



Presentation of compartment syndrome without an obvious cause can delay treatment. A case report

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Compartment syndrome is a serious condition which leads to chronic morbidity unless an urgent decompression of the affected area is performed. An increased intra compartmental pressure commonly occurs after a physical insult though rarer causes have been identified.

We report an atypical presentation of compartment syndrome and subsequent delayed intervention where there was no identifiable aetiological factor. Frontline medical staff must rule out compartment syndrome early so that complications secondary to compartment syndrome can be avoided.

INTRODUCTION

Increased intracompartmental pressure decreases blood supply to muscles eventually leading to necrosis and long term morbidity unless an urgent fasciotomy is performed. Trauma is the commonest cause of compartment syndrome, but other rare aetiologies have been identified.

We report the case of a man having compartment syndrome but being referred as a peroneal nerve palsy after spending four hours in the casualty waiting room. This man had an atypical presentation and a cause for his pathology has not been identified.

CASE REPORT

A 40-year-old caucasian male architect with no previous medical problems presented to the emergency department with an acute history of pain in his left lower leg, associated with weakness in the

toes and altered sensation in the lower leg and dorsum of the foot. The symptoms had first been noticed when he had woken up from a normal sleep the previous morning. Over the subsequent twenty-four hours, the leg pain had become steadily worse. There was no history of trauma, strenuous exercise and consumption of any medication or recreational substance which could have altered alertness.

On inspection, he was in pain and walked with an antalgic gait. On examination of his lower leg, the anterior compartment of the tibia was tender on palpation and was noted to be "woody hard". There were no visible signs suggesting prolonged compression of the leg. Vascular examination was unremarkable. Neurological examination revealed there was altered sensation in the L4 and L5 dermatomes and extension power of the first, second and third toes was reduced (MRC grade 3/5). A diagnosis of acute compartment syndrome was made and this was confirmed by measurement of his compartment pressures. The pressure was grossly elevated in his anterior compartment and measured 114 mm Hg.

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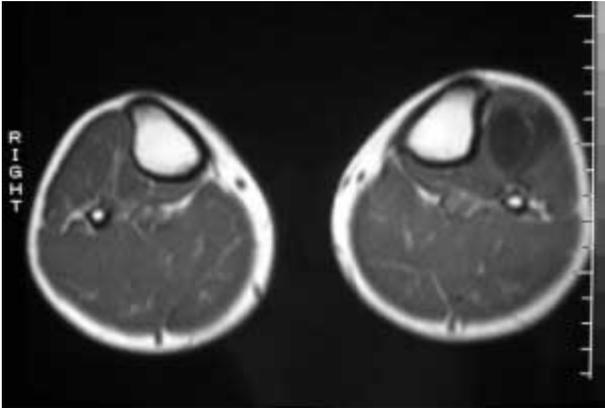


Fig. 1. — MRI showing collection in anterior compartment

Because of the unusual history, we wished to exclude the possibility of a vascular anomaly prior to decompression. An urgent MRI scan, obtained within a few hours of admission to the accident department, revealed a collection within the anterior compartment (fig 1) and a normal vascular tree. A prompt fasciotomy was performed. Intra-operative findings were the presence of a pink amorphous substance and no muscle necrosis. Histology was non-specific and microbiological culture was negative. The wound was inspected 48 hours later, and was closed primarily. At follow-up in the clinic a month later, the patient was pain free with improved sensation and toe extension power had recovered to MRC grade 4 out of 5.

DISCUSSION

Due to the various causes of lower leg pain, compartment syndrome can easily be overlooked if there is no obvious aetiological factor in the history (4). Acute compartment syndrome is a surgical emergency with calls for prompt surgical fasciotomy, if irreversible muscle necrosis is to be avoided.

There are numerous rare causes of compartment syndrome other than trauma but very few cases are totally idiopathic (1). Muscle infarction secondary to microangiopathic disease in diabetes has been reported to cause compartment syndrome (2). Effort-related chronic compartment syndrome is well documented and is still underdiagnosed (3). In our case there was absolutely no history of any physical insult (commonest cause). Blood results were negative, which ruled out coagulopathies, acute infection, and diabetes. The histology was non-specific with no evidence of microangiopathic disease, the negative cultures ruling out any form of infection. A normal vascular tree and no tumours were seen on CT scan. In view of the above it is impossible to elaborate on a specific cause for the problem in our patient.

This case was interesting because we were unable to identify any aetiological factors which could have resulted in an acute compartment syndrome. The collection in the anterior compartment seems to have resulted from the increased pressure rather than initiating it.

We believe that the important message in this case is that in acute compartment syndrome, there is not always an obvious cause. The diagnosis of a compartment syndrome should therefore not be discarded simply because no aetiological factor has been identified.

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