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Lesson of the Week

Crush syndrome following unconsciousness: need for urgent orthopaedic referral

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Patients who have been unconscious may develop crush syndrome, which requires urgent orthopaedic referral

The acute compartment syndrome occurs when increased pressure within osteofascial compartments results in local muscle ischaemia. If left untreated it may lead to muscle necrosis and contractures. The systemic manifestations of this—the crush syndrome—are the results of haemodynamic and metabolic disturbances and acute renal failure.¹ Failure to appreciate the importance of muscle necrosis as the underlying problem in the crush syndrome may have disastrous consequences.

The crush syndrome was originally described during the London Blitz in civilians who had been buried beneath the debris of destroyed houses.² Nowadays, victims are typically encountered in war zones, in mining disasters, after earthquakes, and in industrial or road traffic accidents.³ The syndrome may also develop after isolated compression of arms or legs by the victim's own body—for example, during unconsciousness after a drug overdose.¹ Such patients often delay seeking medical attention or have other more apparent complications that need urgent

attention. The local signs of muscle compression and necrosis may therefore initially be overlooked. We describe 11 such cases referred to our unit over 51 months.

Case reports

During April 1989 to July 1993, 11 patients were admitted to this infirmary with the crush syndrome secondary to a drug overdose. They were all referred for orthopaedic assessment after a considerable delay—mean 35 hours (table). Nine patients were men. The mean age was 31 years (range 20-54 years). As all the patients had been unconscious for an unknown length of time before presentation, the delay from time of injury until medical attention was given could not always be assessed. Four patients were unconscious on admission. Ten patients had taken an overdose of sedatives or painkillers, and one patient had carbon monoxide poisoning. During the period of unconsciousness their torso had compressed one or

Details of 11 patients with crush syndrome secondary to drug overdose 1989-93

Case No	Sex	Age (years)	Limb affected	Symptoms on admission	Time between admission and surgery (h)	Dialysis	Surgical treatment	Outcome	
								Functional	Renal
1	M	20	Upper arm	Yes	29	Yes	Fasciotomy with excision of necrotic muscles	Loss of flexion of elbow	Normal
2	M	24	Upper arm	Yes	24	Yes	Fasciotomy with excision of necrotic muscles	Stiff shoulder	Normal
3	M	35	Calf	NR	>62	Yes	Below knee amputation	—	Failure
4	M	54	Both calves	Yes	17	Yes	Bilateral above knee amputation	—	Failure
5	F	43	Calf	Yes	21	Yes	Through knee amputation	Died	
6	M	23	Forearm	Yes	30	No	Fasciotomy	Contractures	Normal
7	M	25	Calf	?		No	Through knee amputation	—	Normal
8	F	28	Forearm	Yes	51	No	Fasciotomy	Good	Normal
9	M	38	Forearm	Yes	22	No	Fasciotomy	Died	
10	M	24	Thigh	Yes	53	Yes	Fasciotomy	Died	
11	M	29	Forearm	Yes	43	Yes	Fasciotomy with excision of necrotic muscles	Loss of digital flexors	Normal

NR=not recorded.

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more limbs: in six patients an arm, in five patients a leg, and in one patient both legs. None of the patients had any bony injuries. Signs of raised intracompartmental pressure were noted on admission in nine patients.

We describe two typical cases in detail. The table summarises all 11 cases.

CASE 11

After a domestic argument a 29 year old man took an overdose of methadone and temazepam. He was found lying on his left side on the floor and was taken to the accident and emergency department. On admission his Glasgow coma score was 3, and he had severe respiratory insufficiency, with a tachycardia of 125 beats/min. At this stage pressure marks were noted over the dorsum of his left forearm and over the left side of his chest. He was intubated and taken to the intensive care unit. He was oliguric and acidotic.

On admission blood concentrations of note were sodium 139 mmol/l (normal range 132-144), potassium 7.6 mmol/l (3.3-4.7), carbon dioxide 33 mmol/l (24-30), urea 8.3 mmol/l (2.5-6.6), creatinine 194 μ mol/l (55-150), hydrogen ions 60 nmol/l (36-44), bicarbonate ions 27 mmol/l (21-27.5), and creatinine kinase 16 294 U/l (30-200). Carbon dioxide pressure was 8.7 kPa (4.4-6.1) and oxygen pressure 8.0 kPa (12-15). Paracetamol and salicylates were not detected. His urine was not tested for myoglobin. On the suspicion of rhabdomyolysis initial treatment consisted of forced alkaline diuresis. This produced an improvement in his acidosis, hyperkalaemia, and renal function.

