

Bilateral popliteal entrapment syndrome associated with plantar flexion of a phenomenon

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ABSTRACT

Popliteal artery entrapment syndrome (PAES) presents with intermittent claudication; however, in the later stages, acute ischemia can occur as a result of complete arterial occlusion or embolism. We present a patient that symptomatic with plantar flexion. PAES was diagnosed with magnetic resonance imaging (MRI) and angiography. In operation medial head of gastrocnemius was resected and popliteal artery resection end to end anastomosis technic was performed. After surgery patient discharged with no symptom and problem. Ischemic symptoms in the lower leg in young patients with extremity movement, PAES must be considered. Correct diagnosis for PAES are important for preventing deterioration in the patient's clinical condition.

Key words: Popliteal entrapment syndrome, Claudication intermittans, Plantar flexion, Medial head of gastrocnemius.

INTRODUCTION

Popliteal artery entrapment syndrome is an uncommon entity typically affecting young athletic males who present with symptoms of calf claudication.¹ This syndrome is related to an abnormal embryological development with an aberrant anatomical relationship between the popliteal artery and its surrounding structures.² Repetitive insult to the popliteal artery can cause arterial damage and lead to aneurysm, thromboembolism, and arterial thrombosis. This aggressive natural history warrants early diagnosis and treatment.¹ The anomalous anatomic relationship responsible for compression of the popliteal artery is caused by abnormal embryologic development of the popliteal fossa. The muscle or the artery can be responsible for the abnormal anatomy, and numerous variations can result. It is important to recognize the normal appearance of the popliteal fossa anatomy.¹ It is commonly classified into six variants on the basis of its relationship with surrounding structures.³ Therapeutic interventions are dependent upon the anatomical abnormality and symptom severity.³

Doppler sonography has a limited role in the diagnosis of popliteal artery entrapment syndrome. Angiography has been long used for the diagnosis of popliteal artery entrapment syndrome. Arterial luminal changes with plantar or dorsiflexion of the foot are well shown on conventional angiography. MRI can nicely show the abnormal anatomy responsible for the entrapment. In addition, the dynamic compression can also be shown with MRI techniques. The abnormal muscle or fibrous slip responsible for entrapment can be prominent or subtle. MRI, and the popliteal artery occlusion was at the soleal sling site as a result of compression by the soleus muscle, lateral head of the gastrocnemius muscle, plantaris muscle, and popliteus muscle.⁴

We report an unusual bilateral PAES case presenting with claudication intermittans and has ischemic symptoms with plantar flexion.

MATERIAL AND METHODS

A 36 year old police officer has claudication intermittans for 3 years. He referred us with bilateral lower extremity pain, paresthesia and coldness. The patient had no history of smoking. On physical examination he height was 180 cm and weight was 86 kg. Blood pressure was 124/68 mmHg. Bilateral anterior and posterior tibial arteries pulses were

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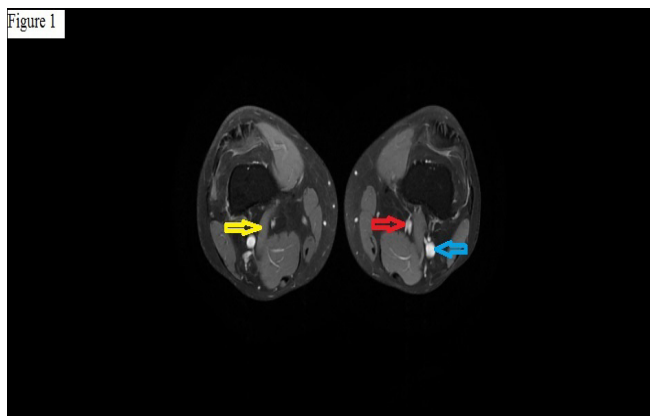


Figure 1: Preoperative MR angiography Medial Head of Gastrocnemius muscle (Yellow arrow), Popliteal artery (Red arrow), Popliteal vein (Blue arrow)

okay. But when he made plantar flexion, bilateral tibial pulses was not taken. During the patient made plantar flexion, the ankle-brachial pressure index was 0.87 and 0.92 on the right and left lower limbs. MRI scan (Figure 1), and conventional angiography (Figure 2) performed. Occlusion was seen bilaterally. He was diagnosed with bilateral popliteal entrapment syndrome.

He was operated one-sided because of postoperative comfort is not good. At operation, prone position was given and S incision was applied to right popliteal fossa. We saw that medial head of gastrocnemius (M.H.G.C) put pressure on popliteal artery medially (PAES type 1) (Figure 3). After partial resection of M.H.G.C, we saw the injury of popliteal artery. Popliteal artery was resected and end to end anastomosis was performed. We gave asetil salisilic asit 100 mg per day after operation. Postoperative follow-up right lower extremity pulses was okay with normal and plantar flexion position. Popliteal artery was normal flow with doppler USG and have no compression with plantar flexion. The patient made a good postoperative recovery, and the ankle-brachial pressure index improved



