### CASE REPORT

**Compartment syndrome and systemic hypertension**

We present two rare variations related to compartment syndrome. The first is a 69-year-old hypertensive man with compartment syndrome of the arm. The second is a 58-year-old man with compartment syndrome of the forearm with severe compensatory hypertension.

Compartment syndrome exists when the intracompartmental pressure exceeds the perfusion pressure across the capillaries.\(^1\) The diagnosis is made clinically and when necessary, with compartment pressure measurements.\(^2,3\) Confirmation is by the bulging of muscle groups at fasciotomy.\(^2\) The indications for fasciotomy remain controversial.\(^1,3,4\) Hypotension is supposed to predispose to compartment syndrome,\(^2,5\) whereas hypertension is believed to protect from tissue ischaemia.\(^5\)

**Case reports**

**Case 1.** A 69-year-old man with systemic hypertension for 20 years and with severe left ventricular hypertrophy, presented with severe pain in his right upper arm following a trivial injury, sustained nine hours before. He had discontinued aspirin and antihypertensive medicines (losarten potassium, 25 mg and metoprolol 25 mg daily) 13 days before this injury. His blood pressure was 200/120 mmHg on admission. His right arm was swollen and firm to palpation and the hand was also swollen. There was bluish discoloration of the skin on the anteromedial aspect of the arm with blisters. The radial pulse was equal to the unaffected side, and the capillary refill time in the fingers was normal. There was generalised hypoaesthesia. The power of the elbow flexors and extensors and forearm muscles was decreased to MRC grade 4.\(^6\) Movement of the elbow was painful and restricted to an arc of 40°. All the laboratory investigations, including bleeding, clotting and prothrombin times were within normal limits. A diagnosis of acute compartment syndrome of the arm was unequivocal and an emergency fasciotomy performed. Immediately before operation, the blood pressure was 190/110 mmHg and sublingual nifedipine was given.

The operation was performed under supraclavicular block supplemented with local infiltration of lignocaine. Fasciotomy of the anterior compartment was undertaken through an anterolateral approach. There was a haematoma of about 750 ml anteromedially in the subcutaneous plane but no single bleeding or ruptured vessel was found. The muscles bulged out readily as the overlying fascia was incised, confirming the diagnosis. There was no muscle necrosis. The posterior compartment was released through a separate incision. A corrugated rubber drain was inserted and the wounds closed primarily as the skin became loose after evacuation of the haematoma. The blood pressure at the end of the operation was 130/80 mmHg. Post-operatively he was restarted on antihypertensive and antiplatelet medication (losarten potassium 25 mg, metoprolol 25 mg and aspirin 150 mg daily) and the blood pressure was controlled steadily. He was discharged on the fifth post-operative day. The hypoaesthesia resolved over a period of two weeks, and at four months there was only minor weakness of the muscles. At five years’ follow-up, he had normal function of the affected limb.

**Case 2.** A 53-year-old man sustained an injury to his left forearm in a road traffic accident. He was admitted for observation as he had a high intake of alcohol. His blood pressure was 130/80 mmHg on admission. He was seen by an orthopaedic consultant 12 hours after the initial trauma. The forearm and hand were swollen and tense and the skin had turned bluish with blisters. Passive movement of the fingers elicited severe pain in the forearm. There was no sensory or motor deficit in the hand and the radial and ulnar pulses were the same as in the normal limb. Capillary refilling was also normal. Radiographs of the elbow, fore-
arm and wrist showed no bony injury. His blood pressure was 200/110 mmHg. He had not been hypertensive previously. His chest radiograph, ECG and basic blood parameters were normal.

Having made a diagnosis of acute compartment syndrome urgent fasciotomy of both anterior and posterior compartments of the forearm was undertaken under general anaesthesia using the technique advised by Whitesides and Heckman. The blood pressure rose to 210/120 mmHg just prior to the incision and a nitroglycerine drip was started. The diagnosis was confirmed by the prompt bulging of the muscle groups in all compartments (the superficial and deep flexor compartments and the extensor compartments). There was no haematoma or muscle necrosis. The blood pressure normalised to 132/84 mmHg towards the end of the operation. Post-operative recovery was uneventful. After five days, the posterior wound was closed and a split-skin graft placed anteriorly. The blood pressure was normal during the second procedure and during the hospital stay without medication. When reviewed at six weeks, the function of the upper limb was normal.

Discussion

Acute compartment syndrome is more commonly seen in the limbs of young muscular individuals. Many predisposing factors have been described including systemic hypotension. Elderly people, with thin muscle and loose skin are generally spared. There are very few reports of acute compartment syndrome developing in the upper arm. The indications for fasciotomy have been extensively discussed. Many authors still recommend clinical criteria, while others rely on absolute compartment pressure measurements of 30 mmHg, 40 mmHg or 50 mmHg and others on criteria based on systemic blood pressure. Currently, a compartment pressure within 20 mm or 30 mm of the diastolic blood pressure is increasingly considered as an absolute indication for fasciotomy. Since we treat only patients who are haemodynamically and neurologically stable, and due to the discrepancies of compartment pressure measurements, we have made it our practice to take decisions based entirely on clinical grounds, with a high index of suspicion. To date, we have not faced any untoward sequelae from undertreatment of a compartment syndrome.

Hypotension is considered a risk factor and hypertension a protection from compartment syndrome. In the first case, hypertension appeared to be the predisposing factor by producing continuous bleeding from ruptured small vessels allowing a large haematoma to develop. The pressure of a haematoma approaches the systolic blood pressure in the larger vessels whereas the pressure in the microvasculature is much less. There is experimental evidence that even an external pressure of 12 mmHg can close capillaries. The higher the compartment pressure, the less time is required to initiate tissue damage. Thus, in the clinical situation underlying hypertension could be more detrimental than protective. A similar clinical situation was described by McLaughlin, Paulson and Rosenthal in a patient receiving low-molecular-weight heparin, where the compartment pressure was 110 mmHg while the blood pressure was 124/86 mmHg.

The second case shows that there can be significant compensatory hypertension with compartment syndrome. This might also have been a contributory factor in the first case. Compensatory hypertension follows trauma, and is supposed to protect the muscle from ischaemia. This problem and its implications have not been discussed in the literature. If there is underlying bleeding into the compartments, systemic hypertension, whether pre-existing or compensatory, will be detrimental in a patient with compartment syndrome. Hypertension will cause more bleeding and the increased compartment pressure in turn, will necessitate a higher systemic blood pressure to maintain perfusion. Thus, a vicious circle could generate. It may be wiser in these circumstances to depend on the clinical signs and absolute compartment pressure measurement rather than waiting to take a decision based entirely on measurements which are dependent upon the diastolic pressure.

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References


