Popliteal Artery Entrapment Syndrome: A Case Series With Variable Timing of Diagnosis and Outcome

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ABSTRACT: Popliteal artery entrapment syndrome (PAES) is a rare condition that occurs primarily in young active patients. It is most commonly a result of aberrant anatomy involving the popliteal artery and the surrounding musculoskeletal structures. Patients typically present with a lack of cardiovascular risk factors and most commonly describe intermittent claudication in the early stages. If undiagnosed, PAES can lead to acute ischemia and a threatened limb as a result of complete arterial occlusion or embolism. A high index of suspicion, early diagnosis, and surgical release of the entrapment are crucial for good outcomes and limb loss prevention. We present a case series that highlights the diagnosis of PAES and represents the variability of outcomes based on timing of diagnosis and treatment.

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Popliteal artery entrapment syndrome (PAES) was first characterized in 1879 by T.P. Anderson Stuart and is one of the more unique causes of lower-extremity vascular insufficiency.1,2 Patients affected by PAES are usually younger active individuals who may have hypertrophied musculature, without significant cardiovascular risk factors. They typically present with unilateral intermittent symptoms of lower-extremity claudication.3-6 Other symptoms may include paresthesia, pallor, pain, and in severe cases, paralysis of the affected limb.1,3,4 Symptoms may classically occur during or following activity in early stages of the disease and may progress to symptoms at rest if the condition is undiagnosed and complications develop.1,3 It has been proposed that the mechanism of a flexed knee with repeated active plantar flexion while driving a vehicle may result in gastrocnemius muscle hypertrophy causing popliteal artery entrapment.3 To make the diagnosis, a careful history and physical exam are necessary. Physical exam findings in early disease include decreased posterior tibial and dorsalis pedis pulses upon leg hyperextension and active plantar flexion. Duplex ultrasonography of the popliteal artery, computerized tomography (CT) scans, and contrast arteriography may also be used to detect stenosis.5 The lack of cardiovascular risk factors and active lifestyle can make recognizing PAES challenging; however, it is important to recognize this disease due to the high complication rate in the patient group.3

CASE #1
A 28-year-old, healthy, athletic female was referred for outpatient vascular evaluation due to progressive bilateral lower-extremity claudication with ambulation of <50 feet. She had no cardiovascular risk factors, no significant family history, and no significant past medical history. She reported a history of bilateral exertional calf pain over the last 10 years with more recent subacute worsening. Eight years prior, she reported being diagnosed with bilateral exertional compartment syndrome and undergoing bilateral lower-extremity dual compartment fasciotomy. She denied any significant improvement in her symptoms after the surgery. Due to her recent progressive symptoms, she was referred for a repeat vascular evaluation. The patient underwent selective digital subtraction angiography bilaterally with provocative maneuvers including active plantar flexion and dorsiflexion. This revealed normal bilateral angiographic images at rest, including normal appearance of the left popliteal artery (Figure 1A). With active plantar flexion, angiography revealed complete occlusion at the P1 segment of the popliteal artery (Figure 1B) consistent with popliteal artery entrapment. She was referred to a tertiary-care center for definitive treatment and underwent surgical decompression of the popliteal artery with partial excision of the proximal gastrocnemius and popliteus muscles. She made a full recovery with resolution of claudication and paresthesia with a gradual full return to activity.

