SHORT REPORT

Posterior compartment syndrome associated with clopidogrel therapy following trivial trauma

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Haematomata caused by blunt trauma may potentially induce a compartment syndrome by raising intra-compartmental pressure. We report a case of acute posterior compartment syndrome following minimal trauma to the leg of an elderly patient on the antiplatelet agent clopidogrel. This case highlights the high index of clinical suspicion required to detect compartment syndrome in those on long term antiplatelet therapy and prompt surgical decompression is recommended.

Compartment syndromes are usually associated with fractures, closed soft tissue injuries, revascularisation procedures, and crush injuries.¹ Anticoagulant related haematoma formation has also been reported as a cause of compartment syndrome.² It has been suggested that anticoagulation may even be a risk factor in the development of acute compartment syndromes.³ Antiplatelet therapy has not previously been associated with increased risk of compartment syndrome although aspirin and clopidogrel are increasingly being prescribed for the secondary prevention of atherosclerotic vascular events.⁴ We present the case of an elderly patient on long term clopidogrel therapy, with acute superficial and deep posterior compartment syndrome following minimal trauma to the leg.

CASE REPORT

An 80 year old man presented to the emergency department following a fall in his garden. He had sustained a minor injury to his left calf, but within an hour he experienced acute, severe, and progressive pain associated with significant swelling of his left lower limb. His background history revealed ischaemic heart disease, peripheral vascular disease, and an inoperable abdominal aortic aneurysm. He had been treated with the anti-platelet agent clopidogrel (75 mg once daily) for 2 years prior to this event.

On examination, his left calf was tense and swollen and he complained of exquisite calf pain on passive dorsiflexion of his left foot. His distal pulses were diminished but present, consistent with his arteriopathy history, and his neurological examination revealed no abnormalities. Radiographic examination of the affected limb was normal. The clinical diagnosis of compartment syndrome was confirmed by intra-compartmental pressure measurements. Pressure in the superficial posterior compartment measured 87 mm Hg with a diastolic blood pressure of 66 mm Hg, giving a critical closing pressure of –21 mm Hg; the deep posterior compartment measured 77 mm Hg, giving a critical closing pressure of –11 mm Hg. Pressures in the medial and anterolateral compartments were 46 and 27 mm Hg, respectively.

A single medial incision, two-compartment fasciotomy was performed within 3 h of presentation to the emergency department. An extensive haematoma was evacuated

between the soleus muscle and the transverse intermuscular septum, with no evidence of muscle ischaemia. No source of haemorrhage was identified and the wound was closed with two drains. A back slab was applied with the foot held in gravity equinus.

Post decompression, the patient was commenced on a prophylactic low molecular weight heparin regime. At re-inspection 36 h later, the wound was healing well and the drains were removed. On discharge, following consultation with his cardiologist, clopidogrel therapy was recommenced.

DISCUSSION

Compartment syndrome results from excessive pressure within a closed osseo-fascial compartment.⁵ The condition may be induced by penetrating trauma, exercise, or reperfusion after ischaemia.^{1–6} Increased intra-compartmental pressure, due to either increased compartmental contents (such as haematoma) or reduced compartmental size (such as caused by a tight cast), may potentially raise compartmental pressure levels up to or above 100 mm Hg reflecting low fascial compliance.⁷ Without surgical decompression, this condition leads to muscle and nerve ischaemia and eventual tissue death.⁸

Clinical history and physical examination remain the mainstays of diagnosis in compartment syndrome. Pain out of proportion to the injury, and pain on passive stretching of the affected compartments are pathognomonic of this condition.⁸ Delay to diagnosis has been shown consistently to be associated with a poor outcome regardless of further treatment.¹ To date, the only proven treatment for this condition remains decompression by surgical fasciotomy.⁸

Clopidogrel is an antiplatelet drug increasingly used in the secondary prevention of atherosclerotic vascular events. It is also used widely as antithrombotic therapy after percutaneous coronary intervention.⁹ To date, no previous cases of acute compartment syndrome associated with clopidogrel therapy have been reported.

In conclusion, the increasing number of patients on prolonged antiplatelet therapy increases the potential for haematoma associated compartment syndrome. It is particularly important to recognise that this condition may complicate relatively innocuous trauma or even occur spontaneously.¹⁰ Blunt trauma in elderly patients treated with such medications should excite a high index of suspicion regarding compartment syndrome with prompt treatment by surgical fasciotomy.

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