar flexion.⁸

MRI can be useful for structural assessment of the gastrocnemius musculature and identification of abnormal fascial bands that may be tethering the popliteal artery. It is thought that dynamic MRI performed with provocative maneuvers offered promising results, although patients often complain of severe pain while required to maintain the foot in prolonged plantar flexion or dorsiflexion, resulting in significant motion artifact.⁶ MRA has also been found to have a high false-negative rate, particularly when stenosis is <50%.⁹ Functional PAES is especially difficult to diagnose, and thus IVUS may aid significantly in the accurate diagnosis and localization of compression in this disorder.

Digital subtraction angiography is another modality that can help to identify flow-limiting lesions of the popliteal artery. Especially when it is performed with provocative maneuvers, alterations in flow based on positional changes can be assessed. However, as the patient in this report demonstrated, digital subtraction angiography with active plantar flexion identified a long-segment popliteal occlusion without identifying the specific area of compression.

In this report, we propose IVUS as an adjunctive tool in the diagnosis of functional PAES. We recommend use of IVUS as a staged procedure during preoperative workup as an adjunct to angiography to help guide operative planning as not only can IVUS provide information on exact location of compression, it can also provide further information on the quality of the vessel wall, particularly in more advanced cases. Fortunately, this patient did not appear to have vessel wall degeneration from repetitive external compression on IVUS. Thus, IVUS can assist with precise operative planning on the basis of both

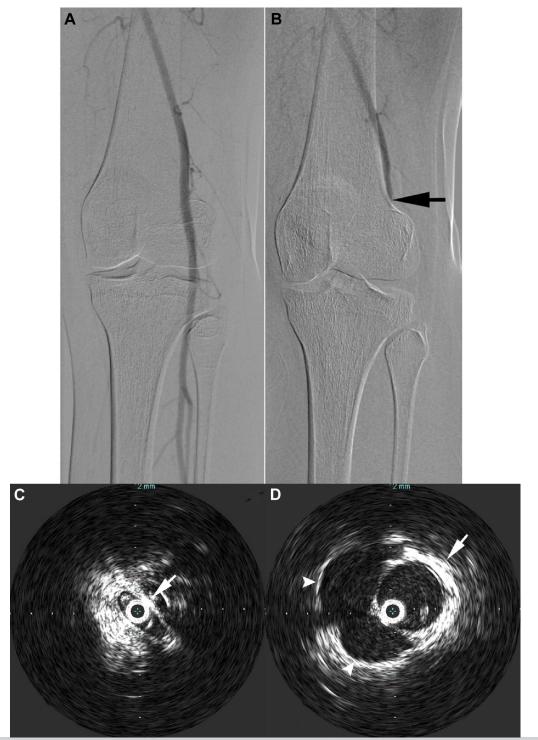


Fig 2. A and **B**, Digital subtraction arteriogram of the right popliteal artery revealing patency in neutral position **(A)** and long-segment occlusion on active plantar flexion **(B)**; the *arrow* denotes the area of occlusion. **C** and **D**, Intravascular ultrasound (IVUS) images of both compression on active plantar flexion **(C)** and resolution of compression on neutral positioning **(D)**; the *arrow* denotes the popliteal artery, and the *arrowheads* denote medial head of gastrocnemius muscle.

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Fig 3. A, Digital subtraction arteriogram after surgical release revealing resolution of compression with active plantar flexion. **B**, Digital subtraction arteriogram in neutral position pictured for comparison.

the exact location of the lesion and the potential need for arterial repair.

CONCLUSIONS

PAES is an uncommon cause of vascular claudication in the young, athletic population without risk factors for vascular disease. Given the ambiguity of the multiple available diagnostic imaging modalities, we propose use of IVUS as a definitive diagnostic technique to aid in the diagnosis and treatment of PAES.

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