Case report

Popliteal Artery Entrapment Syndrome: A Rare Cause of Claudication in a Young Athlete

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Abstract

Popliteal artery entrapment syndrome is an important infrequent cause of serious disability among young adults and athletes. We hereby describe a case of popliteal artery syndrome and its clinical implications. Physicians should be aware of the possibility of popliteal artery entrapment in young patients presenting with progressive arterial insufficiency.

Keywords: Popliteal, artery, syndrome, treatment, radiology

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Introduction

Popliteal artery entrapment syndrome is an important infrequent cause of serious disability among young adults and athletes because of anomalous arrangement of popliteal artery and its surrounding musculotendinous structures (1). This anomaly usually affects young men (aged 20 to 40 years) (1). It is the most common cause in the rare category of intermittent claudication in young adults (1).

Extrinsic arterial compression causes chronic vascular microtrauma, early arteriosclerosis and thrombus formation that causes distal ischemia (2). We hereby report the case.

Case report

A young 35-year-old football player presented with one-year history of intermittent claudication of the right calf precipitated from playing football and during training. The symptoms had been persistent and had worsened lately. The symptoms were accompanied by coldness, numbness, and blanching of the foot. A short rest relieved the symptoms. The patient was a non- smoker and had been active in sports from his teenage years.

Physical examination revealed an athletic young man, weighing 70 kg, 175 cm in height. His pulse rate was 75 beats/min, in sinus rhythm, and a blood pressure recording of 125/80 mmHg. In the lower limbs, there were no skin or temperature changes. With the knee extended, no pulse could be detected from the popliteal artery, dorsalis pedis and posterior tibial arteries of the right foot.

Patient underwent a lower limb angiogram due to the symptoms of intermittent claudication. It revealed a stenotic segment of the right popliteal artery with a short segment narrowing of the posterior tibial artetry and thrombus in the peroneal artery. Accompanying collaterals were also noted (Fig. 1A & B). Patient however defaulted follow up and presented eight months later with worsening of the symptoms.

Magnetic resonance imaging (MRI) of the right knee that was performed at his second presentation showed medial deviation of the course of the distal superficial femoral artery (SFA) and the popliteal artery coursed through the distal semimembranosus and the proximal aspect of the medial head of gastrocnemius muscle. The popliteal vein and tibial trunk which followed the normal tract contributed more evidence to the medially deviated course of the popliteal artery (Fig. 2).

Magnetic Resonance Angiography (MRA) of the lower limb revealed a long segment with no signal flow of the entire right popliteal artery posterior to the femoral condyle. There were collaterals arising from the distal right femoral artery that reconstituted the trifurcations (Fig.3). On review of the raw data, the flow signal of the right anterior tibial artery was faint. There was almost no flow signal in the common tibioperoneal trunk, posterior tibial artery and the peroneal artery. The common femoral and proximal right superficial femoral artery as well as the left lower limb arteries were normal in calibre.

There was an abrupt cut off of the flow signal of the normal right popliteal artery with total compression of its caliber superior to the musculotendinous junction of the head of the medial gastrocnemius muscle. Throughout the 8 months, there was worsening of the symptoms evidenced by progressive stenosis of the right popliteal artery with presence of collateral arteries supplying the trifurcation. Thus MRI findings concluded as Type I Popliteal artery entrapment syndrome.

Case was discussed with the patient for surgical intervention. However, he has not decided for further treatment.

Discussion

Popliteal artery entrapment refers to compression of the popliteal artery due to an abnormal anatomical relationship between the vessel and the neighbouring musculotendinous structures or surrounding muscle hypertrophy. This may lead to functional impairment or arterial compression, which may cause chronic vascular microtrauma of the arterial wall with intramural haematoma or thrombus, episodes of distal embolisation, aneurysm, dissection, and later causes thrombosis with acute distal ischaemia (3).

Popliteal artery entrapment syndrome (PAES) affects young sports participants, athletes, or soldiers who have no known cardiovascular risk factors. These groups habitually undertake vigorous exercise producing a high degree of muscular development, which masks the occult disorder. The most common



Fig. 1A&B: Digital substraction angiography of the right lower limb showing the stenotic segment of the right popliteal artery (white arrows), which is at the level of the right femorotibial joint (1A). Distally, there are collaterals with evidence of narrowing of the posterior tibial artery (white arrow) and thrombus within the proximal peroneal artery (white arrowhead).

sporting activities associated with PAES include basketball, football, rugby, and the martial arts. The true prevalence of PAES is unknown. Patients are typically young (60% <30 years old), healthy males (15:1 male predilection) and PAES can be bilateral in 25% of cases (3). Our patient was a 35 year-old-male, an active football player with no known medical history and a non-smoker.

