Popliteal Artery Entrapment Syndrome

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Abstract

Ms X, a 22-year-old lady, was referred to the vascular unit in view of a long-standing history of bilateral exertional calf pain. Her pain-free walking distance was roughly 100 metres. Stopping to rest alleviated the pain within 5 minutes, allowing her to walk another 100 metres pain-free. The patient reported an insidious onset of these symptoms in 2016, with a gradual increase in time of onset from exertion over the next 2 years, to the current state she presented with, having remained stable since. On examination, the pedal pulses were palpable and on duplex ultrasound scanning (DUS), she had normal triphasic waveforms bilaterally in the distal arteries. There was no significant change in the waveforms on plantarflexion. However, a dynamic MRI-angiography (MRA) of the legs confirmed the diagnosis of a rare condition called popliteal artery entrapment syndrome (PAES). DUS was repeated with the addition of forced plantarflexion of the foot and on the right leg, triphasic waveforms changed to monophasic continuous with this manoeuvre. In view of this, Ms X underwent surgical exploration of the right popliteal fossa to release the popliteal artery from its entrapment.

Keywords: Popliteal artery; Popliteal artery entrapment syndrome

Fact file on PAES

PAES is a rare vascular disorder that can cause intermittent claudication (IC) in athletic young people without cardiovascular risk factors (1). Since it is rare and the symptoms it produces overlap with other, commoner disorders, diagnosing PAES can be challenging and it is not uncommon for patients to present with a long-standing history coupled with multiple unfruitful consultations (2). The overall incidence of PAES is believed to range from 0.17% to 3.50% within the general population (2), but this may not reflect the true prevalence due to mis- and undiagnosed cases (1). Active young males are predominantly affected (85%) and up to 30% of patients present with bilateral PAES (3).

Vascular compromise, often due to an underlying congenital anomaly within the popliteal fossa, results in an insidious and progressive onset of exercise intolerance. The patient is usually an athlete or active person in their 2nd-3rd decade of life (3), however, cases of patients as young as 7 years old have been reported (4). Patients often complain of reproducible calf pain that resolves quickly on cessation of exercise. The pain is typically in the back of the calf but if the anterior tibial artery is involved, it can also occur laterally or in the anterior leg (5). The pain and claudication distance vary for each patient and can range from mild enough that 'only' quitting their sport allows them to lead an otherwise

normal life (6), to severely disabling with a claudication distance of just a few metres. Apart from exertional pain, patients may also complain of pallor, coolness, paraesthesia, extremity numbness, foot pain, lower limb swelling and a muscular ache that persists for hours after cessation of exercise (7). Examination of a patient with PAES may reveal tight, hypertrophied calf muscles and absent foot pulses when the patient has been asked to walk a distance that normally reproduces their symptoms (8).

There are 6 subtypes of PAES, as demonstrated in Table 1. Types I–V are due to anatomical (congenital) causes and type VI describes acquired or functional cases in which the anatomy is normal. Symptomatic onset in both functional and anatomical PAES is thought to be related to a change in use of the gastrocnemius muscle, for example the commencement of athletic training, which results in muscular hypertrophy and overcrowding of the popliteal fossa. In functional PAES (fPAES), the muscular hypertrophy causes symptomatic compression of the popliteal artery with impaired blood flow during exertion. This results in repeated microtrauma of the muscles and subsequent development of constricting scar or fibrous tissue around the artery (9). Similarly, in anatomical PAES, muscular hypertrophy coupled with a pre-existing anatomical aberration, results in clinically significant and symptomatic impingement of the artery (10).

Diagnosis of PAES poses a challenge as there is no clear-cut consensus regarding the diagnostic workup (11). Ankle brachial pressure indexes (calculated by dividing the systolic blood pressure at the ankle by the systolic blood pressure at the arm) are often found to be less than the normal value of 0.9 in patients with anatomical PAES, but a false negative will likely be obtained if a patient has fPAES.

Table 1: An overview of the 6 popliteal artery entrapment syndrome subtypes (1).

