Compartment syndromes

Compartment syndrome occurs when pressure within a closed muscle compartment exceeds the perfusion pressure and results in muscle and nerve ischaemia. Two distinct conditions are recognized: acute and chronic (exertional) compartment syndromes. Differences in aetiology, pathophysiology and management are elaborated on in this article.

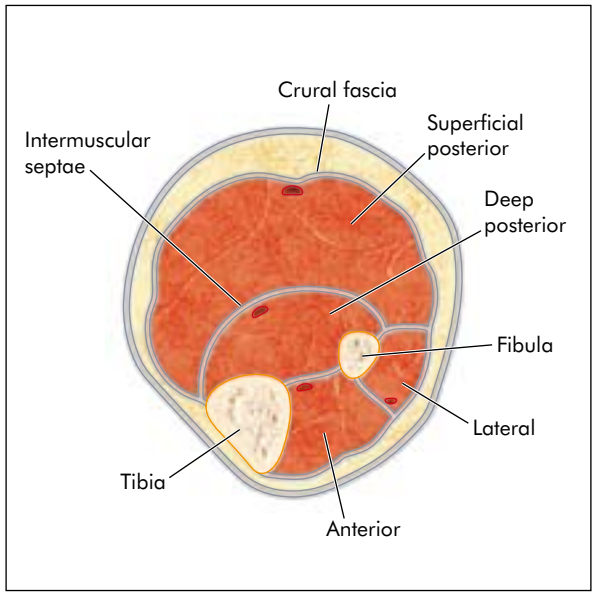
Compartment syndrome exists in two distinct forms: acute and chronic. These have very different aetiology and pathophysiology, and thus have different methods of management.

Acute compartment syndrome

Acute compartment syndrome (ACS) is a condition which occurs when increased tissue pressure within a myofascial compartment (*Figure 1*) compromises the vascular supply and the function of structures within that space. A prerequisite for the development of increased tissue pressure is an envelope restricting the volume available to the enclosed tissue. Such envelopes include the epimysium, the fascia, the skin, and casts or other circumferential dressings.

The increase of hydrostatic and osmotic pressure in the anatomical compartment leads to increased local venous pressure that results in a decrease in the arteriovenous gradient and a subsequent decrease in arterial inflow. A cascade of injury follows, with disruption of the metabolic processes of the muscle, cytolysis and the release of osmotically active cell contents. This results in further extravasation of fluid from capillaries and added pressure (on the structures) within the compartment, compromising the function of structures such as blood vessels and nerves and the end muscle units they supply.

Figure 1. Compartments of the lower limb.



Aetiology

Volksmann (1872) suggested that paralysis and contracture of limbs 'too tightly bandaged' resulted from ischaemic change of the muscles. Matsen (1975) suggested that this might be the result of either decreased compartment size or increased compartmental contents. Decreased compartment size may be the result of localized external pressure (e.g. a tight dressing), or closure of fascial defects. Increased compartmental contents may result from bleeding (e.g. vascular injury, bleeding disorders), capillary permeability (post ischaemic swelling, exercise, trauma, burns, intra-arterial drugs) or capillary pressure (exercise, venous obstruction, muscle hypertrophy, infiltration of infusions, nephrotic syndrome).

Fractures are the cause in some 75% of cases, and of these, fractures of the tibia are most commonly responsible. Comminuted fractures are more likely to give rise to ACS and this probably reflects the greater degree of force required to cause this type of injury. Indeed any high energy trauma is more liable to cause ACS, and penetrating injuries such as gunshot wounds often cause severe muscle laceration and arterial tears, which in turn lead to increased intracompartmental pressure. ACS has been recorded in patients with no history of trauma, although such cases appear to be extremely uncommon (Vanneste et al, 2003).

It must be borne in mind that ACS can affect the hand, foot, forearm, thigh, lower leg and rarely the abdomen.

Pathophysiology

Ischaemia

There are three theories about the development of ischaemia:

1. It is the result of arterial spasm caused by increased compartment pressure
2. The theory of critical closing pressure. Because of the small diameter and high mural tension in the arterioles, a significant transmural pressure difference (arteriolar pressure – tissue pressure) is required to maintain patency. If tissue pressure increases or arteriolar pressure decreases so that this difference does not exist, i.e. critical closing pressure is reached, they will close (Burton, 1951)

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3. Because of their thin walls, veins will collapse if tissue pressure exceeds venous pressure. If blood continues to flow from capillaries, the venous pressure continues to increase until it exceeds the tissue pressure and venous drainage is re-established. However, this reduces the arteriovenous gradient and reduces arterial inflow.

Response of muscle to ischaemia

Histamine-like substances are released that dilate the capillary bed and increase endothelial permeability. This leads to intramuscular transudation of plasma with red cell sludging and decreased microcirculation. The muscle gains weight (up to 50% increase). Muscles tolerate 4 hours of ischaemia well, but by 6 hours results are uncertain and after 8 hours, the damage is irreversible (Whitesides and Heckman, 1996).

