COMPARTMENT SYNDROME AFTER PRESSURIZED INFUSION

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SUMMARY

We report two cases of compartment syndrome caused by extravasation of infused fluids and suggest a rationale for management and treatment based upon slit catheter monitoring of pressure in the compromised compartments.

KEY WORDS

Complications: compartment syndrome. Fluid balance: intravenous fluid administration.

Compartment syndrome is caused by any condition which increases interstitial pressure within a muscle compartment to the extent that blood flow is compromised. It is seen classically after trauma to an extremity with crush injury, vascular damage or subsequent tight bandaging. Its sequelae may include irreversible neural and muscular damage secondary to the ischaemic insult. Previously described iatrogenic causes of particular interest to anaesthetists include limb compression in the insensible patient [1], arterial cannula extravasation [2], hyperosmolar Bier block [3] and intra-arterial barbiturates [4]; these reports appear in orthopaedic surgery and neurology literature. Pressurized infusions through infiltrated i.v. catheters represent a potential source of compartment syndrome with resulting dysfunction [2, 5, 6].

We report two cases of compartment syndrome caused by extravasation of infused fluids and suggest a rationale for management and treatment based on monitoring of pressure in the compromised muscle compartments with a slit catheter.

CASE REPORTS

Patient No. 1

An 81-yr-old female was admitted to the Accident and Emergency department after a low velocity motor vehicle accident in which she suffered injuries to the body and head, with loss of consciousness. A CT scan of the head and abdomen revealed a subdural haematoma and retroperitoneal bleeding. When the patient's arterial pressure decreased from the admission value of 180/100 mm Hg to 90/60 mm Hg she was subjected to concurrent emergency craniotomy and exploratory laparotomy. Standard monitoring included ECG, pulse oximetry, capnography, oesophageal temperature and auscultation. A 20-gauge radial artery catheter and a 16-gauge i.v. catheter were placed in the right forearm. An 8.5-French gauge catheter was placed in the left antecubital fossa by a modified Seldinger technique. Anaesthesia was induced with fentanyl 15 µg kg^{-1} and pancuronium via the right forearm i.v. catheter. After the abdomen was opened, the patient's arterial pressure decreased to 70/30 mm Hg. Three units of packed cells were administered rapidly through the catheter in the left antecubital fossa and dopamine was infused via the right forearm to maintain the systolic pressure at approximately 100 mm Hg. When a roller pump for rapid infusion was connected to the 8.5-French gauge catheter in the left arm, the perfusionist reported that the delivery pressure exceeded 30 mm Hg. Investigation revealed tense swelling of the left arm. An alternative i.v. route was obtained in the right arm and the remainder of the case was uneventful.

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After operation, slit catheters (Stryker) placed in the left arm by an anaesthetist revealed interstitial pressures (ISP) of 38 mm Hg in the biceps brachii compartment, 28 mm Hg in the triceps compartment, 28 mm Hg in the volar forearm compartment, and 25 mm Hg in the dorsal forearm compartment. Concurrent mean arterial pressure was 83 mm Hg. Fasciotomy was deferred in favour of conservative therapy. The patient's left arm was elevated and interstitial pressure monitoring was continued. The left biceps compartment ISP returned to 4 mm Hg approximately 24 h later.

Patient No. 2

A 77-yr-old man was admitted to hospital for elective repair of a symptomatic abdominal aortic aneurysm. In the operating theatre, initial monitoring comprised ECG, non-invasive arterial pressure and pulse oximetry. A radial arterial catheter and a 14-gauge antecubital fossa catheter were placed in the left upper extremity. An 18-gauge i.v. catheter was inserted into the right hand. A pulmonary artery catheter was advanced to wedge position via an introducer sheath in the right internal jugular vein.

Anaesthesia was induced with i.v. fentanyl 10 μg kg^{-1} followed by pancuronium via the catheter in the right hand and maintained with nitrous oxide and isoflurane. The patient's left arm was placed at his side to facilitate positioning of a surgical retractor.

Fluids were allowed initially to flow into the left antecubital i.v. catheter by gravity but later, as blood loss increased, were infused under pressure. A total of 3 units of packed red blood cells, crystalloid 2 litre and starch solution 500 ml were infused through the catheter; no difficulties were noted by the anaesthetist. Approximately 90 min after surgical incision, the anaesthetist had difficulty obtaining an accurate arterial wave form and in withdrawing blood from the left radial artery cannula. Investigation revealed that the left arm was mottled and tense from the wrist to the border of the left pectoralis muscle. A presumptive diagnosis of compartment syndrome was made and all infusions to the left arm were discontinued.

