CASE REPORT

Vascular claudication in a young patient: popliteal entrapment syndrome

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Popliteal artery entrapment syndrome is an important, albeit infrequent, cause of severe disability among young adults and athletes with anomalous anatomic relationships of the musculotendinous structures surrounding the popliteal artery. We report a case where duplex ultrasonography and dynamic magnetic resonance arteriography was used to diagnose popliteal artery entrapment. We used a posterior surgical approach to give the best view of the anatomic structures compressing the popliteal artery. Compression had not yet damaged the arterial wall, therefore operative decompression of the artery by resection of the aberrant muscle was sufficient. The result was complete recovery, with absence of symptoms and with patency verified by Doppler examination. We conclude that emergency physicians who encounter young patients with progressive lower limb arterial insufficiency should be aware of the possibility of popliteal artery entrapment. Early diagnosis using physical examination, history taking, and imaging is necessary. The treatment of choice is surgical correction to achieve normal anatomy within the popliteal fossa.

Introduction

Popliteal artery entrapment syndrome (PAES) is an uncommon cause of lower limb claudication, usually occurring in younger patients who lack the risk factors for atherosclerosis. Most of the people who develop this entity exercise actively, leading to calf hypertrophy, which unmasks the anatomical abnormality. We present a case of PAES and comment on the presentation, methods of diagnosis, and treatment.

Case report

A 34-year-old personal athletic trainer noticed a progressive deterioration in his walking distance secondary to sharp pains in the left calf and cramps and paraesthesia in his left foot while training. A physical examination was unremarkable and no abnormal biochemical or haematological data were detected, and he had no relevant medical history. Doppler ultrasonography revealed normal bilateral ankle-brachial arterial indices when his feet were in a neutral position. After forced plantar and dorsi flexion, however, the left-sided arterial signals were diminished and the arterial index dropped. Magnetic resonance angiography (MRA) demonstrated no popliteal artery blood flow during forced active plantar flexion of the foot against resistance (Fig a), thus confirming PAES. Due to the progressive nature of this entity, a prophylactic operation was planned. At operation, a constricting band formed by the medial head of the gastrocnemius was excised and no arterial damage was documented (Figs b, c). He made an uneventful recovery.

Discussion

In 1879, Anderson Stuart, a medical student, became the first to describe the anatomical basis of popliteal entrapment.[1] Love and Whelan[2] established the term PAES in 1965. 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[3] 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, thrombosis with acute distal ischaemia.[4]

Popliteal artery entrapment syndrome 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 unmasks the occult disorder. The most common 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

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PAES can be bilateral in 25% of cases.[5]

Popliteal artery entrapment syndrome is a developmental abnormality that results from an abnormal relationship between the popliteal artery and the gastrocnemius muscle or, rarely, an anomalous fibrous band or the popliteus muscle. The abnormal position causes deviation and compression of the artery. There are essentially four anatomic variants of PAES (Table[6]). Type V is any of the four anatomic variants with the popliteal vein included.[5] Recently, a 'functional' PAES has been described in patients with normal anatomy (type VI). In such cases, compression of the popliteal artery may be due to an anatomically normal but hypertrophic muscle.[7] This entity is usually seen in well-conditioned athletes, sometimes coinciding with the use of anabolic steroids.[8] The frequency of presentation is as follows: type 1, 20%; type 2, 38%; and type 3, 26%.[6]

Symptoms include transient tingling or coldness in the foot, with later intermittent claudication. If diagnosis is delayed, there may be irreversible arterial damage, which can impair the viability of the affected limb. The differential 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.

Physical examination requires careful palpation of the popliteal and pedal pulses with the ankle in both passive dorsiflexion and forced plantar flexion. Pulse loss during these manoeuvres is considered pathognomonic[9] although pulse reduction can occur in individuals without abnormalities.[10] Use of Doppler ultrasonography and duplex mapping, which detect alterations in the three-phase pulse pattern and allow dynamic visualisation of the popliteal artery during different manoeuvres, can also help to establish a diagnosis. Magnetic resonance imaging and MRA show the popliteal arteries and veins together with the surrounding tissues. They can be undertaken at rest and during provocative manoeuvres, thus showing functional entrapment even in people with no PAES symptoms. For these reasons, this is the diagnostic method of choice.[4]

Treatment is indicated in all cases of anatomical entrapment. If the diagnosis is made during the dynamic or functional entrapment stage, before the artery shows evidence of parietal or intraluminal damage, ceasing the exercises causing muscle hypertrophy will sometimes suffice; otherwise, resection of the causative anomalous muscle or fibrous tissue is required. In cases with local damage to the popliteal artery, where the distal vessels remain patent, resection of the affected segment and

