

Peroneal compartment syndrome of non-traumatic origin: A case report

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ABSTRACT

A patient with acute peroneal compartment syndrome is presented. This case is unusual because the pathology was localised to the peroneal compartment only and because trauma was not an aetiological factor. Acute and chronic compartment syndromes are discussed and differentiated, and the importance of a high index of suspicion in all cases is emphasised.

Key words: *compartment syndrome, peroneal, nontraumatic*

INTRODUCTION

Acute compartment syndrome with its attendant risk of irreversible muscle and nerve necrosis has been described most frequently in association with trauma, usually following direct trauma, occurring after open or closed fractures,¹⁶ crushing injuries,¹² ischaemia-reperfusion episodes,¹⁸ or after prolonged localised pressure in comatose patients.²⁷ Non-traumatic acute compartment syndrome has been associated with undue exertion,¹¹ hypothyroidism,²⁵ virus-induced myositis,¹⁷ bleeding diatheses,⁴ leukaemic infiltration,²⁶ nephrosis,²⁴ and ganglion cysts of the proximal tibio-fibular joint.²⁸

Compartment syndrome occurs most commonly in the lower leg but has also been reported in the posterior thigh compartment¹⁹ and the tensor fascia latae muscle.²⁴ It has been reported in the forearm following fractures,¹⁴ overuse,¹⁰ and avulsion of the origin of the flexor digitorum superficialis muscle.⁷ It has also been reported in the upper arm following a direct blow.⁹

Reports of acute compartment syndrome affecting either single or multiple compartments in the lower leg have been described for all four lower limb compartments.^{2,18,19} Peroneal compartment syndrome has been described following rupture of the peroneus longus muscle³ and as a rare occurrence following exertion.⁵ The other lower leg compartments, particular anterior are more frequently implicated.⁸ This case is presented as a rare occurrence of isolated acute peroneal compartment syndrome without trauma as the precipitant.

CASE REPORT

An eighteen year-old shop assistant and keen amateur rugby league player experienced a dull ache in the anterolateral area of his right leg while warming up for a rugby league match in May 1999. He thought it may have been a muscle cramp and performed some stretches before the game. He had no previous history of leg pain or exercise. The pain gradually increased during the game but did not prevent him from

completing the match. The pain continued to worsen over the next four hours until he presented to the Emergency Department with severe right lateral leg pain.

On examination he was unable to bear weight on his right leg due to pain. The foot was warm with strong pulses and good capillary return. He had marked tenderness on palpation of the lateral compartment and the underlying musculature was noted to be tense and swollen. Active and passive plantarflexion and dorsiflexion of the ankle and toes, and inversion and eversion of the ankle was possible although markedly decreased. Any active or passive movement was extremely painful. Sensation over the distribution of the superficial peroneal nerve was decreased to pin prick, although sensation in the deep peroneal nerve distribution was unaffected.

X-rays of the tibia as well as electrolytes and creatine kinase (CK) levels were performed. The CK level was elevated at 1663 IU/L (normal 40–300). Other tests were normal.

The same evening, 9 hours after the initial onset of pain and 4 hours after the pain became severe, he was transferred to the operating theatre and underwent a decompressive fasciotomy of his right peroneal compartment. The anterior compartment was not affected clinically and did not require decompression. Fasciotomy was performed through proximal and distal 5 cm longitudinal anterolateral incisions. The peroneal musculature bulged impressively through the fascial incision but was otherwise pink and contractile. Free blood and bruising were noted in the compartment but the muscle did not require debridement. The fascia was left open; the skin was closed without difficulty with a suction drain in situ. Postoperatively, pain relief was immediate and complete, and passive and active foot and ankle exercises could be performed comfortably. Sensation over the superficial peroneal nerve distribution remained altered but returned to normal over two weeks. Power in all muscle groups was normal. Magnetic Resonance Imaging (MRI) was performed at two weeks and the peroneal musculature demonstrated changes consistent with oedema.

He resumed full activities including a return to football at six weeks without limitation. Renal function and CK levels were not measured postoperatively but urinary function was normal and has remained so.

DISCUSSION

Acute compartment syndrome is of particular concern because the diagnosis must be made essentially on

clinical grounds and must be acted upon promptly if serious and potentially irreversible injury to the relevant compartment is to be avoided. The literature differentiates quite clearly between acute and chronic compartment syndromes. In chronic compartment syndrome the patient reports pain, cramps and muscle tightness with exertion. The pain appears to arise fairly reliably after a threshold is reached and symptoms usually resolve within minutes of the cessation of activity. Clinical findings are minimal, with slight tenderness and swelling over the affected compartment being the classical findings.¹⁵ Intracompartmental pressure measurements have been used although criteria have varied regarding the accepted most useful diagnostic readings.²² Rorabeck et al. recommended a pressure reading greater than 15 mm Hg occurring 15 minutes after exercise combined with a supportive history as diagnostic.²² MRI has been studied whereby T₁ and T₂ parameters immediately after exercise have been shown to correlate well with raised pressure studies of greater than 15 mm Hg.²

Acute compartment differs in that it is progressive and irreversible unless surgical decompression is performed. The classical clinical findings are pain on palpation of the affected compartment, tense and swollen compartments, and pain on passive stretch and attempted active movements. These findings progress to neurological changes and vascular findings if the diagnosis is delayed. The role of pressure readings has been investigated, and continued interstitial pressures of greater than 15 mm Hg are to be regarded as dangerous.¹¹ Continuous tissue pressures of greater than 30–40 mm Hg lead to irreversible nerve and muscle damage after 6–12 hours.¹³ Other authors have described 30 mm Hg less than the patient's diastolic blood pressure as diagnostic.⁶

A currently unanswered question is what causes a progression from the chronic and hence reversible compartment syndrome to the acute and irreversible process requiring surgical decompression. Interestingly, Reneman found 17% of patients with the acute form had a history of pain suggestive of previous compartment syndrome.²⁰ Another unanswered question is why certain compartments are more susceptible to compartment syndrome than others. Literature reports suggest that the anterior compartment of the lower leg is the most susceptible and that this may be because the fibrous and osseous boundaries are far more defined and rigid than in the other compartments.⁸ Ger et al. goes so far as to state "it is something of a rarity that any compartment other than the anterior undergoes strangulation".⁸

The case presented here is of interest for two reasons. Firstly, it demonstrates that the clinician must not dismiss the possibility of an acute compartment syndrome in the absence of trauma. Secondly, it demonstrates that while certain compartments may be more susceptible to developing acute changes, a high index of suspicion must be entertained with any

presentation of acute muscle pain referable to an anatomical compartment. Unfortunately literature reports abound with details of the sequelae when acute compartment syndrome is undiagnosed, and it is our hope that this case may help to raise the awareness of what is essentially a clinical diagnosis requiring prompt intervention.

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