A pulse oximeter probe placed on one of the digits of the left hand showed a saturation of 100 % and good correlation of the pulse rate with the electrocardiograph heart rate. Distal pulses in the left arm were palpable. Slit catheter monitoring (Stryker) was established and showed interstitial pressures of 23 mm Hg in the dorsal forearm compartment and 34 mm Hg in the triceps compartment. The biceps brachii compartment pressure was 26 mm Hg and the forearm volar compartment pressure 15 mm Hg. Simultaneous mean arterial pressure was 110 mm Hg.

The procedure was concluded uneventfully, and the arm was treated conservatively, with ice, elevation and topical agents to an area of epidermolysis which developed over the antecubital fossa. The swelling resolved, and the patient suffered no permanent muscular, neural or epidermal sequelae.

DISCUSSION

Compartment syndrome occurs when diminution of blood flow is sufficient to cause ischaemic damage to neurovascular and muscular components. The full syndrome, as described by Richard Von Volkmann in 1881, includes paralysis and post ischaemic contractures. These late sequelae are now seen infrequently because surgical fasciotomy is performed usually as soon as compromise is suspected. While normal interstitial pressure is approximately 2–5 mm Hg, there is no absolute measured interstitial pressure at which fasciotomy is indicated. The decision to perform fasciotomy should be based upon the perfusion pressure (ΔP) of the compartment [7]—the difference between the mean arterial pressure and the interstitial pressure, and a major determinant of oxygen delivery. Phosphorus-31 NMR spectroscopy has been used to determine the perfusion pressure at which anaerobic metabolism begins in resting canine muscle [7, 8]. Aerobic metabolism ceases when the perfusion pressure is less than 30 mm Hg in normal muscle or 40 mm Hg in traumatized muscle. These perfusion pressures are the accepted thresholds for decompressive fasciotomy. The venous P_{O_2} at these perfusion pressures is approximately 1.4 kPa, reflecting increased oxygen extraction [9], and is the threshold for injury to capillary endothelium [10]. Endothelial injury tends to widen the area of ischaemia by augmenting oedema formation, thereby further decreasing perfusion pressure. This cycle of ischaemia and oedema is characteristic of the compartment syndrome. Surgical fasciotomy increases perfusion pressure by decreasing interstitial pressure to near atmospheric pressure.

Infiltration of i.v. catheters occurs often, but usually the driving force is gravity alone. Al-

though anaesthetists frequently infuse fluids under pressure, only one report of compartment syndrome secondary to infiltration of a pressurized transfusion was found in the anaesthesia literature [6]. With the marked increase in the amount of blood and blood products being transfused under pressure in emergencies, this cause of compartment syndrome may become more common. The 8.5-French gauge untapered catheter, designed to give maximal flow by maximizing diameter, may distend fragile veins and predispose to rupture during cannulation or transfusions under pressure.

If large quantities of fluid are infused inadvertently into muscle compartments, slit catheters should be placed in the affected compartments. The technique for insertion of this device is simple. With a 14-gauge plastic i.v. catheter used as an introducer, the slit catheter is threaded into a muscle belly of the affected compartment. The slit catheter is connected to a low compliance transduction system. All air bubbles must be removed carefully or flushed from this relatively low pressure transducer system. The slit catheter provides more accurate intracompartmental pressures than transduction of the i.v. catheter. Transduction of the slit catheter signal may be performed with any standard pressure monitor. Specially designed monitors are manufactured by Stryker Surgical. The arterial and compartmental pressures used to calculate the perfusion pressure should be transduced with a common zero reference point. Fasciotomy should be considered if the perfusion pressure remains less than 30 mm Hg for more than a few minutes.

In the two cases presented, the perfusion pressures were well above the established threshold at which tissue damage ensues. Fasciotomy was avoided with little risk of further tissue damage.

In order to reduce iatrogenic complications, transfusion catheters should be placed in readily observable places, and re-evaluated when increased resistance to inflow is noted. Intermittent monitoring of transfusion catheter pressure may alert the anaesthetist to this hazard. If inadvertent extravasation of infusion substances occurs and fasciotomy is considered, slit catheter monitoring may help to direct rational therapy.

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