Review Article on Compressive Vascular Syndromes

Popliteal artery entrapment syndrome

Stanley Bradshaw, Peiman Habibollahi, Jayesh Soni, Marcin Kolber, Anil K. Pillai

Division of Vascular and Interventional Radiology, Department of Radiology, University of Texas Southwestern Medical Center, Dallas, TX, USA

Contributions: (I) Conception and design: S Bradshaw, P Habibollahi, M Kolber, AK Pillai; (II) Administrative support: P Habibollahi, M Kolber, AK Pillai; (III) Provision of study materials or patients: S Bradshaw, J Soni, AK Pillai; (IV) Collection and assembly of data: S Bradshaw; (V) Data analysis and interpretation: S Bradshaw; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Anil K. Pillai, MD. Associate Professor of Vascular and Interventional Radiology, Division Chief of Vascular and Interventional Radiology, Department of Radiology, University of Texas Southwestern Medical Center, Dallas, TX, USA. Email: Anil.Pillai@utsouthwestern.edu.

Abstract: Popliteal artery entrapment syndrome (PAES) is an uncommon cause of lower extremity exertional claudication due to external compression of vascular structures in the popliteal fossa. A developmental anomaly due to an aberrant relationship of the artery with the surrounding myofascial structures contributes to the vascular compromise. PAES presents in younger, athletic patients without atherosclerotic risk factors. Typical presentation of unilateral or bilateral, intermittent claudication in the feet and calves specifically after exercise and relieved by rest in a young person should prompt further evaluation. Early diagnosis and intervention is essential for preventing thromboembolic complication and in worst cases limb loss. Initial tests with Ankle Brachial indices or Doppler ultrasound with provocative maneuvers will prompt more definitive cross sectional imaging studies. CTA or MRA also with provocative maneuvers has a high sensitivity and specificity and will clinch the diagnosis. There are six subtypes based on the relationship of the vascular structure with surround myofascial structures. CTA and MRA can characterize the subtypes and guide surgical planning. Catheter directed thrombolysis may be attempted adjunctively to reduce surgical thrombectomy or resolve distal emboli; however, myotendinous decompression with or without vascular repair is the definitive treatment. Long term surgical outcomes are satisfactory when the distal circulation is preserved.

Keywords: Arterial occlusive diseases; popliteal artery; popliteal artery entrapment syndrome (PAES); lower extremity

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Introduction

Popliteal artery entrapment syndrome (PAES) is a rare vascular disorder defined as compression of the popliteal artery by aberrant myotendinous structures in the popliteal fossa. PAES was first identified by Stuart, a medical student in 1879, who noted an anomalous course of the popliteal artery medial to the medial gastrocnemius muscle in an amputated leg (1). PAES often presents as exertional claudication in a young, otherwise healthy individual. These atypical symptoms should raise suspicion and prompt diagnostic workup. While uncommon, early diagnosis of PAES is crucial to prevent chronic vascular injury resulting in arterial stenosis, thrombosis, and possible amputation (2).

Anatomy and pathophysiology

Symptoms of PAES arise from the compression of the neurovascular bundle within the popliteal fossa by the surrounding musculotendinous structures. Popliteal vein and/or tibial nerve compression may exist alone or in combination with arterial pathology (3). The syndrome exists in six forms, divided into two categories: anatomic/congenital and functional. Anatomic PAES results from abnormal embryological development of the popliteal artery, gastrocnemius muscle, or other myofascial structures

