Acute compartment syndrome: diagnosis and immediate care

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Acute compartment syndrome is an uncommon but potentially limb-threatening condition whose early recognition and treatment can prevent or reduce serious complications. Identifying at-risk patients and appropriate investigation is the key.

Clinicians dealing with trauma need to be familiar with the varied clinical presentations of acute compartment syndrome. This condition, if missed, may delay treatment leading to infection, muscle contracture, paralysis and occasionally amputation.

Delay in diagnosis may be the result of lack of awareness or an atypical clinical presentation. A high index of suspicion is required so that an early surgical consultation can be obtained to avoid complications.

WHAT IS ACUTE COMPARTMENT SYNDROME?

The upper and lower limbs are enclosed in a deep fascial covering which divides them into different compartments. The contents of the compartment are skeletal muscles, which form the bulk, and the neurovascular structures which pass through the compartment. Owing to the unyielding nature of this fascial envelope, an increase in the intracompartment pressure may reduce the capillary inflow below a critical level necessary for tissue viability. This may produce irreversible ischaemic changes in the skeletal muscles and nerves leading to muscle contractures (Volkmann's ischaemic contracture) and paralysis respectively (Volkmann, 1881).

PATHOPHYSIOLOGY

Acute compartment syndrome may be caused by a variety of limb injuries. A significant trauma may lead to swelling within a fascial compartment, which causes an increase in intracompartmental pressure. High pressure within this closed compartment results in an increased venous pressure, which lowers the arteriovenous pressure gradient, resulting in decreased local blood flow. Eventually, arteriolar compression occurs, leading to muscle and nerve ischaemia with muscle infarction and nerve damage if prompt treatment is not instituted. Sensory changes in the nerve, such as paraesthesia and hypaesthesia, develop within 30 minutes of onset of ischaemia. Irreversible nerve damage begins after 12–24 hours of total ischaemia (Masten, 1975). It has been shown that irreversible functional changes start in the muscle after 4–8 hours (Whitesides et al, 1971), which may lead to muscle contracture.

AETIOLOGY

Fractures of the tibial diaphysis, soft tissue injury, distal radial fractures in young adults and diaphyseal fractures of the radius and ulna are commonly implicated in acute compartment syndrome (McQueen et al, 2000). The common belief that open fractures adequately decompress the compartments and hence prevent an increase in pressure is often incorrect. Studies in the past have clearly shown that an open fracture runs the risk of developing acute compartment syndrome (De Lee and Stiehi, 1981; McQueen et al, 2000).

Both arterial and venous injury can lead to compartment syndrome. A surgical procedure that revascularizes the limb, such as embolectomy, thrombolysis or bypass surgery, can result in compartment syndrome and is caused by tissue swelling following reperfusion.

Soft tissue injury without a fracture may occur following a direct blow or after a major crush to a muscle compartment. Patients taking anticoagulant or with a bleeding disorder are at higher risk. Prolonged limb compression in patients with altered consciousness can also lead to features of crush syndrome and compartment syndrome.

Clinicians should be beware of the intravenous drug abuser who presents with a swollen and painful extremity. This may result from intravenous injection of substances, e.g. heroin, morphine, lysergic acid diethylamide (LSD) or methadone.

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Young people, especially men, have the highest incidence of compartment syndrome, possibly because young men have relatively large muscle volumes enclosed by the fascial envelope. Common causes of acute compartment syndrome are listed in *Table 1*.

PATIENT ASSESSMENT

In most instances there is a history of trauma such as a direct blow over the shin during a football tackle. It is prudent to have a high index of suspicion where a patient was found unconscious, as the history from the patient may not be reliable.

The extremity is usually swollen, firm to palpation and very tender. The classic signs of compartment syndrome are pain, pallor, paraesthesia, paralysis and pulselessness. It is important to diagnose this condition at an early stage, while still reversible. If one waits for a classic picture to emerge it might be too late for limb salvage.