Type I	An aberrant medial course of the popliteal artery around the normally-positioned medial head of gastrocnemius (MHG)
Type II	MHG attaches abnormally and more laterally on the femur causing the popliteal artery to pass medially and inferiorly
Type III	Abnormal fibrous band or accessory muscle arising from the medial or lateral condyle encircling the popliteal artery
Type IV	Popliteal artery lying in its primitive deep or axial position within the fossa, becoming compromised by the popliteus muscle or fibrous bands
Type V	The entrapment of both the popliteal artery and vein due to any of the causes mentioned above
Type VI or F	Muscular hypertrophy, resulting in a functional compression of both the popliteal artery and vein

Similar problems are encountered when using DUS on patients with fPAES, since at rest the artery is patent with normal waveforms. Occlusion, and therefore a significant change in waveform, only occurs during exertion and resolves soon after. Provocative protocols with active plantarflexion reduce the rate of false negatives but since not all patients with fPAES occlude in this position, there is still a risk of false negatives (12).

Magnetic resonance angiography (MRA) is a valuable modality in the diagnosis of PAES and should be considered after positive ultrasound studies to confirm the type of lesion, or negative studies with a remaining index of suspicion (11).

Untreated, PAES is potentially limb-threatening as it can lead to popliteal artery stenosis, thrombosis, distal arterial thromboembolism and in extreme cases, limb amputation (1, 13). After diagnosis, surgical correction of the anatomical aberration in PAES types I–V is therefore

always recommended. This involves surgical exploration of the popliteal fossa through a medial or posterior approach with myotomy of accessory slips, excision of occlusive fibrous bands or detachment of muscle. Within a few months from surgery, over 90% of patients report complete resolution of symptoms and are able to resume their sports or training (14).

Surgical treatment of fPAES remains more controversial since there is no clear anatomical entrapment that can be fixed but should be considered in patients with significant symptoms. Surgical outcome of fPAES is not as favourable as that of anatomical PAES, with only 77% of patients reporting resolution of symptoms after surgery (15). Guided botulinum toxin (Botox) injections appear to help some patients by causing localised muscle atrophy of the compressing gastrocnemius muscle.

Case report

Presenting complaint

A 22-year-old female patient complains of a 6-year history of bilateral calf pain brought on by walking, that is worse on the right, and is relieved by rest.

History of presenting complaint

The nature of the pain is a burning, fatigued sensation in the muscles that starts off on the posterolateral aspect of each leg and radiates to encompass the calf shortly after. The distance walked before onset of symptoms is remarkably constant at about 100 metres on the right and 150 metres on the left. This distance is reduced if the patient walks quicker than normal or at an incline. Stopping to rest alleviates the pain within 5 minutes, allowing her to walk the same distances before renewed onset of symptoms. If the patient does not stop to rest but keeps on walking through the pain, she experiences paraesthesia in her right toes and limping.

Ms X dates the onset of these symptoms to roughly 6 years earlier. Her earliest recollection of this pain is a cramping sensation in her calves at the end of 90-minute-long football training sessions. Over the course of 2 years, the walking distance regressed, and pain levels increased, forcing her to give up sports. The symptoms have remained constant since 2018.

Previous medical/surgical history

Ms X suffers from Hashimoto's thyroiditis since she was 8 years old. When she was 15, she was diagnosed with polycystic ovarian syndrome. At 18, Ms X was admitted to the psychiatric ward for suicidal plans after suffering for years from undiagnosed depression.

Drug history

The patient has no known drug allergies. Her drug history is listed in table 2.

Family history

Her father suffers from type 2 diabetes mellitus and hypertension. There is a history of Hashimoto’s thyroiditis and coeliac disease in both first- and second-degree relatives.

Social history

The patient is a non-smoker and does not drink alcohol or use illicit drugs. She lives at home with her family and is currently a university student. The symptoms interfere with daily life and she gives the following examples: going to the local pharmacy to buy her medications or crossing the university campus from one lecture to another cause pain.