His left forearm became increasingly swollen and discoloured with the appearance of bullae (figure). Forty three hours after admission he was eventually referred for an orthopaedic opinion. A compartment syndrome was evident, and immediate fasciotomies of all forearm compartments were performed. His pre-operative intracompartmental pressures were 90-100 mm Hg and his blood pressure 130/80 mm Hg. After fasciotomy intracompartmental pressures returned to normal values. Necrosis of several flexor muscles necessitated extensive debridement. Likewise, a considerable portion of his left pectoralis major muscle required excision. All wounds were left open for further debridement 24 hours later. They were closed by split skin grafting 72 hours after initial surgery.

After surgical debridement of necrotic tissues his renal function returned to normal, and he was transferred to an orthopaedic ward for rehabilitation. Six months after admission his hand function was poor owing to the loss of wrist and finger flexors. His renal function recovered completely.

CASE 4

A 54 year old man took an overdose of thioridazine and co-dydramol. He was found unconscious on the floor and admitted to a medical ward. Extensive pressure marks were found on both calves. His conscious level was depressed, but he did not require assisted ventilation. He was hypotensive and tachycardic.

On admission blood concentrations of note were: sodium 142 mmol/l (132-144), potassium 5.2 mmol/l (3.3-4.7), carbon dioxide 4.8 mmol/l (24-30), urea 11.9 mmol/l (2.5-6.6), creatinine 160 μ mol/l (55-150), hydrogen ions 49 nmol/l (36-44), bicarbonate ions 18 mmol/l (21-27.5), and creatinine kinase 154 500 U/l (30-200). Carbon dioxide pressure was 4.8 kPa (4.4-6.1) and oxygen pressure 19.8 kPa (12-15). Paracetamol and salicylates were not detected. His urine was not tested for myoglobin.

Initial treatment was intravenous fluids, dopamine, mannitol, and sodium bicarbonate. Despite these measures his renal function worsened, with an

Forearm in case 11 after compression by chest showing discoloration and bullae (top) and pressure marks (bottom). Fingers show flexion deformities caused by contractures of extrinsic flexor muscles of forearm (Volkmann's contracture)

appreciable increase in his creatinine (390 μ mol/l) and urea concentrations (20.3 mmol/l). Sixteen hours after admission an orthopaedic opinion was sought about the dramatic swelling and discoloration of both calves. The acute compartment syndrome was diagnosed in both calves (the pressure difference between diastolic pressure and intracompartmental pressure was less than 30 mm Hg), and he underwent prompt fasciotomy and muscle debridement of all four compartments. He returned to the operating theatre regularly for further debridement over the next few days, but because of extensive muscle necrosis both legs were amputated above the knee one week after admission. Postoperatively he was given haemodialysis for five weeks, but his renal function did not return to normal. When seen 20 months after primary admission his blood creatinine concentration was 287 μ mol/l and urea concentration 16.8 mmol/l.

Discussion

In a compartment syndrome the raised pressure within a closed osteofascial compartment compromises the circulation to the tissue within that space. As the pressure increases the veins become obstructed first. This obstruction of the venous outflow is not initially accompanied by diminished arterial inflow, and the result is oedema and further increase in the intracompartmental pressure. Peripheral pulses are generally present. The arteriovenous pressure gradient decreases, resulting in impaired tissue perfusion and ischaemia. This self-perpetuating process of oedema and ischaemia results in muscle necrosis and neural damage. If the cycle is not broken by decompressive fasciotomy the entire compartment and its neighbouring compartments may become affected.¹

If a compartment syndrome is suspected the intracompartmental pressure should be measured immediately. A split central venous catheter is inserted into the affected compartment(s), filled with saline, and connected to a pressure transducer and a recorder.⁴ Absolute values are unreliable because blood flow depends on the difference between blood pressure and

compartment pressure. In normal compartments the pressure approaches 0 mmHg.⁵ As intracompartmental pressure increases tissue perfusion decreases, depending on the blood pressure. A pressure difference of less than 30–40 mmHg between the compartment pressure and the diastolic blood pressure is associated with the inability of muscle to maintain a normal metabolic state.⁶ After 15 minutes this will result in functional changes in muscles and nerves, and after four to eight hours it may lead to ischaemic necrosis with permanent functional deficits.⁷

Compression of arms or legs causes considerable increases in intracompartmental pressure. In a group of healthy volunteers compression of the forearm or the leg by the subject's own torso consistently produced raised intramuscular pressures in the area of direct contact.⁷ Pressures of 100–225 mmHg (mean 178 mmHg) were recorded in forearms compressed by the rib cage. Legs compressed by the other leg showed pressures of 29–160 mmHg (mean 64 mmHg). The pressures recorded were sufficient to cause muscle ischaemia and necrosis by local obstruction of the circulation.