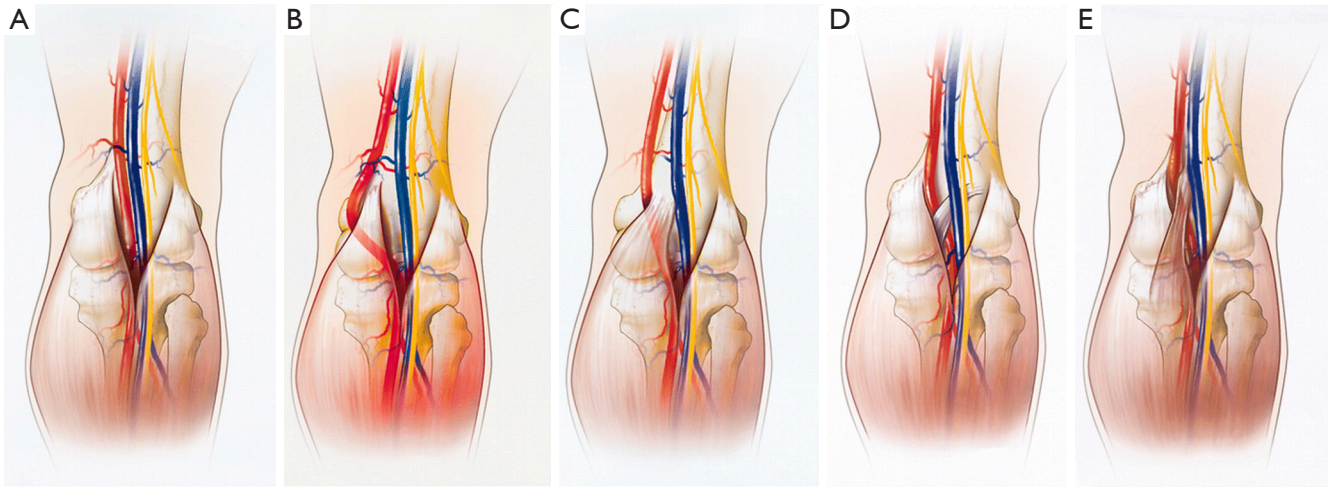

Figure 1 Graphic illustrations show normal anatomy of popliteal fossa and common variants responsible for arterial entrapment. (A) Normal popliteal artery is adjacent to and lateral to medial head of gastrocnemius muscle, which is normally attached just superior to medial femoral condyle. (B) Type I. Popliteal artery takes abnormal course medial to normally attached medial head of gastrocnemius muscle. (C) Type II. Abnormal embryologic development results in medial head of gastrocnemius attached more laterally than is normal. (D) Type III. Popliteal artery and gastrocnemius are normally positioned, but fibrous band is responsible for entrapment. (E) Type IV. Popliteal artery courses beneath popliteus muscle. Adapted from Macedo TA, Johnson CM, Hallett JW, *et al.* Popliteal Artery Entrapment Syndrome: Role of Imaging in the Diagnosis. Am J Roentgenol 2003;181:1259-65; used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.

within the popliteal fossa (4). Variants are classified into five types (*Figure 1*). Types 1 and 2 involve medial deviation of the popliteal artery relative to the medial head of the gastrocnemius muscle. Types 3 and 4 arise from a normal popliteal artery compressed by slips of the gastrocnemius or fibrous bands from the popliteus muscle. Type 5 involves abnormal location of the popliteal vein (5). The functional variant, Type 6 PAES, occurs without anatomic abnormality but with hypertrophy of the gastrocnemius, soleus, and/or plantaris (6).

Early in the clinical course, the artery is patent except during muscle contraction. The ensuing ischemic symptoms are sudden in onset during exercise but completely resolve with rest. However, progressive popliteal artery injury can occur leading to luminal narrowing and occasionally occlusion, median prevalence 24% (7). Prompt diagnosis and treatment are indicated to prevent chronic sequelae such as critical limb ischemia, thromboembolization, and post-stenotic dilatation with aneurysm formation, median prevalence of 13.5% (7-9).

Epidemiology

The incidence of PAES has been reported between 0.6% and 3.5%, primarily in a young, athletic population without atherosclerotic risk factors (10). Type 4 lesions are the most common, constituting about a third of cases, followed by Type 2 and Type 3 (10). The incidence of functional PAES is not well characterized, though may be more prevalent than its anatomic cousin (11). Mean age at diagnosis is 32 years, with a median male proportion of 83% (3). Bilateral disease is identified in approximately 40% of cases (7). The anatomic variant causes of PAES tend to occur in middle-aged men with lower functional demands, while functional PAES presents in younger, highly conditioned athletes, such as cyclists or military personnel (11-13).

PAES is a challenging diagnosis due to non-specific symptomatology and is often misdiagnosed as exertional compartment syndrome (11,14). The median delay to diagnosis has been reported as 12 months, so a high degree of suspicion is warranted for young, active patients presenting with lower extremity peripheral arterial symptoms (7).