SIGNS AND SYMPTOMS OF ACUTE COMPARTMENT SYNDROME

Pain

This is perhaps the most important sign, which is different to the pain experienced by the initial trauma. This is produced by muscle ischaemia and is only partially relieved with usual analgesics used for fractures. Therefore pain out of proportion to that expected with the injury should provoke a strong suspicion of acute compartment syndrome. Passive stretching of an ischaemic muscle exacerbates the pain, which is an important early finding suggesting need for referral.

Pallor

Pallor is a late and unreliable sign for the diagnosis of this condition.

TABLE 1.
Common causes of acute compartment syndrome

Fracture	Tibial shaft fracture
	Forearm bone fracture
	Distal radius fracture in young patients
Soft tissue injury	Prolonged limb compression
	Crush injury
	Burns
Vascular	Acute arterial injury
	Delayed or reperfusion injury
	Associated with anticoagulation
	Intravenous/intra-arterial drug injection

Paraesthesia

The presence of paraesthesia in the dermatomal pattern of the involved peripheral nerve in the compartment may be an early sign of impending nerve ischaemia.

Paralysis

Paralysis is a late feature, which is sometimes seen in patients who present late.

Pulselessness

The peripheral arterial pulse rarely disappears. Peripheral pulses are usually palpable except in cases of arterial injury and should not be relied upon.

DIAGNOSIS

Diagnosis can be made on clinical judgment, which may be supplemented by measurement of intracompartmental tissue pressure. Pressure monitoring should be considered in all patients listed in *Table 2*.

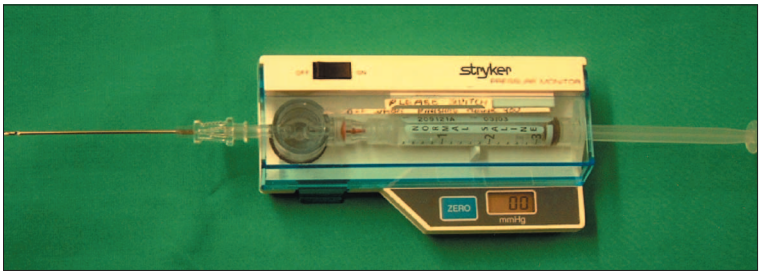
The normal tissue pressure is approximately 0 mmHg (-2 ± 2 mmHg) (Hargens et al, 1978; Garfin et al, 1981). The ideal pressure threshold for performing a fasciotomy is still not known. McQueen and Court-Brown (1996), in a study of 116 patients with tibial fractures, suggested decompression of the involved compartment if the differential pressure between tissue pressure and diastolic blood pressure falls to under 30 mmHg.

A number of instruments are available to measure intracompartmental pressure. Stryker (Newbury, Berks) produced a hand-held device which provides an accurate single pressure reading (*Figure 1*). Whitesides and colleagues

TABLE 2.
Patients in whom pressure monitoring should be considered

Unconscious patients
Patients who are difficult to assess, such as children
Patients with equivocal signs and symptoms
Patients with multiple injuries
Crush injury of the foot

Figure 1. Stryker hand-held instrument for compartment pressure measurement.



