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CASE REPORTS

Bilateral compartment syndrome complicating prolonged lithotomy position

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Summary

A case of bilateral compartment syndrome after prolonged Lloyd-Davies lithotomy position is described. The diagnosis was made early, despite effective extradural bupivacaine–fentanyl analgesia. The aetiology, diagnosis, pathology and treatment of compartment syndrome are described. Complications of the syndrome may be life-threatening and permanently disabling. The anaesthetist should be aware of the potential complications of the operative positions of the unconscious patient. (*Br. J. Anaesth*. 1996;**77:**546–549)

Key words

Position, lithotomy. Position, effects. Complications, compartment syndrome.

The compartment syndrome describes a self-propagating cycle which occurs in the osseofascial compartments of the extremities. After a period of ischaemia, with reperfusion, the integrity of the capillaries may be compromised. This causes interstitial oedema and an increase in compartmental pressure. Untreated, it may lead to permanent neurovascular damage, myoglobinuria, renal failure, sepsis and death[1]. The compartment syndrome may complicate fractures, burns and crush injuries of the extremities[2]. It has also been described as a complication of some positions adopted for surgery, notably the lithotomy and knee–chest positions[3].

Case report

The patient was a 28-yr old man with a 10-yr history of ulcerative colitis. The previous year he had undergone an emergency total colectomy with ileostomy formation for acute exacerbation of the colitis. After colectomy he continued to pass blood and mucus per rectum and did not tolerate the ileostomy. Therefore, he was admitted for formation of an ileal pouch, excision of rectum and pouch–anal anastomosis. He had no other medical conditions of note.

Anaesthesia was induced with thiopentone 3 mg kg^{-1} and fentanyl 2 $\mu g\ kg^{-1}$ and neuromuscular block was produced with atracurium 0.5 mg kg^{-1}. The trachea was intubated and the lungs ventilated with 1–2% inspired isoflurane and 35% oxygen in air. An extradural catheter was introduced at the T12–L1 interspace. Extradural analgesia was produced using fentanyl 100 μg and 0.5% bupivacaine 17 ml, with a 3-ml test dose. An extradural infusion of fentanyl 4 $\mu g\ ml^{-1}$ and 0.125% bupivacaine was commenced during operation at a rate of 8 ml h^{-1}, 2 h later. Right internal jugular cannulation provided measurement of central venous pressure, and left radial arterial cannulation enabled continuous arterial pressure monitoring.

The patient wore thigh-high anti-embolism stockings and was positioned in the Lloyd-Davies lithotomy position, with the legs well padded and the pressure points protected. For most of the procedure, the table was in the Trendelenburg position. Surgery lasted 7 h 35 min.

Arterial pressure was maintained at or greater than 90/50 mm Hg throughout the procedure. Haemoglobin concentration was maintained greater than 10.2 g dl^{-1} in the peroperative period. Total blood loss was 4.2 litre. The patient's body temperature was 34.5 °C at the end of surgery. He was transferred to the intensive care unit for artificial ventilation of the lungs until his core temperature had returned to normal and fluid status was stable. He was sedated lightly with propofol until the tracheal tube was removed 3 h after operation. By then he was warm, had a stable cardiovascular state and the extradural infusion provided satisfactory analgesia.

The following day, 15 h after operation, with the extradural providing complete abdominal wound analgesia, the patient complained of bilateral painful lower legs. On examination, both lower legs were tense and swollen. The pedal pulses were present and sensation was intact, but the legs were very tender on palpation and pain was elicited on minimal dorsiflexion of the ankle.

No calf muscle pressure monitoring was available. A diagnosis of bilateral compartment syndrome was made clinically. Decompressing fasciotomies were scheduled urgently to minimize the risk of permanent sequelae.

The patient was transferred immediately to theatre. On each side, a 12-cm lateral incision was made under general anaesthesia. Bilaterally, a 3-cm segment of fibula was excised allowing four-compartment decompression. The wounds were left open. The extradural infusion was maintained.

Despite repeated wound inspection under general anaesthesia, on day 4 after operation footdrop was noted. The patient was pyrexic and unwell and so an

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infection screen was carried out. Blood cultures yielded coagulase-negative *Staphylococcus aureus*, and the leg swabs grew coliforms and bacillus. Appropriate antibiotics were given and he was returned to theatre for further leg wound inspection. The extradural had become dislodged and was therefore removed.