Table 2: Ms X's drug history.

Drug	Dose	Frequency	Formulation	Reason for prescription
Thyroxine	100 micrograms	Once daily	Oral tablet	Thyroid replacement therapy
Escitalopram	20 mg	Once daily	Oral tablet	Major depressive disorder
Trazodone	100 mg	Once daily	Oral tablet	Major depressive disorder
Metformin	500 mg	3 times daily	Oral tablet	Polycystic ovarian syndrome

Systemic enquiry

Nil to note.

Differential diagnosis

1. Intermittent claudication:
    - This is typically secondary to peripheral arterial disease but due to the patient's age and lack of cardiovascular risk factors, unlikely to be the aetiology of the symptoms.
    - Rarer causes of IC include PAES and cystic adventitial disease of the popliteal artery. However, IC typically resolves within 1-2 minutes of ceasing exercise. In this patient's experience, it takes less than 5 minutes but more than 1-2 minutes.
2. Chronic exertional compartment syndrome (CECS):
    - This occurs in susceptible individuals when during exertion, an increase in muscle volume within a rigid fascial compartment causes a marked raise in intra-compartmental pressure. This results in exertional muscular pain (16). CECS most commonly affects the anterior compartment of the lower limb (17).
    - Symptoms are similar to those of PAES, with two exceptions. Firstly, peroneal nerve dysfunction may occur in CECS, which is not typical of PAES. Secondly, after exertion brings on the pain, longer periods of rest (sometimes 30 minutes) are required to alleviate it in CECS. However, recent case reports show that a significant number of patients with CECS also have PAES, and so in practice these differences may not be so relevant (18, 19).
3. Neurogenic claudication:
    - This is caused by lumbar spinal stenosis. Symptoms include pain, weakness and paraesthesia that extend into one or both legs and/or lower back. These occur with walking, standing or extension of the back.
    - However, patients with neurogenic claudication typically report an improvement in symptoms when walking uphill or flexing their spine (20), neither of which apply to this patient.

Physical examination and preliminary investigations

On examination, the lower limbs had no stigmata of peripheral vascular disease. They were symmetrically warm with normal capillary refill time. The pulses were palpable throughout both lower limbs.

Preliminary investigations included DUS. This ruled out cystic changes in the popliteal artery and revealed triphasic waveforms, both at rest and with plantarflexion, in the distal arteries and popliteal artery of both limbs.

Diagnostic investigations

1. MRI of the whole spine. This test was ordered to exclude potential underlying pathologies such as lumbar spinal stenosis and spinal disc herniation.
2. MRA of the legs with and without manoeuvres. This test involves two sets of imaging; one at rest and another after the patient performs provocative movements until the symptoms are produced. MRA was chosen as a first-line investigation since it is non-invasive, unlike angiography.

Findings and Diagnosis

1. The MRI of the whole spine showed no pathologies or abnormalities.
2. The MRA images obtained after performing provocative manoeuvres showed evidence of PAES at the level of the knee in both legs (Figure 1). As no apparent anatomical cause was observed for this entrapment, a diagnosis of fPAES was made.
3. Following the MRA, DUS was repeated with the addition of forced plantarflexion of the foot. On the left, no changes were apparent; however, on the right, waveforms changed dramatically from triphasic to