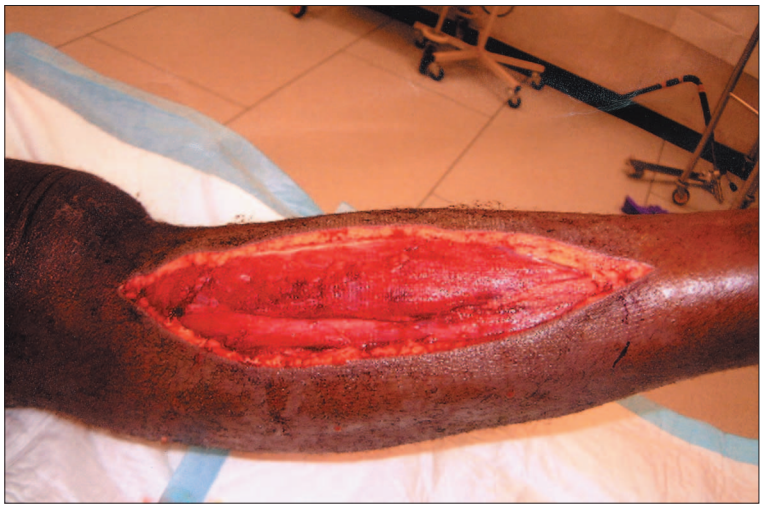
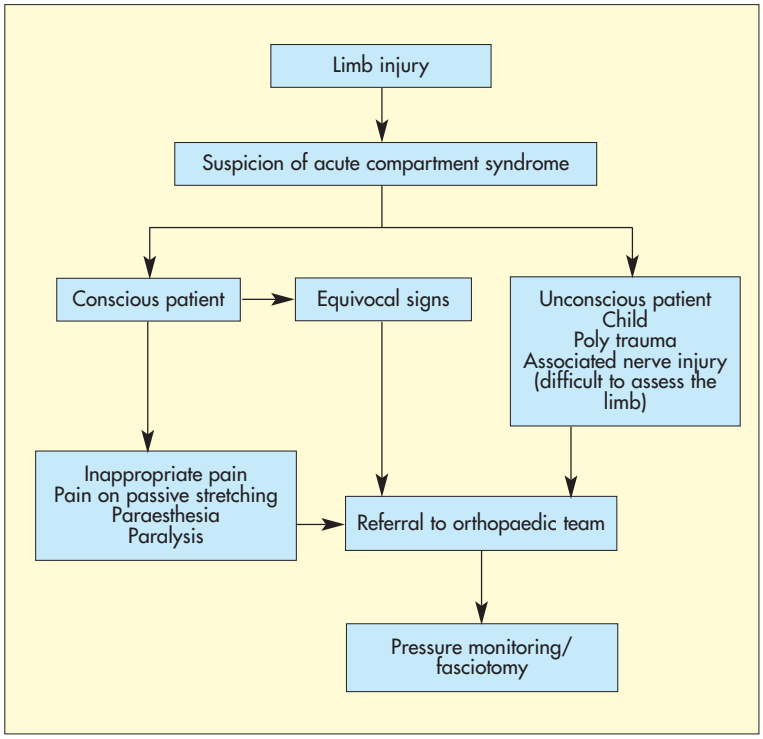


Figure 2. Fasciotomy for a leg compartment syndrome – note the wide skin gap on release of fascial constraint.

Figure 3. Flow diagram for management of potential compartment syndrome.



KEY POINTS

- A high index of suspicion is required in at-risk patients.
- Inappropriate pain response to the injury should be viewed with suspicion: compartment pressure measurement should be considered.
- Patients with open fractures may also be at risk.
- Absence of the peripheral pulse is a late and unreliable sign.
- Early diagnosis and treatment is important to prevent complications.

described a simple and useful method using intravenous tubing, a three-way stopcock, a syringe and a mercury manometer, which are easily available in any hospital (Whitesides et al, 1975).

EARLY MANAGEMENT

The goal of treatment of compartment syndrome is reduction of the intracompartmental pressure thus facilitating reperfusion of the ischaemic tissue. Time is a critical factor; the longer the duration of elevated pressure, the greater the potential for tissue hypoxia.

External pressure should be released by removing any cast, splint or occlusive dressing which may lower the compartment pressure by as much as 85% (Garfin et al, 1981). When splitting the cast, it is important to remember that all the layers should be divided, including the wool, to expose the skin.

Elevation of the limb up to the level of the heart may also be used to reduce the compartment pressure. However, this may adversely affect the perfusion pressure and frequent observation of the limb is necessary to monitor the progress of the symptoms and signs.

Communication is the key, with early consultation with the appropriate surgical team. The primary treatment of acute compartment syndrome is decompression by a fasciotomy which should be performed on an urgent basis (*Figures 2* and *3*). HM

Conflict of interest: none.

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