CASE #2
A 37-year-old, healthy, active male was referred to a local wound clinic by his primary-care physician; the patient’s chief complaints were left lower-extremity pain and non-healing superficial hallux ulceration. He described a 2-year history of slowly progressive numbness and pain in his left lower leg that initially presented with vigorous activity and was now present with light to moderate activity. He denied any smoking history and any family history of vascular disease. Due to the non-healing ulceration, non-invasive
testing was ordered including arterial Doppler (ankle brachial index [ABI]) and arterial duplex. The ABI was noted to be abnormal at 0.7 on the left, and the arterial duplex showed blunted monophasic flow below the knee. He underwent invasive selective digital subtraction angiography revealing complete left popliteal occlusion (Figure 2). Due to progression to critical limb ischemia with rest pain, he underwent multiple endovascular interventions in attempts for durable revascularization. This was met with short-term success, but persistent re-occlusion. Ultimately, he presented to an outside community hospital, was diagnosed with exertional compartment syndrome, and underwent left lower-extremity anterior and lateral compartment fasciotomy. Due to no improvement in symptoms, he was transferred to a tertiary-care center for further evaluation. CT angiogram of the left lower extremity showed muscular hypertrophy causing popliteal artery entrapment, resulting in his presenting condition of critical limb ischemia due to functional occlusion. A femoral to anterior tibial artery bypass surgery was performed that was technically successful. Despite this, his pain continued to be uncontrolled. Due to repeated re-occlusion of the popliteal artery, he underwent surgical exploration of the popliteal fossa and Hunter’s canal with decompression. Ultimately, he received an above-the-knee amputation. Following intensive physical rehabilitation and use of a prosthetic, he has since returned to an active lifestyle.

**CASE #3**

A 30-year-old, healthy, active female was referred to vascular surgery evaluation with complaint of bilateral lower-extremity pain. A history of running long-distance marathons led to the presentation. Physical examination revealed an increased left popliteal pulse. Dorsiflexion was limited with a positive Abduction Test (Figure 1). Ankle brachial index testing revealed an abnormal left ABI of 0.6. Angiogram of the left popliteal artery showed no significant stenosis or occlusion. Further imaging was obtained in the form of digital subtraction angiography. This showed complete occlusion of the popliteal artery. The patient underwent endovascular therapy with a stent placement with technical success. Despite this, his pain continued to be uncontrolled. Due to persistent re-occlusion, he underwent surgical exploration of the popliteal fossa and Hunter’s canal with decompression. Ultimately, he was found to have severe muscular hypertrophy causing entrapment of the popliteal artery. A femoral to anterior tibial artery bypass surgery was performed that was technically successful. Despite this, his pain continued to be uncontrolled. Due to repeated re-occlusion of the popliteal artery, he underwent surgical exploration of the popliteal fossa and Hunter’s canal with decompression. Ultimately, he received an above-the-knee amputation. Following intensive physical rehabilitation and use of a prosthetic, he has since returned to an active lifestyle.
Claudication. She reported exacerbation of symptoms with exertion and relief with rest, and worse symptoms on the right. She also noted paresthesias and numbness in her lower legs while in a seated position. She denied any history of coronary disease, tobacco use, or other medical problems. At her initial visit, her peripheral pulse exam was normal and she had no skin changes or ulcerations distally. A diagnostic selective angiogram of the bilateral lower extremities was then performed, including provocative maneuvers of the right leg (plantar flexion, dorsiflexion, and knee flexion). This showed normal resting flow and abrupt cessation of flow of the popliteal artery with active plantar flexion (Figures 3A and 3B). Arterial duplex also showed complete occlusion of the P2 segment of the popliteal artery with plantar flexion (Figures 4A and 4B). She was sent for surgical evaluation, and decompressive surgical

**Figure 3.** (A) Angiography showing right popliteal artery compression with plantar flexion. (B) Angiography of passive lateral bent-knee angiography with grossly patent popliteal vessel.

**Figure 4.** (A) Duplex ultrasound showing brisk patent popliteal flow at rest. (B) Duplex ultrasound with plantar flexion showing complete popliteal compression and obliteration.
release is planned. Due to early identification, she is expected to make a full recovery and remains under active surveillance.

CASE #4

A 17-year-old, active male was referred for evaluation due to complaints of progressive claudication of his left lower extremity over 1 month prior to presentation. Duplex ultrasound was performed, showing normal inflow, a patent popliteal artery at rest with focal aneurysmal change, thrombus, and complete compression and cessation of distal flow with active dorsiflexion. Due to progressive symptoms and duplex findings, he was brought urgently for diagnostic angiography and endovascular intervention. Angiography was performed, with active provocation maneuvers showing complete compression of the popliteal artery with dorsiflexion and cessation of flow (Figures 5A and 5B). Infrapopliteal flow had already been compromised with occluded peroneal and anterior tibial arteries (Figure 5C). He underwent directed local tPA lytic therapy for 24 hours with follow-up angiography and no further endovascular intervention. He was placed on oral anticoagulation and movement restrictions to limit further trauma to the popliteal artery and was referred for urgent surgical treatment. He underwent successful surgical decompression of the popliteal artery by resection of the left medial head of the gastrocnemius and vein patch angioplasty of the popliteal artery without complication and is recovering well. The patient remains under active surveillance and is expected to make a full recovery.