On this occasion, large amounts of liquefied muscle were found bilaterally. In the lateral and anterior compartments, extensive areas of necrotic muscle were excised, with blood loss of 4.5 litre. The patient was septicaemic and required cardiovascular support with inotropic agents guided by pulmonary artery catheter studies. Myoglobinuria was detected and creatine kinase concentration peaked at 13 000 iu $litre^{-1}$ on day 5 after the original operation. Urine output was maintained throughout, facilitated by daily infusion of mannitol 0.5 g kg^{-1} and dopamine 3 $\mu g\ kg^{-1}$ min^{-1}.

He subsequently required seven additional operations for debridement of wounds and change of dressings during the next 3 weeks. The ankles were splinted to minimize the equinus deformity. The final procedure involved application of split skin grafts to close the wounds.

The abdominal procedure was successful. The patient was discharged 3 months after admission with bilateral foot drop. He could walk short distances with the aid of crutches and ankle supports, after intensive physiotherapy. Despite early diagnosis, the development of compartment syndrome in this patient resulted in significant long-term handicap.

Discussion

Compartment syndrome is defined as a symptom complex that is caused by increased pressure of tissue fluid in a closed osseofascial compartment of the limb and this interferes with circulation to the myoneural components of the compartment[4]. It was first described as a complication of the lithotomy position in a healthy 38-yr old man after urethroplasty lasting 6.5 h[5].

Prolonged lithotomy position may predispose the patient to compartment syndrome in several ways. Leg perfusion may be decreased by elevation of the limb above the heart, excessive local pressure from improper placement of the legs in holders, external pressure from equipment or personnel, or excessive dorsiflexion of the foot. Other intraoperative factors may include hypotension, hypovolaemia, compression of the pelvic blood vessels in association with surgery and hypothermia with vasoconstriction. Initial hypotension on lowering the legs at the end of the procedure may compound the problem. Preexisting arterial disease may further reduce peripheral perfusion.

Ischaemia results in tissue membrane damage and leads to "leakage" of fluid through capillary and muscle membranes. With arterial reperfusion, the damaged membranes continue to leak and aggravate oedema formation and thus increase the pressure in the closed osseofascial compartment.

It has been shown that at rest the pressure within the calf compartments is normally less than 20 mm Hg[6]. Pressures as high as 50–55 mm Hg for 4–8 h have been cited to result uniformly in compartment syndrome[6]. The patient's baseline arterial pressure must be taken into account. For example, a patient with ankle systolic pressure of 100 mm Hg requires greater occlusion pressures and a longer exposure to these than a patient with peripheral vascular disease with an ankle systolic pressure of 40 mm Hg.

It is important to identify patients at risk of developing compartment syndrome. Patients in the lithotomy position for more than 5 h are at risk[7]. Although the lithotomy position is useful, patients should remain in this position no longer than is necessary.

During a staged operation, repositioning patients during the procedure so that they are in the lithotomy position only when necessary for surgical access should reduce the risk[7]. Care should be taken to ensure correct padding of the extremities. While elevation of the legs above heart level is unavoidable, direct pressure on calf muscles is preventable by using stirrups of the conventional Allen type, where the feet are supported but no pressure is exerted on the muscles of the posterior compartment. In the Lloyd-Davies leg supports, the lower legs rest in cradles, which increase the pressure in the calf muscles.

Irreversible muscle damage is likely to occur with occlusion times greater than 6 h[7]. In those cases in whom prolonged use of the lithotomy position is unavoidable, clinical vigilance is necessary.

Some workers have recommended pulse oximetry of the toes as a monitor of adequate lower limb perfusion during the lithotomy position[1,8]. Others found that the presence of an oximeter signal and a normal saturation reading did not necessarily imply adequacy of tissue perfusion[9]. In the absence of arterial trauma, peripheral pulses are usually present in the compartment syndromes. Pulse oximetry does not appear to be a reliable aid in the diagnosis or monitoring of impaired perfusion caused by increased intra-compartment pressure[10].

Because compartment syndrome is a reperfusion injury, signs and symptoms may not be immediately apparent and may develop insidiously. It develops during the postoperative period where the level of clinical observation may be less intense if the patient is not being nursed in a high dependency area. Residual lower extremity sensory block produced by intraoperative regional block with local anaesthetic may interfere with the early detection of subtle sensory changes. In our patient, and in other reported cases[4,11], effective extradural block did not mask the pain of compartment syndrome.

