Atypical Presentation of Popliteal Artery Entrapment Syndrome: Involvement of the Anterior Tibial Artery

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Popliteal artery entrapment syndrome (PAES) is a rare condition that should be suspected in a young patient with exertional lower extremity pain. We report the case of an 18-year-old female volleyball player with bilateral exertional lower extremity pain who had been previously diagnosed with tendinitis and periostitis. Diagnostic studies showed entrapment of the left popliteal artery and the left anterior tibial artery. To our knowledge, there has only been 1 previous report of anterior tibial artery involvement in PAES.

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INTRODUCTION

Popliteal artery entrapment syndrome (PAES) is a rare condition that causes intermittent claudication in the lower extremities of young persons. Symptoms are due to an abnormal extrinsic compression of the popliteal artery as it courses through the popliteal fossa [1]. It can be potentially limb threatening if it is not recognized early [2]. Unfortunately, PAES can be confused with alternative diagnoses such as tendonitis, periostitis, stress fractures, and chronic exertional compartment syndrome (CECS) [1]. This case illustrates the challenge in diagnosing PAES, while presenting a variant that has previously been reported only once.

CASE PRESENTATION

An 18-year-old female volleyball player with no past medical history presented with pain in her legs, approaching 2 years in duration. There was no associated trauma. Her pain was worse on the left and was located laterally and posteriorly, involving her calves. Her pain was related to repetitive jumping as well as prolonged standing while working. She had no rest pain. The pain, when severe, was associated with numbness and tingling in the lateral feet. On examination, sensation, strength, and range of motion of her lower extremities were normal. Pulses at rest were also normal. Palpation revealed diffuse tenderness throughout both lower legs, particularly laterally and posteriorly.

She had been previously evaluated by other providers and was diagnosed with tendonitis and periostitis. Plain films of her bilateral tibia and fibula were unremarkable. Magnetic resonance (MR) imaging of her bilateral tibia and fibula performed 1.5 years before her visit showed a very-low-grade stress reaction in the posterior aspect of the left mid-tibial shaft. Her pain persisted despite conservative management including activity modification, anti-inflammatory medications, and a course of physical therapy.

At this point, it was thought that her MR imaging findings were an asymptomatic stress reaction and other diagnoses needed to be investigated. The diagnosis of CECS was considered as a possible etiology of her symptoms. Compartment pressure testing was performed before and 5 minutes after exercise, but this proved to be unremarkable (Table 1). Although the pre-exercise compartment pressures in the right anterior and deep posterior compartment were high ($\geq$15 mm Hg), the pressure did not increase after exercise. In addition, all of the post-exercise compartment pressures were below the diagnostic threshold for CECS ($\geq$20 mm Hg).

Considering the failed management of tendonitis and periostitis and the unremarkable compartment pressure testing, a vascular entrapment syndrome was considered. Doppler

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Table 1. *Compartment pressure measurements*

Compartment	Before Exercise (mm Hg)	After Exercise (mm Hg)
Left anterior	12	11
Left lateral	9	11
Left superficial posterior	5	11
Left deep posterior	10	12
Right anterior	16	14
Right lateral	12	14
Right superficial posterior	12	12
Right deep posterior	22	17

Measurements were obtained with a Stryker Intra-Compartmental Pressure Monitor System after injection of approximately 0.2 cc of normal saline within each compartment. Five-minute post-exercise measurements were taken after 45 minutes of exercise, in an effort to reproduce symptoms.

ultrasound was used to evaluate waveforms and systolic pressures of lower extremity arteries with their associated ankle—brachial indexes (ABI) at rest, plantar flexion, and dorsiflexion. The results are noted in Table 2. At rest, these were normal (Figure 1). With passive and active dorsiflexion, there was significant dampening of the waveform as well as decreased systolic pressures (indicated by ABIs significantly below 1.0) at the posterior tibial arteries (Figure 2). These results suggested PAES, because of the arterial compression noted with dorsiflexion.

The patient was referred to a vascular surgeon. Computed tomographic angiography was performed, which did not reveal any anatomic abnormalities. Dynamic angiography was then performed, with popliteal artery patency assessed with the ankle in neutral position and then with active dorsiflexion, passive dorsiflexion, active plantar flexion, and passive plantar flexion. This showed narrowing of the left popliteal artery in the neutral position, which worsened with active plantar flexion. The left anterior tibial artery, noted to have an abnormally proximal origin, was also impinged with active plantar flexion. No change was noted with entrapment maneuvers on the right side.

Table 2. *Bilateral lower extremity arterial physiology study*

	Left (mm Hg)	Right (mm Hg)
Rest		
Brachial	111	112
Dorsalis pedis	134 (ABI 1.21)	123 (ABI 1.10)
Posterior tibial	132 (ABI 1.19)	131 (ABI 1.17)
Passive plantar flexion		
Posterior tibial	135 (ABI 1.22)	142 (ABI 1.27)
Active plantar flexion		
Posterior tibial	131 (ABI 1.18)	130 (ABI 1.16)
Passive dorsiflexion		
Posterior tibial	46 (with severely dampened waveform) (ABI 0.41)	48 (with severely dampened waveform) (ABI 0.43)
Active dorsiflexion		
Posterior tibial	73 (with severely dampened waveform) (ABI 0.66)	79 (with severely dampened waveform) (ABI 0.71)

Given the result of these studies combined with the unsuccessful treatment of other possible causes, the vascular surgeon offered surgical release of the vascular entrapment in the left lower extremity. The patient underwent release with resection of the popliteus, a portion of the medial head of the gastrocnemius (volume of approximately half of a golf ball), and some of the tendons of the medial head of the gastrocnemius, which inserted on the lateral side of the popliteal fossa. Postoperatively, the patient continued in physical therapy. Over time, this resulted in significant improvement of her symptoms at follow-up. Afterward, she was able to return to playing volleyball.

DISCUSSION

PAES is a rare disorder that can be potentially debilitating. The true incidence of the syndrome is unknown. The rarity has been described in a retrospective review of records for 843 patients who underwent surgical treatment of symptomatic lower extremity claudication without an apparent vascular or orthopedic etiology. In this study, 796 patients (94%) were diagnosed with CECS, whereas 33 patients (4%) were diagnosed with PAES [3]. However, PAES may be underrecognized, in part because CECS and PAES can coexist. A recent case report describes a patient with continued symptoms despite a diagnosis of CECS treated with fasciotomies. He was later found to have PAES [4].

The literature is conflicting in regard to gender predilection. Zünd et al reported that 17 of 20 of their own patients with PAES were male [5]. In contrast, Dany et al noted a predilection toward females in a study with 279 subjects, in which 24% of females and 13% of males in the study had the entrapment [6]. PAES is generally a condition of young persons lacking atherogenic risk factors. Zünd et al reported that onset of symptoms in their patients with PAES occurred at a mean age of 33.5 years, with a diagnosis made at a mean age of 38.5 years [5]. Gourgiotis et al noted that surgery in their patients with PAES was performed in 38 patients at a mean age of 21 years [7].

PAES is caused by an anomaly in the relationship between the popliteal artery and the medial head of the gastrocnemius (types I-III), although the popliteus may be involved instead (type IV). The popliteal vein may also be compressed in PAES (type V). Levien and Veller also described a functional entrapment, indicating the lack of an anatomical abnormality (type VI) [8]. PAES can also be caused by other surrounding musculature, such as the soleus and hamstrings [6]. In addition, anterior tibial artery involvement has been reported in a 23-year-old man with pain in his legs upon exertion, who was eventually diagnosed with PAES [8].