DISCUSSION

The true incidence of PAES can only be estimated due to lack of awareness, misdiagnosis, and the relative rarity of presentation. Likely only the most severe cases present for evaluation and workup and obtain a true diagnosis. The pathology often involves abnormal congenital anatomy surrounding the popliteal artery. The different types of PAES are categorized based upon the embryological development of the popliteal fossa. During development, the neurovascular bundle and developing muscle groups are maturing in the popliteal fossa in a dynamic manner. Any disruption of the migration pattern or timing of development can result in abnormal anatomy.7

Figure 5. (A) Resting angiogram showing popliteal stenosis. (B) Active dorsiflexion with complete popliteal artery compression. (C) Infrapopliteal angiogram showing thromboembolic occlusion of the peroneal and anterior tibial arteries.
There are six types of PAES described. The first four describe the anatomical anomalies ultimately causing the entrapment (Figure 6). Type I entrapment is caused by the popliteal artery forming prior to the migration of the medial head of the gastrocnemius muscle. This results in medial deviation of the popliteal artery, causing the popliteal artery to become entrapped between the muscle and the femoral condyle.5,8,9 Type II entrapment involves disruption in migration of the medial head of the gastrocnemius by a prematurely formed popliteal artery. In Type III entrapment, abnormal remnants of mesoderm may be present, causing slips of muscle or fibrous bands to entrap the artery. Type IV entrapment is the result of the popliteal artery remaining in the primitive position, deep to the popliteus muscle. Type V entrapment occurs when both the popliteal artery as well as the popliteal vein are involved.7,10,11 Type VI entrapment is acquired due to gastrocnemius or plantaris muscle tendon hypertrophy causing popliteal artery occlusion during active plantar flexion or leg extension.1,4,5,7,12

Other acquired forms of popliteal artery entrapment can result from compressive masses, local edema, or articular knee disorders.13 Movements that may exacerbate symptoms include repetitive leg extension and plantar flexion. This repetitive trauma of the popliteal artery may result in arterial injury, atherosclerosis, arterial aneurysm, and thrombus formation.11,14,15

The differential diagnosis includes atherosclerotic disease, exertional compartment syndrome, thrombosed popliteal artery aneurysm, and cystic adventitial disease. Diagnosis of PAES early in its course is crucial and relies upon disease awareness and suspicion based on the history and physical. Non-invasive and sometimes invasive tests are needed to demonstrate both the anatomic and functional aspects of the disease. Tests including arterial duplex or angiography with provocative maneuvers can show the functional result of compression. CT or magnetic resonance imaging may demonstrate the anatomic variant causing the compression.

Treatment of PAES is based on the type of entrapment and degree of arterial pathology. It can range from simple myotomy of the medial head of the gastrocnemius, to vascular repair or grafting if arterial damage is present, to endarterectomy in the presence of a thrombus.3,4 However, surgical treatment is indicated in all symptomatic patients.5,3 Young athletes may be advised to limit or stop contact sports to reduce the chance of trauma to the popliteal artery until definitive therapy.4 Early recognition based on history, physical exam findings, and imaging results is paramount to initiate treatment and avoid the severe late presentation of acute limb ischemia.

CONCLUSION

PAES is a rare and potentially devastating condition that affects otherwise healthy, active individuals. Its potentially severe clinical outcome can be avoided if diagnosed early. Due to the lack of cardiovascular risk factors and otherwise functional nature of the patients, there is often a delay after first presentation while other diagnoses are entertained. Sometimes, accurate diagnosis and definitive treatment are delayed until irreversible damage is inevitable. Awareness of PAES and an index of suspicion need to be maintained for timely diagnosis and definitive treatment, which may involve endovascular intervention and surgical decompression.

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