



QUESTION | MY QUESTION IS RELATED TO COMPARTMENT SYNDROME (CS). I HAVE HAD SEVERAL PATIENTS WHO HAVE HAD CHRONIC DEBILITATING CS CONFIRMED BY STANDARD COMPRESSION TESTS, HAD RESULTANT BILATERAL FASCIOTOMY PROCEDURES PERFORMED ONLY TO HAVE THEIR SYMPTOMS RETURN UPON RETURNING TO SPORT. POPLITEAL ARTERY ENTRAPMENT SYNDROME (PAES) HAS SIMILAR PRESENTING SIGNS AND SYMPTOMS WITH SUBTLE VARIATIONS HOWEVER NONE OF THE ABOVE CASES HAD MRI OR CT ANGIOGRAM (CTA) TO RULE OUT PAES TYPES I, II OR III PRIOR TO THEIR FASCIOTOMY. WHAT ARE THE CLINICAL SIGNS AND SYMPTOMS THAT GUIDE DOCTORS TO PERFORM FASCIOTOMY BEFORE FIRST CLEARING PAES AND WHY IS MRI OR CTA NOT ROUTINELY PERFORMED AS A DIFFERENTIAL DIAGNOSTIC TOOL? WHAT ADVICE CAN YOU GIVE PHYSIOTHERAPISTS IN THE MANAGEMENT OF CONDITIONS THAT CAUSE INTERMITTENT CLAUDICATION IN YOUNG ADULT ATHLETES?

ANSWER: | Exercise related lower leg pain is a common complaint in athletes. It is second only to the knee in overuse injuries to the lower limb in runners, and makes up around 20% of injuries in runners.

By far the most common causes of lower leg pain are tibial periostitis (the old 'shin splints'), chronic exertional compartment syndrome (CECS) and stress fracture. There is a fairly equal spread between these entities and some overlap between them.

CECS is felt to occur as the fascial tissue surrounding the muscle becomes thickened and loses its compliance. When the exercising muscle swells with increased blood volume, then the pressure within the muscle compartment rises, causing a reduction in the blood flow to the muscle. CECS therefore has what would be described as a claudicant presentation – that is an initial pain free period when starting to run, followed by a gradual build up of pain as exercise continues. Often this will cause the patient to have to stop exercising and the pain will slowly improve as the pressure decreases and the blood flow returns.

When considering claudicant pain then the main differential diagnoses are vascular. In the older patient this always includes peripheral vascular disease, especially if there are other risk factors such as diabetes, hypertension, lipid disorders, a family history and the patient smokes.

Another cause of vascularly related claudicant lower leg pain is the popliteal artery entrapment syndrome (PAES). This is actually a very rare condition, making up only 0.16% of all claudicants (1). Entrapment occurs because of an abnormal relationship between the popliteal artery and the myofascial structures as this vessel passes across the knee joint. Ten such anomalies have been described and most authors use a definition that includes the five most common ones. These classifications have no influence on diagnosis, treatment or prognosis. This anatomical entrapment occurs more commonly in young men (85%; mean age 33.5 years), and occurs bilaterally in 25% of cases.

Functional PAES is even rarer and usually occurs in athletes when a hypertrophied gastrocnemius head compresses an anatomically normal popliteal artery against the bone. It occurs in younger, usually female patients and appears to result from neuromuscular irritation as the neurovascular bundle is impinged within the soleal canal. Muscular compression on the popliteal artery from the gastrocnemius may occur repeatedly, causing damage to the vessel wall and premature localized atherosclerosis. This may result in fibrosis, thrombosis, and aneurysm or embolus formation and may go on to occlusion and critical ischemia. Athletes are most commonly affected because of their muscular hypertrophy and an inability to tolerate symptoms during their chosen activity.

On examination distal pulses are absent when tension is placed through gastrocnemius on the affected side. The manoeuvres of ankle plantar flexion (active), dorsiflexion (passive) and knee extension will cause popliteal artery compression. However, it is also positive in some unaffected individuals.

This next section will attempt to answer to the specific concerns raised in this question. As with many conditions in medicine, the precise answer to the problem raised by a patient is not always clear cut. As doctors we try to sort the problem out by taking a good history, performing a specific examination and using investigations as indicated.

There is an axiom in medicine that 'common things occur commonly'. CECS is far more common than PAES and, as such, more likely to be the first diagnosis considered in the young athlete with claudicant lower leg pain. This, however, doesn't rule out PAES as a clinical entity.

