UNRECOGNISED COMPARTMENT SYNDROME
IN A PATIENT WITH TOURNIQUET PALSY

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We report a case in which compartment syndrome and tourniquet paralysis occurred simultaneously. This is a previously unreported combination which presents a diagnostic problem. We recommend that electrophysiological studies and continuous monitoring of compartment pressures should be used to minimise morbidity in high-risk cases.

The tourniquet is one of the most useful appliances in limb surgery and, with the introduction of the pneumatic tourniquet, complications arising from uncontrolled pressure are becoming less and less common (Hamilton and Sokoll 1967; Middleton and Varian 1974) and permanent paralysis very rare. Despite extensive laboratory and clinical studies on the effect of pressure on neurovascular tissues and muscles, no agreement has been reached as to the maximum permissible pressure of the tourniquet and the safe duration of tourniquet time (Flatt 1972; Klenerman and Hulands 1979; Estersohn and Sourifman 1982; Sapega et al. 1985). Sporadic reports of cases of tourniquet paralysis are still found (Denny-Brown and Brenner 1944; Calderwood and Dickie 1972; Fowler, Danta and Gilliatt 1972).

Tourniquet paralysis can confuse the clinical picture of a compartment syndrome, which, especially in the lower leg, is not uncommon after closed fractures or surgery (Matsen 1980). The diagnosis of this potentially serious complication depends upon a high index of suspicion and monitoring of the intracompartmental pressure.

The patient described below developed an anterior compartment syndrome of the leg after operation but, because of concomitant tourniquet paralysis, diagnosis was delayed.

CASE REPORT

A 26-year-old clerk was admitted with a closed comminuted trimalleolar fracture of the left ankle sustained while playing football. There was no neurovascular deficit and the fracture was treated by open reduction and internal fixation. The operation was performed under general anaesthesia with a tourniquet applied to the proximal thigh at a pressure of 450 mmHg. Because of technical difficulties, the operation was prolonged and the total tourniquet time was 3 hours 15 minutes. After the first two hours the tourniquet was deflated for 25 minutes and then reinflated.

Postoperatively, the patient was found to have complete motor paralysis and almost complete sensory loss below the knee. Passive movement of the toes caused no pain. Where the tourniquet had been applied there was a ring of pigmented skin, which was thought to be due to a reaction to the iodine skin preparation. The patient was regarded as a case of tourniquet paralysis and was observed. The pneumatic tourniquet and the pressure gauge used were examined and found to be in good order.

About 30 hours after operation, the patient complained of severe pain in the front of the leg and a burning sensation in the sole of the foot. Active movements of the toes were detected for the first time, but passive planterflexion of the toes caused significant pain over the anterior tibial compartment. The appreciation of pinprick and touch over the lower leg were still absent. Emergency open fasciotomies of the anterior, lateral and superficial posterior compartments were performed. The muscles in the anterior compartment were found to be pale and their contractility was diminished; the other compartments were not under tension.

Subsequently debridement of the long toe extensors and the tibialis anterior muscle was required because of muscle necrosis. Three months later, after the fracture had healed, anterior transfer of the tibialis posterior tendon was performed for the drop-foot.
DISCUSSION

The diagnosis of compartment syndrome is usually made on clinical grounds and four criteria have been suggested (Matsen 1980; Matsen, Winquist and Krugmire 1980).

1. Pain out of proportion to the clinical situation.
2. Weakness and pain on passive stretching of the muscles of the compartment.
3. Hypoaesthesia in the distribution of the nerves running through the compartment.
4. Tension within the fascial boundaries of the compartment.

However, in the presence of concomitant proximal neurological damage the first three of these criteria may not apply and it becomes difficult to analyse the ambiguous clinical findings.

In tourniquet paralysis, the damage to the nerve is most severe where pressure has been applied and the larger fibres are usually involved earlier than the smaller ones. There is motor loss and diminished sensation affecting touch, light pressure, vibration and position sense distal to the lesion. In 1954 Moldaver suggested that the absence of paraesthesia or pins-and-needles which are mediated by touch fibres is diagnostic of this syndrome. Temperature, pain and sympathetic fibres, however, should still be functioning and only if damage is severe would the fast pain fibres be impaired. This is in contrast to a compartment syndrome where the pain fibres are affected early by ischaemia and there is hypoaesthesia in the affected area (Matsen 1980; Mubarak and Hargens 1981).

When a patient presents with complete motor and sensory loss, the anatomical distribution of the neurological deficit establishes the level of the lesion. With compartment syndromes, the muscle are directly involved and only the nerves that run through the compartment are paralysed; by contrast, in tourniquet paralysis the nerves and muscles distal to the level of damage are normal and will react to external electrical stimulation (Matsen et al. 1980). In our patient the complete motor and almost complete sensory loss below the knee can be explained by sciatic nerve compression in the thigh. Compartment syndromes involving all the compartments of the leg can cause similar motor deficiency, but this would be most unusual and even if it did occur the cutaneous sensory nerves should be spared.

Prophylactic compartment pressure monitoring is an important aid to the diagnosis of early compartment syndrome (Allen et al. 1985). It has been suggested that the duration of pressure is as important as its magnitude; thus, a continuous rise in pressure above 40 mmHg or an absolute rise above 50 mmHg are both indications for fasciotomy. However, because this syndrome can develop as late as three days after injury, it is only practical to monitor patients who are particularly at risk.

In the recovery phase of tourniquet paralysis, the order in which the components of sensation recover is: fast pain, position, pressure, vibration, motor and finally touch (Moldaver 1954). Motor and sensory impairment may last for three months or more but permanent paralysis is rare. By contrast, if fasciotomy for compartment syndrome is performed late or inadequately, muscle damage may be irreversible. Nerve function in such cases, although rapidly affected by ischaemia, may eventually recover completely. It has been shown by Lundborg (1970) that intraneural microcirculation returns fully even after 6 to 8 hours of circulatory arrest.

Unfortunately, in our patient, although there were the high-risk factors of a closed comminuted fracture and a prolonged tourniquet time, compartment syndrome was not suspected early. By the time pain and motor function had recovered, the muscles in the affected compartment had already suffered irreversible damage.

The cause of the tourniquet paralysis in our patient is still unknown, since the tourniquet and the pressure gauges were not found to be at fault and the duration of tourniquet application (and the interval of release) had been within the permissible range suggested by various authors (Flatt 1972; Klenerman and Hulands 1979; Estersohn and Sourifman 1982). It is possible that the duration of ischaemia in the muscles was long enough to produce postoperative swelling which contributed to the development of compartment syndrome. This, however, is difficult to prove, although it has been shown that ischaemia for more than two hours produces significant swelling in the limb due to increased hyperaemia and capillary permeability associated with tissue anoxia (Klenerman 1983).

The association of tourniquet paralysis and compartment syndrome is rare. In order to make a prompt diagnosis, prophylactic continuous compartment pressure monitoring should be performed. In the presence of concomitant proximal neurological lesions, the diagnosis can be made from the clinical pattern of neurological deficit with the aid of electrophysiological studies. Most important, however, is the acumen of the clinician.
REFERENCES