Clinical manifestations

Symptoms of PAES are variable and typically intermittent, depending on the chronicity, acuity, and extent of the pathology. In fact, some popliteal artery occlusion on provocation is estimated to be as high as 80%, though is typically benign and asymptomatic due to the transient nature of entrapment and/or development of collaterals (7). The most common symptoms are intermittent claudication and pain in the feet and calves after exercise (4). Additionally, paresthesia, cramps, coldness, and blanching may occur, typically with an acute onset after exertion. Symptoms resolve with rest or change in position (15). As the disease progresses, vascular injury induces remodeling that can lead to leg swelling, aching rest pain, and fatigue (7). If compressive vascular damage is not corrected early, especially in patients with hypercoagulable risk factors, arterial occlusion and thromboembolism may occur presenting with the hallmark signs of acute limb ischemia (16-18).

However, these manifestations are non-specific to PAES, making the diagnosis difficult to solidify on clinical findings alone. A thorough differential should include exertional compartment syndrome, neurogenic claudication, and atherosclerotic peripheral arterial disease (PAD). A useful differentiation factor is the location of pain. PAES invariably causes pain and tightness in the calf, while exertional compartment syndrome typically localizes to the anterolateral leg via the superficial peroneal nerve (19,20). Spinal etiologies of neurogenic claudication classically cause pain along the posterior aspect of the upper thigh and leg, frequently present bilaterally, and may involve concomitant back pain (21,22).

On physical exam, lower limb arterial pulses are likely to be normal unless severe popliteal artery stenosis or occlusion is present. An ankle-brachial index (ABI) can be useful to screen for PAES. A baseline should be measured at rest, then again after sustained dorsiflexion or other exertion to the point of reproducing the patient's symptoms. ABI drop greater than 30% and/or exertional ABI <0.9 have been suggested as a positive result with nearly 100% sensitivity for anatomic PAES. However this finding is not specific for PAES, nor does it capture all cases of the functional variant (11,23-26). Further, ABI measures do not correlate with the subtype of PAES (27). Therefore, imaging is indicated to make a definitive diagnosis.

Imaging

For the patient with suspected PAES, imaging serves to demonstrate exertional occlusion of the popliteal artery, to localize compressing myofascial structure(s), and to identify thrombosis, aneurysm, or other vascular injury needing repair. Assessment should begin with duplex ultrasound (DUS). An initial scan should be done with the patient prone and at rest to visualize occlusive disease, aneurysm formation, or variant course of the popliteal artery. Then, following provocative measures, particularly maximal plantarflexion against resistance, a second scan should be performed to identify occlusion. Loss of signal, dampened signal, or peak systolic velocity ratio exceeding 200% compared with velocities in the proximal normal segment are considered positive findings (28-30). The major drawback of DUS for diagnosis of PAES is a high false positive rate, reported 72%, as up to 53% of normal subjects may have positional popliteal artery compression (17,31). Despite these limitations, a recent review by Shahi demonstrated that DUS detected stenosis in 65% and full occlusion of the popliteal artery or vein in 27% of patients with confirmed functional PAES (6).

Positive DUS findings or high clinical suspicion should prompt further imaging, necessary to delineate the anatomical subtype of PAES and inform surgical planning. Traditionally, direct angiography was considered as the gold standard, but CTA or MRI are far superior for visualizing the position of the popliteal artery in relation to the gastrocnemius muscle and fascia (7,30,32,33). Both modalities rely on provocative maneuvers by the patient to demonstrate occlusion and imaging can be performed prior to and during these maneuvers (*Figure 2*). Only fat should surround the normal popliteal artery and vein within the popliteal fossa. Any slip of muscle or fascia abutting the vessels may signify occlusive potential, which can be demonstrated as luminal stenosis with exertional positioning (34). MRI with or without MRA is often preferred over CTA to better identify the level and length of compression of the popliteal artery by adjacent hypertrophied muscle (31,35). However, patients may complain of pain while required to maintain the foot in prolonged flexion during MRI acquisition, resulting in significant motion artifact (36). Further, MRA has been found to have a comparatively higher false negative rate, particularly when stenosis is less than 50%, due to underestimation of stenosis relative to