Anterior compartment syndrome

Anterior compartment Syndrome should be a straight forward diagnosis. A patient with claudicant pain occurring anterolaterally, a feeling of increased tension or 'woodiness' in the muscle, muscle herniation, or even paraesthesia in the top of the foot will undoubtedly have CECS of the anterior or peroneal compartments. When the pain is in the calf or medial lower leg, then the diagnosis may be less straight forward. Historically PAES will most likely cause claudicant calf pain and there may not be a feeling of muscle tension that comes with CECS.

On examination an assessment of the peripheral pulses is essential. In compartment syndrome there may be little to find clinically, but the finding of muscle hernias in the anterior compartment is usually suggestive of compartment syndrome. As part of the vascular examination the pulses need to be palpated in knee extension with active plantar flexion or passive dorsiflexion, which may cause kinking of the artery and a reduced pulse in PAES. Peripheral pulses should remain normal in CECS. If CECS is considered then even running the patient on a treadmill for 10 minutes should increase the tension in the compartment, giving a tense feeling in the muscle post exercise that slowly resolves with time. In a classic case of anterior CECS the diagnosis can be made post exercise by simple palpation before even putting the pressure needle in.

Investigations:

The pathway to investigation, as in all areas of medicine, will be guided by the history and examination. Generally this will lead to a pre and post exercise compartment pressure test in the young patient with claudicant pain, especially if there are clinical features of CECS. If the symptoms are less clear, more related to the calf muscle or the pressure tests are normal, then vascular tests may be considered. Initially a pre and post exercise Doppler ultrasound study may be useful. It has the advantage of being non-invasive, easily organised, can involve an exercise component and also provocative positioning, such as active plantarflexion of the foot to see if this dynamically narrows the popliteal artery. If this test is normal then it is unlikely that there will be a PAES. It does not differentiate structural from functional PAES. If the Doppler US is abnormal, then further follow-up with an MRI angiogram (MRA) may be required to further define the lesion. This may again be performed at rest and with active plantar flexion. The arrival at a diagnosis in the young claudicant patient may not always be straight forward, but will generally follow this pathway.



Fig 1. Angiographic appearance of PAES

On occasion a patient may, as mentioned, have pressure tests that are abnormal and fasciotomy is unhelpful. Another diagnosis, such as PAES, may need to be considered, or it may be a simple failure of the decompression surgery. The reported success rates for deep posterior compartment release are around 75%, as opposed to 90% for the anterior compartment. This could be because of failure of adequate decompression, fascial re-scarring or incorrect diagnosis. In these cases consideration of repeat compartment pressure testing may be required, although generally vascular tests (exercise dopplers, MRA) will be performed prior to this to rule out another diagnosis. Whilst ultimately it is preferable to explain symptoms with a single diagnosis, on rare occasions there may be more than one pathology present. Potentially the fascial thickening that causes CECS may be in some way related to the fascial bands affecting the popliteal artery in PAES. In addition, the deep posterior compartment is technically more difficult to test than either the anterior or peroneal compartments, which could potentially explain a false positive result. This might explain why compartment release surgery has been unhelpful. As a rule the cause of medial shin and calf pain is not always as well defined as anterolateral pain. If there is any doubt on the diagnosis then full range of investigations may be required prior to contemplating any surgical treatment.

Conclusion:

The most important advice I could give to any young physiotherapist is to consider the symptoms presented in the history and make the diagnosis of claudicant pain. On the whole physiotherapists diagnose CECS much better than GP's, who often don't recognise the causes of claudicant pain in the younger patient. Often the physiotherapist is the first point of call in these patients. Prompt referral to a doctor with expertise in managing lower leg pain in athletes would be suggested, especially if the condition is not improving within 4-6 sessions of manual therapy and rehabilitation exercises. There are only a couple of studies showing a trend towards improvement in CECS with manual therapy alone, and on the whole whilst deep tissue massage may cause some relief of symptoms, it is generally not curative. If historically the pain sounds claudicant (when running, initially no pain, pain worsens with continued exercise, pain resolves rapidly with rest and the patient is generally symptom free outside of exercise) then referral sooner rather than later for diagnostic purposes is essential.

1. The conundrum of claudication. Varcoe RL, Taylor CF, Annett P, Jacobsen EE, McMullin G., ANZ J Surg. 2006 Oct;76(10):916-27.

Dr Paul Annett