CASE REPORT

Intermittent foot claudication caused by a dynamic compression of the posterior tibial artery

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SUMMARY

A young adult presented with an atypical intermittent foot claudication caused by a dynamic compression of the posterior tibial artery by a ganglion. This case highlights the diagnostic challenges when dealing with an entrapment syndrome. Subsequent open surgical treatment was successful, and the patient has made a good recovery.

BACKGROUND

Intermittent claudication defined as muscle pain precipitated by walking and rapidly relieved by rest is frequent in the general population above 65 years.¹ However, intermittent foot claudication in a young and healthy adult is very rare. A review of the literature revealed only three case reports describing foot claudication due to an entrapment of the dorsalis pedis artery (DPA) by the extensor hallucis brevis and by a fibrous tissue band.^{2–4} To the best of our knowledge, this is the first case of a foot intermittent claudication that was caused by compression of the posterior tibial artery (PTA) by a ganglion.

CASE PRESENTATION

A 43-year-old male patient presented with the right forefoot numbness and an intermittent claudication at 250 yards. Medical history included only factor V Leiden mutation, which did not require any form of anticoagulation. He was a non-smoker. On examination, the patient had a full complement of infrainguinal pulses except for reduced pulse in the DPA on the right. The right forefoot became pale and cold on active, forced dorsiflexion.

INVESTIGATIONS

Handheld Doppler revealed a triphasic signal in the DPA that became damped on dorsiflexion. Bilateral resting and exercise Ankle Brachial Pressure Index (ABPI) was normal. Digital plethysmography (DP) revealed an abnormal pulse wave contour on the right in a neutral position when compared to the left. On dorsiflexion, the right pulse wave contour was obliterated ([figure 1](#)). Dynamic duplex revealed normal flow within the right anterior tibial artery (ATA) in a neutral position, and then a complete cessation of flow in dorsiflexion. DPA was occluded at the origin, but there was a large collateral vessel taking off just above the level of the occlusion (distal ATA) into the forefoot, where DPA was patent. Additionally, a complete occlusion of the PTA on dynamic dorsiflexion was demonstrated. On plantar flexion, the PTA returned to a normal

calibre. These unusual findings prompted a CT angiogram (CTA), which was unremarkable. Subsequently, dynamic angiography showed an occluded DPA from its origin to the level of the talonavicular joint. In effect, DSA confirmed reduced flow on dorsiflexion in the ATA via the aforementioned collateral vessel and no flow within the DPA. Flow in the PTA was best in plantar flexion and ceased in dorsiflexion ([figure 2](#)).

TREATMENT

The above findings were indicative of an arterial entrapment syndrome and an orthopaedic review excluded other potential causes such as chronic exertional compartment syndrome (CECS) and tarsal tunnel syndrome (TTS). However, in view of the altered PTA flow a combined orthopaedic and vascular PTA exploration was performed. This demonstrated a small ganglion arising from the medial aspect of the subtalar joint immediately adjacent to the PTA ([figure 3](#)). The ganglion was excised, and synergistic release of the flexor retinaculum was also performed. The limb was placed in a backslab to aid healing.

OUTCOME AND FOLLOW-UP

The operation led to the resolution of the patient's symptoms. After a period of routine follow-up, the patient was discharged.

DISCUSSION

Atypical ischaemic symptoms in young adults are rare and should prompt an urgent clinical evaluation. While DPA entrapment syndrome is described in the literature, there are no reports regarding PTA entrapment as a cause of atypical foot claudication.^{2–4} The differential diagnoses include popliteal artery entrapment syndrome (PAES), cystic arterial disease (CAD), CECS, TTS and various anatomical anomalies causing an external arterial impingement.^{1–4} Consequently, various investigations are used in establishing a correct diagnosis, and these include ABPI, DP and several other imaging modalities.

ABPI is a simple method for detecting peripheral arterial disease (PVD), but in our case the obtained measurement was normal.¹ Therefore, we used another easy and non-invasive tool such as DP. DP has been found to be a reliable and reproducible technique for the assessment of PVD.¹ DP is a secondary output of a pulse oximeter which uses an infrared light transmitted through the skin of a digit to measure the pulse wave velocity (PWV).¹ The PWV represents pulsatile peripheral blood flow that is a standard measure of arterial wall

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Figure 1 Plethysmography: obliterated pulse wave form contour on the right on dorsiflexion.

stiffness. In the presented case an obliterated pulse wave form was demonstrated on the index side confirming the clinical findings.

In our case PAES as well as CAD were excluded by duplex ultrasound scan, CTA and dynamic angiography. The aforementioned imaging modalities demonstrated widely patent popliteal arteries in the normal anatomical position. Another potential diagnosis was CECS, which was excluded on clinical grounds. CECS is caused by a raised pressure within a muscle compartment, which is unable to accommodate an increase in a muscle volume on exertion.⁵ As a result, patients affected by this

condition are asymptomatic at rest.⁵ However, in the presented case the patient remained symptomatic even at rest. TTS is an entrapment neuropathy caused by a compression of the posterior tibial nerve under the flexor retinaculum.⁶ Arguably, TTS could be responsible for the right forefoot numbness as the ganglia can lift, encircle or move onto the tibial nerve on dorsiflexion.⁷ However, arterial compromise is not a normal component of the TTS. In our opinion, the ganglion was dynamically compressing the PTA leading to an obliteration of flow on dorsiflexion ([figure 2](#)). Furthermore, the patient was symptomatic because of the lack of a retrograde flow in the partially occluded

Figure 2 Dorsiflexion: reduced flow in the ATA, no flow in the DPA and PTA (A); plantar flexion: normal flow in the PTA (B). ATA, anterior tibial artery; DPA, dorsalis pedis artery; PTA, posterior tibial artery.

Figure 3 A small ganglion arising from the medial aspect of the subtalar joint. PTA, posterior tibial artery.

DPA from the pedal arch via the PTA. In effect, the plantar arch was incomplete, and PTA was unable to provide sufficient flow to perfuse the foot. Finally, several anatomical anomalies have been reported to cause an external arterial impingement. These include musculoskeletal bands, hypertrophied and accessory muscles.^{2–4} In such circumstances, reported surgical excision of fibrotic bands or extensor hallucis brevis tendon transection with or without transposition can be performed.^{2–4} Whether an additional revascularisation procedure (bypass) is required, needs to be judged on an individual case basis.

Learning points

- Foot intermittent claudication is very rare.
- A multidisciplinary team approach ensures correct diagnosis and optimal treatment.
- If entrapment syndromes are considered and identified early, this can avoid the development of an arterial injury.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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