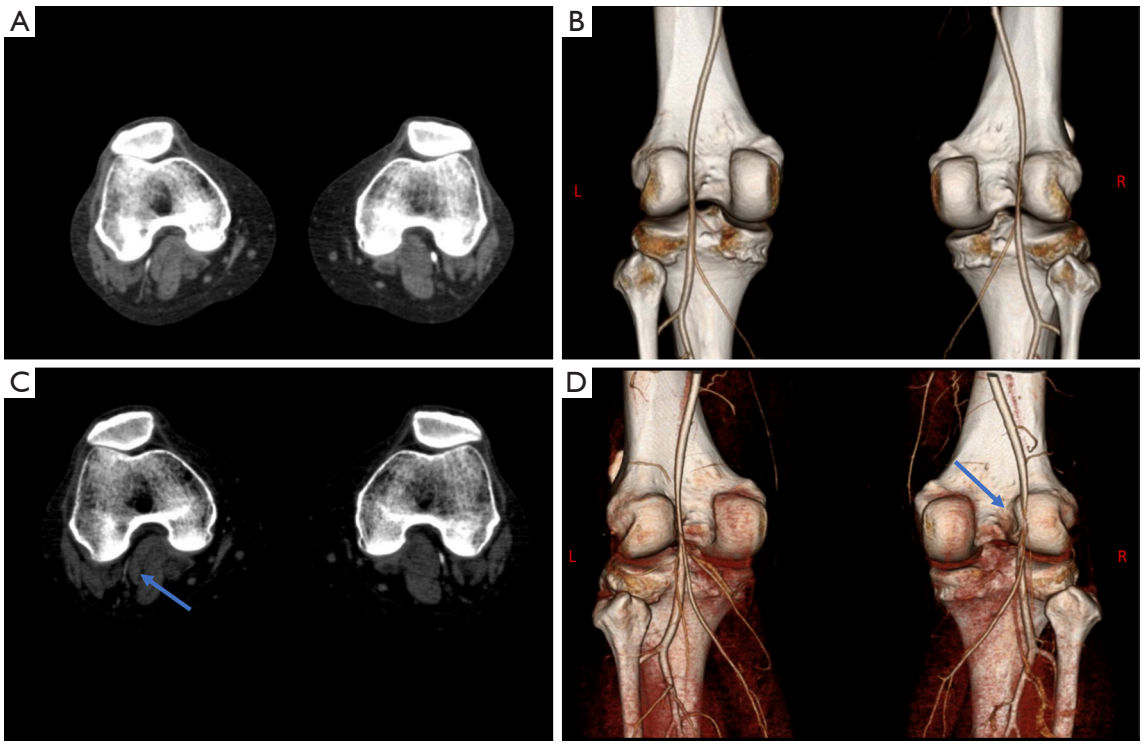

Figure 2 Axial contrast enhanced CT angiogram in the neutral (A) and plantarflexed (C) positions demonstrating bilateral compression and displacement of popliteal artery by the medial head of the gastrocnemius. The right knee (blue arrow) shows greater compression. Coronal CTA reconstructions from the posterior side in the neutral (B) and plantarflexed (D) positions. Complete occlusion of the popliteal artery is shown on the right side (blue arrow).

direct angiography (37,38) (*Figure 3*).

If cross-sectional imaging findings are equivocal or a large thrombus is identified in the popliteal vessels, catheter-directed angiography should be pursued (30). Digital subtraction angiography (DSA) typically reveals medial deviation of the proximal part of the popliteal artery, segmental occlusion of the vessel, and possible post-stenotic dilatation distally during active plantarflexion (39) (*Figure 4*). A modern series from Liu identified significant stenosis in 71% and complete occlusion in 29% with plantarflexion via DSA, in concordance with axial imaging findings (35). Additionally, distal crural emboli have been identified in 43% of cases, prompting consideration of preoperative thrombolysis (39,40). Catheter-directed thrombolysis (CDT) does not have a definitive role in the management of PAES; however, it has been reported as an adjunct to allow a less invasive surgical procedure than would otherwise have been warranted (33,39,41,42). Finally, Causey has demonstrated the use of intravascular ultrasound (IVUS) to better characterize the location and extent of occlusion both pre- and intra-operatively (30,43).

