Acute compartment syndrome with an atypical presentation: a useful clinical lesson

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A high index of suspicion for compartment syndrome must be maintained, even if all diagnostic criteria are not met.

Introduction

Acute compartment syndrome has been reported following a number of insults to the limb causing vascular, metabolic or other mechanical disruption within the compartment.¹ This leads to an increase in the tissue pressure within the compartment, with subsequent reduction in tissue perfusion and eventually tissue hypoxia/necrosis within the compartment affected.

It is frequently seen in young patients, with the highest incidence in men under the age of 35 years.¹ The outcome from compartment syndrome has been shown to be worse in those patients where diagnosis is delayed.² The potential sequelae of missed diagnosis include muscle necrosis, infection and progression on to contracture and possible limb amputation.

The diagnosis is reliant on a high index of suspicion. The diagnostic criteria include: (1) pain out of proportion to the initial injury; (2) pain on passive stretch of the muscles within the affected compartment; (3) palpably tense compartment; and (4) weakness and parasthesia of the areas supplied by nerves crossing the compartment. Loss of pulses is considered a late sign due to arterial occlusion.³

In equivocal cases and where available, compartment pressures can be measured. A compartment pressure of >35 mmHg or within 30 mmHg of diastolic pressure being suggested as indicative of compartment syndrome.⁴

This report considers the diagnostic criteria for compartment syndrome. It reflects the fact that pain-based assessment criteria cannot be absolutely relied upon when the presentation is atypical.

Case report

A 39-year-old man presented to the Accident & Emergency Department (A&E) following a crush injury to his left lower leg. His left lower leg was crushed between the bumpers of two cars, one stationary and the other slowly rolling forwards. He was initially able to partially bear weight, but was unable to by the time of his arrival in A&E two hours later.

He had no significant past medical history, was a non-smoker, and was employed as an office worker.

Initial A&E assessment revealed bruising and tenderness over the proximal tibia. The patient was reported as having no neurovascular deficit on arrival. Emergency management involved oral analgesia (Co-codamol; 1 g paracetamol/60 mg codeine phosphate).

AP and lateral radiographs of the tibia and fibula demonstrated a minimally displaced fracture of the fibula head (Figures 1 and 2).

On repeat assessment, the patient was noted to have developed absent sensation over the first dorsal web space with a ‘foot drop’ (foot dorsiflexion and hallux extension MRC power grade 0/5). Sensation and power in the left lower leg was otherwise intact.

His anterior compartment was noted at re-assessment to be very firm to palpation, with the lateral and posterior compartments palpably soft. He was not complaining of any significant pain at this point despite limited analgesia, and passive stretch of the extensor hallucis longus did not cause significant exacerbation of the pain. Dorsalis pedis and posterior tibial pulses were palpable and of normal volume.
Compartment pressure monitoring was not performed because it was not readily available and would have delayed surgery.

Informed consent was gained and the patient was taken to the operating theatre as a matter of urgency (within 4 hours of presentation). The procedure was performed with the aid of a tourniquet inflated to 300 mmHg. The anterior compartment was found to be clinically under very high pressure and only this compartment was surgically decompressed because the lateral and two posterior compartments felt soft.

On release of the tourniquet, tibialis anterior was seen to be bleeding but was rather ‘sluggish’ on direct stimulation of the muscle.

Within 20 min of the completion of surgery the patient had regained some sensation in the first dorsal web space and power of one-fifth of ankle dorsiflexion. Power of ankle and great toe dorsiflexion improved to four-fifths over the following days with rapid full return of first web space sensation. The patient returned to the theatre for a second look and attempted delayed primary closure at 72 h, but complete closure was not possible. The patient was referred to the plastic surgeons and split-skin grafting was subsequently performed for the residual skin defect.

Discussion

The annual incidence for compartment syndrome has been reported as 7.3 per 100,000 for men and 0.7 per 100,000 for women. Fractures of long bones are often associated with the development of compartment syndrome, although 23.2% of occurrences are associated with soft tissue injury only.\(^1\)

In this case, the patient presented with signs of isolated deep peroneal nerve palsy associated with an anterior compartment under extreme pressure, but without pain being a major feature. The classic test of passive stretch of tendons within the involved compartment was uncomfortable to the patient, but not severely painful, despite minimal analgesia. The patient had a good dorsalis pedis pulse. Compartment pressure monitoring was not readily available and would have delayed surgery.

At operation the anterior compartment was found to be clinically under high pressure and
postoperative rapid nerve recovery supported the decision to operate urgently and to selectively decompress only the anterior compartment. For compartment syndrome of the lower leg, four compartment fasciotomies have generally been considered the gold standard. This however would have resulted in far greater soft tissue insult for what clearly an isolated anterior compartment syndrome.

This case highlights the diagnostic challenge of acute compartment syndrome for more junior doctors, who are often the first to assess the patient. The criteria of pain out of all proportion to injury and pain on passive stretch within the compartment cannot always be relied upon.

There have been a number of cases reported in which the diagnosis of compartment syndrome has been complicated or delayed by the reduced pain response of the patient. This has been reported either with the use of peripheral nerve blocks, epidural anaesthesia or use of patient controlled analgesia. In this patient, the reduced pain response was in the absence of use of regional anaesthesia or strong analgesia, providing the diagnostic challenge.

One must always maintain a high index of suspicion for compartment syndrome even if the documented criteria by which diagnosis is made, are not all met. More junior doctors must look to refer early to senior colleagues in such circumstances, to prevent potentially disastrous consequences.

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