Figure 2: Preoperative angiography

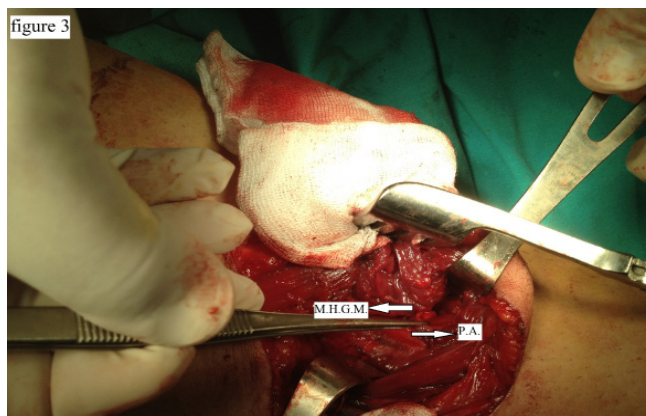


Figure 3: Peroperative Picture of resected Medial head of gastrocnemius and popliteal artery

to 1.24 on the right leg. On postoperative 4th day patient discharged uneventfully. 3 month later patient left side of lower extremity was operated. Follow-up one and six month after operations patient have no pain or coldness with any of movement.

DISCUSSION

PAES was first described in 1879, with a reported incidence of 3.5%.² In 1925, Chambardel-Dubreuil described a case in which the popliteal artery was separated from the popliteal vein by an accessory gastrocnemius muscle.⁵ Hamming and Vink, in 1959, performed the first operative decompression of an entrapped popliteal artery, at Leyden University in the Netherlands.⁶ The first case diagnosed before surgical intervention was reported by Servello in 1962, at the University of Padua in Italy.⁷ Love and Whelan, of Walter Reed General Hospital in the United States, introduced the term “popliteal artery entrapment syndrome” (PAES) in 1965.⁸

PAES is a partial or complete occlusion of the popliteal artery as a result of aberrant anatomy in the popliteal fossa.⁹ Up to 80% of reported cases have occurred in healthy active men. In over half of the cases, the symptom developed before the age of 30 years.¹⁰ Bilateral involvement has been reported in up to 67% of cases.¹¹

The clinical findings can be obscure.¹² A provocative stress test with the ankle either plantar- or dorsiflexed against resistance may demonstrate the disappearance of the pedal pulses in the involved limb.^{13,14} Baltopoulos et al. report the test being positive in all their five PAES patients.¹⁵ In our case, the disappearance of pedal pulses on the active plantar flexion was not consistent, which may reflect varying degrees of effort by the patient. This may also explain the variable degree of arterial compression seen on

different scans and underlines the importance of stressing to the patient that maximal effort must be used during the performance of dynamic studies.

The presenting symptoms are variable and include leg swelling, aching pain, rest pain, and cramping of the calf. Physical signs are usually absent at rest, until complications develop. In young persons, symptoms are limited to intermittent claudication and this is due to the intermittent compression of the artery during plantar flexion or dorsiflexion.¹⁶

A classification of popliteal vessel entrapment has been described. There are essentially four types of anatomical anomalies causing PAES (types I - IV). Type V includes any of the anatomical variants in types I - IV but also involves the popliteal vein. Type VI is a functional PAES caused by hypertrophied muscle.¹⁷ The physical examination may be completely normal if certain provocative manoeuvres and exercise are not included. To elicit a pulse deficit the knee is hyperextended and the foot placed in forced plantar flexion. Pulse loss during these manoeuvres is considered pathognomonic although pulse reduction can occur in individuals without any abnormalities.¹⁸ Chronic extrinsic arterial compression leads to vascular microtrauma, early arteriosclerosis, and subsequent thrombosis.¹⁹ Thrombus formation, in the later stages of PAES, may cause complete obstruction of the popliteal artery leading to acute limb-threatening ischaemia; this is commonly seen in patients who have not developed sufficient collateral circulation.²⁰ Hence, this condition needs to be included in the list of diagnoses in young patients who present with symptoms of peripheral vascular disease. Angiography is the classical screening and diagnostic tool in PAES.²¹ It can demonstrate compression of the popliteal artery with the ankle plantar flexed.¹⁶ pointed out that an irregularity of the wall of the popliteal artery (in an otherwise normal arterial tree) should also raise suspicion of PAES. Transverse T1-weighted MRI is useful to evaluate the aberrant muscular anatomy of the popliteal fossa and also to demonstrate the deviation of the popliteal artery.¹⁹ The most common and successful treatment option is muscle division to release the entrapment. If there is significant delay in diagnosis, the popliteal artery may become occluded, stenotic, or aneurysmal.²² In such complicated cases, division of the anomalous musculotendinous structure and vascular reconstruction is generally required.

CONCLUSION

PAES is difficult to diagnose and, therefore, poses a diagnostic pitfall. Young patients with ischemic symptoms

must be considered PAES. Like our case, history and physical examination is very important and give more details about PAES. MRI and angiography are the most important diagnostic tools for PAES. It should be included other reasons that acute popliteal artery occlusion or claudication in young patients with no cardiovascular risk factors. Early surgery for relax the popliteal artery is the treatment of choice and is important for outcome.

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