Treatment

Management of PAES depends on the syndrome sub-type (anatomic or functional) and the acuity of presentation. Surgical decompression is crucial to relieve the aberrant anatomic causes of arterial compression. Earlier treatment likely consists only of musculotendinous release, rather than additional arterial bypass, which may be required once severe vascular damage has occurred, with inferior results (3). For the patient with emergent symptoms of acute limb ischemia, assessment should be made according to the Rutherford criteria (44). Viable or marginally threatened extremities (Rutherford I or IIa) may be considered for elective surgery with or without CDT. Immediately threatened (Rutherford IIb) limbs should undergo emergent revascularization (45,46).

CDT may be considered for patients with acute PAES with new, severe symptoms for less than 2 weeks (prior

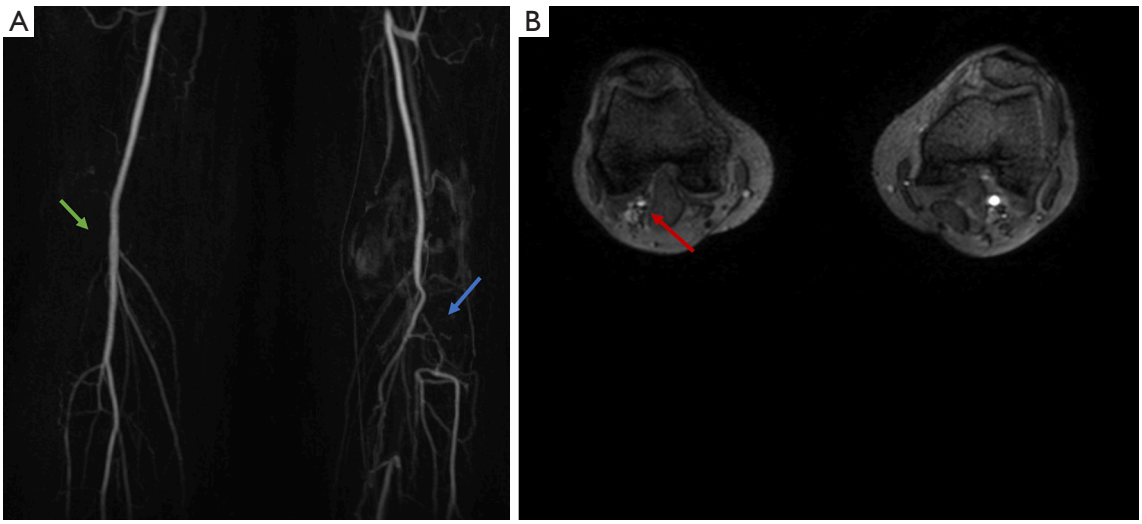

Figure 3 MR angiogram with (A) coronal TOF MAR demonstrating complete occlusion (blue arrow) of the left popliteal, tibio-peroneal trunk with reformation of the anterior, posterior tibial and peroneal arteries. Note the medial deviation of the patent right popliteal artery (green arrow). (B) Axial 2D TOF MAR of a different patient demonstrating extrinsic compression of the right popliteal artery (red arrow) by an aberrantly lateral attachment of the medial head of the gastrocnemius.