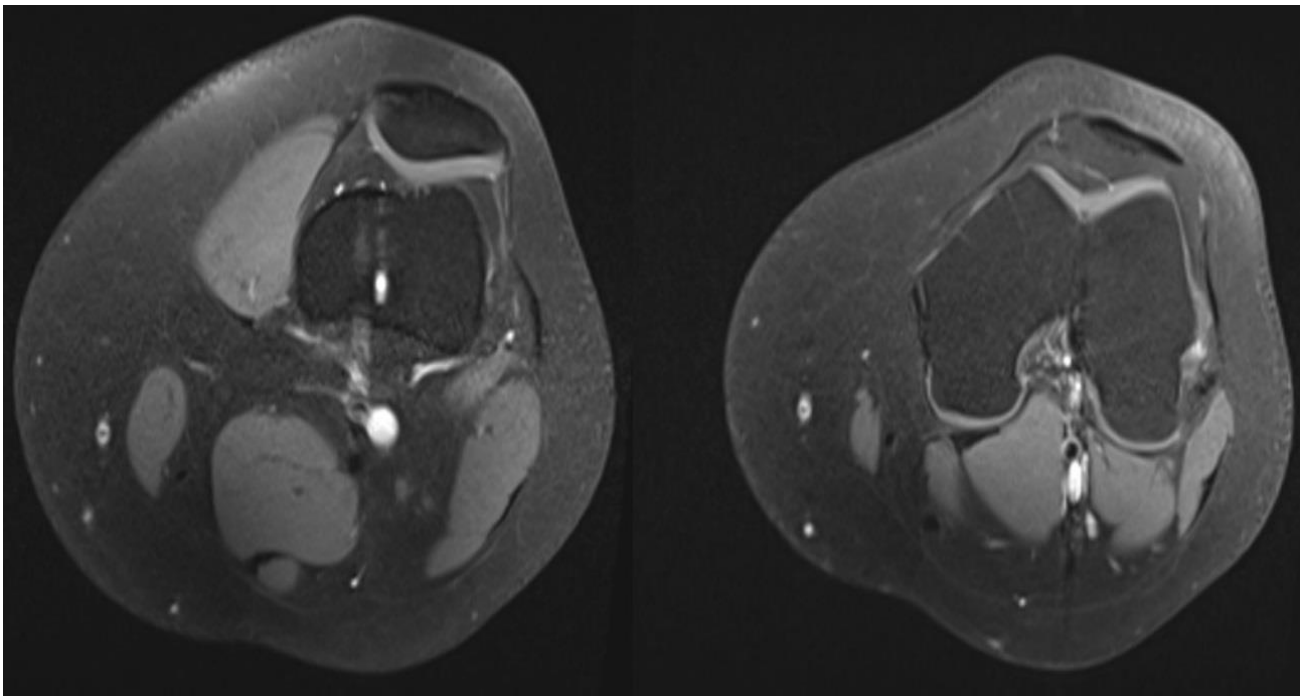

Figure 1: Consecutive sectional images of the patient's left knee. The image on the left shows the popliteal artery (white) prior to entrapment; note its round shape. On the right, the popliteal artery is entrapped between the medial and lateral heads of the gastrocnemius muscle at the level of the femoral condyles; note its elliptical shape. Further down the leg, the artery resumes its round cross-sectional shape.

monophasic continuous with this manoeuvre.

4. Concomitant CECS complicating the diagnosis of fPAES could not be ruled out due to a lack of reliable diagnostic modality.

Management

After discussing the diagnosis with the patient, it was decided to manage fPAES alone initially, with the possibility of revisiting CECS as a potential diagnosis and treatment option in the future in case the symptoms persisted.

Surgical exploration of the right popliteal fossa was carried out with the patient under general anaesthesia. Intraoperatively, the entrapment seen on MRA was confirmed again by the bottleneck appearance of the popliteal artery. The artery was successfully dissected free from a fibrous web constricting it into this shape and myotomy of the MHG was performed to create more space in the fossa.

The patient was encouraged to mobilise a few hours after surgery and happily reported no symptoms on the right when doing so. She was discharged the following day to continue recovery at home.

Follow-up

Unfortunately, during a post-operative follow-up, Ms X reported that her symptoms had begun to return approximately three weeks after surgery. Despite this development, DUS revealed triphasic waveforms in the distal arteries during forced plantarflexion; a significant improvement compared to the pre-surgery monophasic waveforms. It was thus deemed likely that the patient was also suffering from CECS, and a referral to the orthopaedic team for further management was made.

Declarations

Conflict of interest: N.A.
Ethical statement: Consent for publication from Ms X was obtained.

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