The frequency of presentation of PAES according to the type is as follows: type 1, 20%; type 2, 38%; and

type 3, 26% (Table 1) (4). Our patient had type I entrapment syndrome by the radiological features.

A thorough history is pertinent to accurately diagnose this disorder. A history of extreme tenderness, soreness, and tightness of both posterior lower legs with physical activity, asymptomatic periods of rest, and progressive symptoms over time are all possible indicators of popliteal artery entrapment syndrome. Our patient presented with one-year history of intermittent claudication on strenuous activity and relieved with rest.

The absence of a pulse in the lower extremities with tensile plantar flexion may also be a major indicator; however, this indicator may not be a reliable clinical screening test. If the history and screening are used in conjunction with arteriography, as seen in this case, popliteal artery entrapment syndrome could be properly diagnosed (5).

The first diagnostic technique of choice in patients with possible PAES should be duplex color Doppler sonography with high-frequency transducers. This noninvasive technique enables correct vision of the 3 anatomic segments of the popliteal artery. Doppler color sonography, however, provides little information about possible muscle or tendon anomalies, and other diagnostic techniques are necessary (6).

Lower limb angiography, though is the traditional reference standard, is an invasive test; furthermore, it fails to provide information about musculotendinous structures. Computed tomography, with or without 3-dimensional reconstruction, is good for diagnosing popliteal artery entrapment, but it too uses contrast material and ionizing radiation (6).

MRI and MRA, with or without gadolinium, show the popliteal arteries and veins together with the surrounding tissues. They can be undertaken at rest and during provocative manoeuvers, thus showing functional entrapment even in persons with no symptoms of PAES. MRI is therefore the diagnostic method of choice when there is clinical evidence or a duplex Doppler imaging suggestion of PAES (6).

Our patient underwent lower limb angiography firstly due to the symptoms of claudication. The angiogram showed short segment partial stenosis of the right popliteal artery. However, the course of the popliteal artery was not obvious from the angiography thus the possibility of popliteal artery entrapment syndrome was not evident. In view of the significant degree of stenosis, surgical intervention i.e. a bypass was



Fig. 2: Axial T1W image shows a medial deviation of the course of the distal SFA (white arrow) through the distal semimembranosus (white arrowhead) and the proximal aspect of the medial head of gastrocnemius muscle (short black arrow). The popliteal vein (black arrow) is in the normal position surrounded by fat



Fig. 3: Coronal MRA shows non-opacification of the popliteal artery with reconstitution from the adjacent collaterals (white arrow) 8 months after the initial femoral angiogram indicative of disease progression.

discussed but the patient deferred for any intervention to seek for a second opinion. As the symptoms worsened, MRI with MRA of the lower limbs was requested 8 months later. This shows the medially deviated course of the popliteal artery. In addition, it was compressed by the semimembranosus and the tendon of the medial gastrocnemius muscle. These findings clinched the diagnosis of PAES along with evidence to support progression of the disease. Unfortunately, the patient was still not keen for any surgical management at the time this article was prepared. The differential clinical diagnosis of unilateral intermittent claudication in an active sports participant younger than 30 years should include muscle rupture, tendinopathies, acute and chronic compartment syndrome, popliteal artery adventitial cystic disease, stress fractures, medial tibial periostitis, effort-induced venous thrombosis, nerve impingement, and fascial defects (7).

From this case, we conclude that the treating physician should be aware of the possibility of popliteal artery entrapment in a young patient presented with progressive arterial insufficiency. It would save further significant morbidity if the diagnosis is made timely by corroboration of the clinical signs, symptoms and radiological findings.

Table 1. Classification of popliteal artery entrapmentsyndrome (4)

Туре	Findings
Type I	MHG is normal, PA is deviated medially
	and has an aberrant course
Type II	MHG is located laterally, no deviation of
	PA
Туре	Abnormal muscle bundle from MHG
III	surrounding the PA
Туре	PA is located deeply and entrapped by the
IV	popliteus muscle or a
	fibrous band
Type V	Popliteal vein is also entrapped with any
	type of PA

MHG: medial head of gastrocnemius muscle, PA: popliteal artery

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