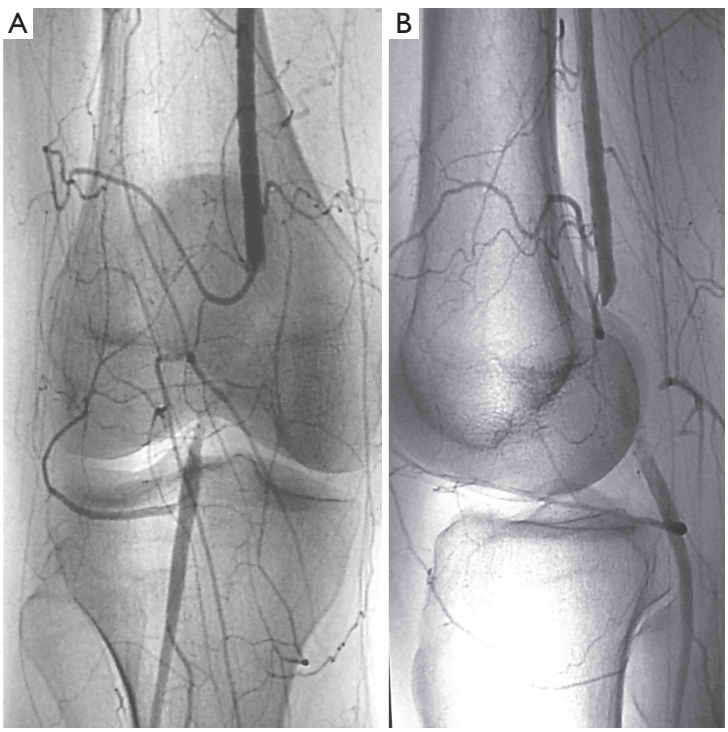

Figure 4 Direct angiography with anterior (A) and lateral (B) views of the knee showing complete occlusion of the popliteal artery with extensive collateral formation. Note the medial deviation of the popliteal artery leading into the popliteal fossa in the anterior view, demonstrating an aberrant course.

to clot organization) and angiographic evidence of acute arterial occlusion (8,47). Both fibrinolytic and mechanical thrombectomy approaches have been successfully reported (8,33,39,41,42,48-52). However, these techniques do not address the underlying anatomic pathology, resulting in high rates of re-stenosis. Thrombolysis may be useful in cases of acute PAES with distal crural emboli to restore distal outflow (53).

Surgical management of PAES is twofold: decompression of the offending musculotendinous structures and repair of vascular injury. In anatomic PAES, surgical correction of the aberrant anatomy is always necessary, as the natural history of the syndrome involves vascular injury and occlusion over time (12). For Types I and II PAES, myotomy of the medial head of the gastrocnemius is performed, followed by re-routing of the popliteal artery. Type III involves resection of the accessory slip. Type IV requires release of the popliteus, re-routing of the popliteal artery, with or without subsequent repair of the muscle. Treatment of Type V is similar, with the additional decompression of the popliteal vein. Outcomes for myotomy are excellent, with 1- and 5-year patency rates of 100%, respectively (54).

If vascular pathology, such as intimal injury and fibrosis with stenosis and/or post-stenotic dilatation, is present repair or revascularization is necessary. Thromboendarterectomy and venous patch arterioplasty versus saphenous vein graft bypass should be considered, with the outcomes for the former reported to be slightly inferior (4,8). Placement of an endovascular stent is not recommended (55). Complications from arterial reconstruction are more frequent, with a median failure rate of 27.5%; however, these events are concentrated among cases with lesions extending beyond the popliteal artery (7,54). Overall, resolution of symptoms has been demonstrated in a median of 77% of patients (7).

While the treatment for anatomic PAES is surgical decompression, management of the functional subtype may vary. Surgical approaches include lysis of fascial attachments, release of the plantaris tendon, and myotomy of the gastrocnemius, soleus, and/or plantaris (11,56,57). Recurrent or residual symptoms occurred in 9.2% of patients, with 7.1% undergoing a revision surgery (6). Botulinum toxin A injection has recently been reported as a non-invasive treatment for functional PAES (6). Similar to its use in chemically denervating the anterior scalene muscles to relieve neurovascular compression in thoracic outlet syndrome, botulinum toxin may relax the hypertrophied gastrocnemius to reduce PAES symptoms (58). One to three injections of 100 MU (mouse units) of botulinum toxin into the medial head of the gastrocnemius and/or plantaris resulted in partial symptomatic improvement in 82.9% of cases. While the duration of action of botulinum toxin is only 3 to 6 months, the lack of any serious complications makes this a reasonable therapeutic trial for patients with functional PAES (6,59).

Conclusions

While PAES is an uncommon cause of exertional claudication, the presentation of symptoms in a young, otherwise healthy individual should prompt timely diagnostic workup. Early surgical treatment before vascular injury and remodeling occur provides for superior outcomes. Specific localization of pain, ABIs, and popliteal DUS can provide rapid indications of pathology. CTA or MRA with provocative maneuvers then classify PAES subtypes and guide surgical planning. Myotendinous decompression with or without vascular repair resolves symptoms in the vast majority of patients and abrogates progression of the syndrome.

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