Urine should be checked for the presence of myoglobin. Serum creatine kinase is increased. The diagnosis of compartment syndrome is essentially clinical. There should be a high index of suspicion in patients at risk. The diagnosis can be confirmed by direct measurement of compartment pressure[12]. It is a simple, although invasive, procedure and therefore there is a risk of introducing infection. The differential diagnoses include deep venous thrombosis and peripheral nerve injury.

The definitive treatment of compartment syndrome is surgical decompression. In our patient, the diagnosis was made early, within 15 h, despite effective extradural analgesia. The fasciotomies must be of sufficient length in all compartments at risk to decompress effectively, and this should be confirmed

by direct compartment pressure monitoring. Development of motor neurone signs and loss of pulses are late signs and indicate that irreversible damage has already occurred.

The end result of untreated or inadequately treated compartment syndrome is rhabdomyolysis which has been defined as reversible or irreversible injury of skeletal muscle which alters the integrity of the cell membrane sufficiently to allow escape of cell contents into the extracellular fluid[13]. The association between rhabdomyolysis, myoglobin and acute renal failure was first described during the World War II in patients who had sustained severe muscle injury[14]. Numerous non-traumatic causes of rhabdomyolysis have been described of which compression, alcohol abuse and generalized convulsions are the most common aetiologies[13 15].

Myoglobin is similar in structure to haemoglobin, but smaller and more easily filtered in the kidney. It gives urine a classic red/brown colour. The amount of myoglobinuria does not correlate with the degree of underlying tissue injury[16–18].

The cause of acute renal failure after rhabdomyolysis is multifactorial[19]. It includes intratubular plugging with myoglobin and urate (see below), nephrotoxicity of iron from haem[20 21] which catalyses the formation of oxygen-free radicals and inactivation of the intra-renal nitric oxide vasodilatory influence[19 22], possible associated hypovolaemic or septic shock, hyperphosphataemia and possible tubular blockage from microthrombi secondary to associated disseminated intravascular coagulation.

The biochemical features characteristic of myoglobinuric acute renal failure include hyperphosphataemia, marked hyperuricaemia and severe hypocalcaemia, in addition to increased urea and creatinine concentrations, hyperkalaemia and metabolic acidosis. Hyperuricaemia may be caused by increased production of uric acid in the liver, secondary to increased liberation of adenine nucleotides by damaged muscle[23]. Severe hypocalcaemia may be caused by a combination of marked hyperphosphataemia and skeletal resistance to the calcaemic effect of parathormone in patients with acute renal failure[24]. A transient hypercalcaemia is sometimes seen during the diuretic phase of myoglobinuric acute renal failure. It has been postulated that this phenomenon could be attributed to a transient secondary increase in parathormone, immobilization or remobilization of calcium previously deposited in the damaged muscle[25].

The treatment and prophylaxis of renal failure associated with rhabdomyolysis should commence with cardiovascular resuscitation, where applicable, with aggressive prompt fluid therapy to encourage urine flow. Thereafter, forced alkaline diuresis is beneficial[26 27]. Better suggests a regimen of 12 litre per day of a solution containing sodium 110 mmol litre^{-1}, chloride 70 mmol litre^{-1}, bicarbonate 40 mmol litre^{-1} and mannitol 10 g l^{-1} in 10% glucose[19]. The end-point is to produce diuresis in excess of 8 litre per day, maintaining pH $\geqslant$6.5. The fluid regimen should be continued until myoglobinuria ceases. Bicarbonate can be discontinued at 36 h. Acetazolamide can be given if arterial pH is $\geqslant$7.45.

Urate and myoglobin are more soluble in alkaline urine. The renal protective effect of alkalinizing the urine has been demonstrated in animals injected with myoglobin[26 28]. Mannitol also has a beneficial action in the prophylaxis of rhabdomyolysis acute renal failure. The beneficial extra-renal effects include increasing mean arterial pressure[19], increasing cardiac contractility[19 29] and decreasing skeletal muscle oedema, hence decompression of muscle tamponade[30]. The beneficial direct renal effects include a decrease in blood viscosity and oncotic pressure across the glomerulus causing an increase in glomerular filtration rate (GFR), dilatation of glomerular capillaries and stimulation of vasodilatory prostaglandin release, increase in proximal intratubular flow and hence reduction of obstruction, and possible reduction of tubular cell swelling in addition to free radical scavenging[31 32].

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