In all our patients the pressure difference between the intracompartmental pressure and the diastolic blood pressure was less than 30 mmHg, confirming the presence of an acute compartment syndrome.⁶ Urgent explorations and fasciotomies of the affected compartments were carried out. If the muscles were necrotic, or did not respond to mechanical stimuli, extensive debridement was performed. All wounds were left open for delayed closure by split skin grafting. If necrotic muscle was found in all compartments amputation through viable tissue was performed.

MUSCLE CONTRACTURE, NECROSIS, AND RHABDOMYOLYSIS

Early fasciotomy may prevent the development of Volkmann's contracture, as well as preventing or diminishing the systemic effects of local muscle necrosis.¹⁸ If intracompartmental ischaemia is left untreated, muscle necrosis may result in rhabdomyolysis. This results in myoglobinaemia, hyperphosphataemia, hyperkalaemia, hyperuricaemia, metabolic acidosis, intravascular volume depletion, and coagulation defects.⁹ Myoglobin in the bloodstream is readily filtered and excreted by the kidneys, which may result in myoglobinuric acute renal failure. The release of muscle constituents due to leakage of potassium and myoglobin may continue for up to 60 hours after the release of the compression.⁹

The delay in diagnosing raised intracompartmental pressure was considerable in all of our cases. The average time from admission to treatment was 35 hours (range 17–62 hours). This delay resulted from a failure to suspect the diagnosis, a preoccupation with the patients' often massive medical problems, and the fact that patients were comatose or poorly compliant. On admission, however, nine out of 11 patients had symptoms suggestive of raised intracompartmental pressure (pain, swelling, pressure marks, altered sensation, and muscle weakness or pain on passive muscle stretching).

Blood results on admission reflected the extent of muscle damage. There was an obvious difference between patients with ischaemia of large muscle compartments (such as legs or shoulders) and patients with forearm ischaemia alone. Those with ischaemia of large muscle compartments had raised serum urea and creatinine concentrations, whereas those with forearm ischaemia alone had normal results. All patients, however, had a metabolic acidosis.

Six out of 11 patients needed haemodialysis. Three patients died. Of the eight survivors, seven had disastrous outcomes: four patients had at least one leg

amputated and four patients had extensive muscle debridement leaving severe residual deficits. Only one patient had a good functional result with minimal residual deficits. Two of the eight surviving patients had permanently impaired renal function.

TREATMENT

Successful treatment of the crush syndrome depends on an immediate diagnosis, which is possible from a history of prolonged immobilisation and the finding of a swollen arm or leg. If possible, a description of the position in which the patient was found should be obtained. In alert and cooperative patients the diagnosis may be obvious. In non-cooperative patients pain on passive stretching of the muscles, motor weakness, and sensory deficits may pass unnoticed, and the presence of peripheral pulses may give a false sense of security.⁸ By the time that obvious motor signs have occurred or pulses have disappeared, however, ischaemia will be well established and irreversible damage will have occurred. When the clinical presentation is unusual, measurement of intracompartmental pressure with a central venous catheter under local anaesthesia is valuable. If the intracompartmental pressure is only marginally raised the pressure should be measured and recorded continuously until a clear fall in pressure is observed. It should be remembered that late rises in pressure due to post ischaemic swelling may be seen. Elevation of the arm or leg should be avoided as it may compromise the perfusion even further by reducing the transmural pressure gradient.⁸ If the difference between the intracompartmental pressure and the diastolic blood pressure is less than 30 mmHg urgent extensive fasciotomy must be undertaken. In our hands the morbidity associated with fasciotomy is low. We do not agree that uncontrollable infection occurs when a closed injury is converted to an open injury¹⁰: none of our 11 patients developed a postoperative infection.

CONCLUSION

The immediate medical treatment of the crush syndrome should be directed at the hypovolaemic shock and oliguria. The underlying problem—the compartment syndrome with muscle necrosis—must be approached urgently, however, to prevent further damage to the arm or leg and to decrease the systemic effects of myonecrosis. The only reliable way to break the vicious cycle of ischaemia and oedema and preserve function is to carry out urgent decompression of all affected compartments by extensive fasciotomies. We hope that heightened awareness of this problem in medical and intensive treatment units will result in reduced referral times to orthopaedic units.

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