一名年輕人的血管性跛行：腘動脈受壓綜合徵

腘動脈受壓綜合徵是因腘動脈與周圍的肌腱的位置關係異常，而導致腘動脈受壓。這種病雖然罕見，卻是導致一些年輕人及運動員嚴重殘缺的一個重要原因。本文報告一個利用複式超聲波及動態磁共振動脈造影來確診腘動脈受壓綜合徵的案例。我們經後路取得壓在腘動脈上組織的最佳檢視位置。由於壓迫並未損及動脈壁，只需把引致壓迫的肌肉切除便可。手術後病人完全康復，用多普勒技術測試亦顯示徵狀消失。醫生如發現年輕病人有下肢缺血症狀，應考慮腘動脈受壓綜合徵的可能性。必須要替病人仔細檢查，查明病史，及進行腘動脈造影。這種病首選手術治療，以緩解及修復腘窩的異常。

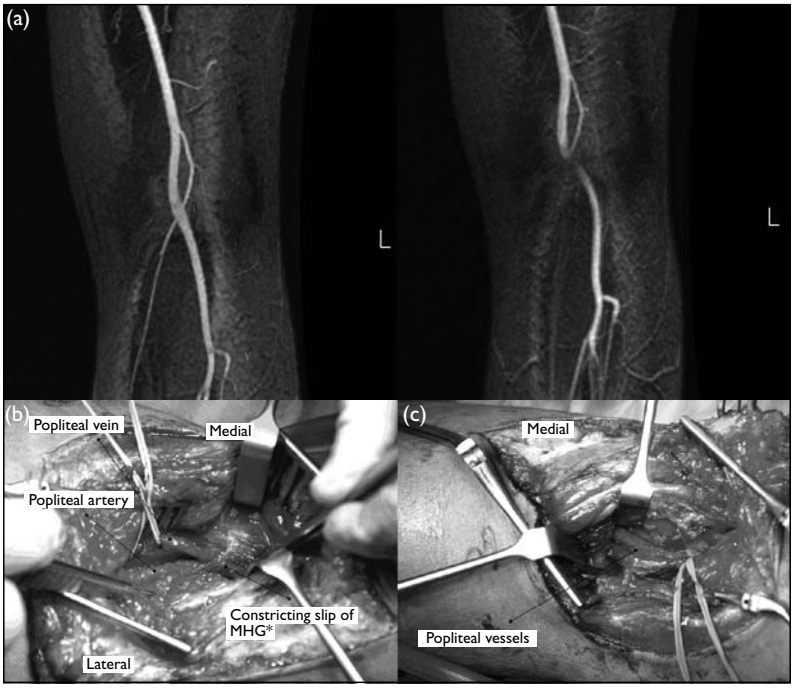

FIG. (a) Magnetic resonance angiography showing compression of the left popliteal artery induced by dorsiflexion of the foot. (b) At operation, a constricting band formed by the medial head of the gastrocnemius was identified, and (c) the vessels skeletonised following excision of the accessory band

* MHG denotes medial head of the gastrocnemius

TABLE. Popliteal vessels entrapment classification[6]

Type	Description
I	Medial deviation of the popliteal artery around the medial insertion of the gastrocnemius muscle
II	Minimal medial deviation of the popliteal artery with entrapment by aberrant medial attachments of the gastrocnemius muscle
III	Accessory bundle of the gastrocnemius muscle, which compresses a normally positioned popliteal artery
IV	Normally positioned popliteal artery entrapped by the popliteal muscle
V	Above abnormalities with popliteal vein compression
VI	Functional entrapment

reconstruction of the arterial axis by placement of an autologous venous bypass graft is advised. In cases where there is obstruction of the popliteal artery due to acute thrombosis, intra-arterial fibrinolysis is indicated. After patency of the affected vessel has been restored, corrective surgery to the musculotendinous lesion and vein grafting are indicated.

In conclusion, this case study serves as a reminder for emergency physicians that athletes can develop PAES, a rare arterial condition. Although this diagnosis is infrequently reported in sports medicine publications, the condition has the potential to create significant morbidity and can cause a devastating debility. Timely, accurate clinical recognition of the signs and symptoms of PAES is necessary to facilitate the appropriate medical referral and treatment needed to effectively restore arterial patency and reduce the risk of potential limb loss.

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