Diagnosis

In the conscious patient, the main clinical features of ACS are divided into early and late signs and symptoms.

Early

These include pain, disproportionate to the trauma to that limb, aggravated by passive muscle stretching, palpable tightness and tenderness of the compartment, and sensory deficit in the distribution of any sensory nerve traversing the involved compartment.

Late

Paraesthesia, weakness of the muscles and pulselessness are late signs and should not be waited for.

ACS is a clinical diagnosis which can be confirmed by measuring the compartment pressure. There are single use probes available but one can be fashioned using a 14G cannula with slit holes in the tip and an arterial line manometer, the commonest manometer available in anaesthetic rooms. A difference of less than 30 mmHg between tissue pressure and the diastolic pressure indicates the need for fasciotomy (McQueen, 1998).

Treatment

Once diagnosis has been made, sensible recommendations have been made by Mars and Hadley (1998). The patient should be kept normotensive as hypotension reduces perfusion pressure and facilitates further tissue injury. Circumferential bandages should be removed. The limb should be maintained at the level of the heart, as elevation reduces the arteriovenous pressure gradient on which perfusion depends, and oxygen should be administered. If a plaster of Paris cast has been applied, this should be split immediately as compartmental pressure falls by 30% when a cast is split on one side and by 65% when the cast is spread after splitting. Splitting the padding reduces it by a further 10% and complete removal of cast by another 15% (Garfin et al, 1981).

Fasciotomy is the definitive and only treatment for ACS. Morbidity from delay is significant and so fasciotomy should be performed immediately (Finkelstein et al, 1996). Thorough debridement of all non-viable tissue including skin should be carried out.

Fasciotomy is associated with a minor risk of wound infection but limb loss and death results from persisting ischaemia, muscle necrosis and multiorgan failure, not from infection of fasciotomy wounds (Rush et al, 1989).

Knowledge of the compartments of the affected limb is paramount to ensure an effective surgical procedure and fasciotomies should routinely be left open and assessed again in theatre 48 hours later. Primary closure is often difficult and split-skin grafting is frequently necessary. If ACS has existed for more than 8–10 hours, supportive treatment of acute renal failure should be considered.

Complications

The prognosis of ACS depends upon a number of factors. These include most importantly the rapidity with which it is diagnosed and treated, and whether or not complications occur.

Infection is a serious complication of ACS, and is more likely in cases where decompression is delayed. When it occurs, it increases the likelihood of amputation. Systemic complications include renal failure, adult respiratory distress syndrome and disseminated intravascular coagulation.

Untreated ACS results in Volkmann's ischaemic contracture, a disabling deformity of the limb.

Cosmetic disfigurement may occur from the surgical procedures involved in treatment.

Chronic compartment syndrome

Chronic compartment syndrome (CCS), also known as exertional compartment syndrome, is characterized by exertional limb pain, swelling and dysaesthesia during and immediately after exercise. It is mostly seen in athletes and military recruits. The pain gradually worsens as exercise continues, ultimately restricting performance.

Although this condition usually involves the lower limb, rare cases do occur in which the upper limb is primarily affected, e.g. in weight lifters. Upper limb symptoms may be reproducible at a specific workload or time interval. Symptoms generally subside within an hour or so of stopping the activity but recur when exercise is resumed.

Muscle weakness of the affected limb may be a feature of these episodes and gradually increasing fullness is a frequent complaint. Pain is increased both on passive stretching and active contraction.

The clinical features of CCS are only evident in the immediate aftermath of exercise, and the nature and location of signs and symptoms will depend on the compartment affected.

Pathophysiology

The pathogenesis of this condition is uncertain, although it probably resembles that of ACS in that structures within a closed myofascial compartment are compressed. During exercise, muscle bulk increases some 20% and, allied with repetitive muscle contraction, may increase intracompartmental pressure to a level which causes transient ischaemia and deoxygenation (Mohler et al, 1997).

Symptoms of CCS probably occur when the pressure between successive muscle contractions remains high within a small unyielding fascial compartment (Schissel and Godwin, 1999).

An alternative explanation is that muscle tissue, damaged by repetitive hard surface exercise, releases protein-bound ions which increase osmotic pressure, provoke oedema and so decrease blood flow within the compartment.

The predisposing factors and pathophysiology of CCS are imperfectly understood and although cases have been reported in which the condition appears to have originated at the time of some minor trauma, such instances appear to be rare (Tubb and Vermillion, 2001).

Each compartment of the leg contains one major nerve. The anterior compartment contains the deep peroneal nerve, the lateral compartment contains the superficial peroneal nerve, the deep posterior compartment contains the posterior tibial nerve and the superficial posterior compartment contains the sural nerve. When the pressure in a compartment increases, the vascular supply to the nerves can be affected causing paraesthesia to occur.

Diagnosis

No abnormalities are usually evident unless the patient is seen immediately after exercise.

Muscle atrophy may be noticed. Tenderness and increased tension in the involved compartment is frequently present. Tenderness directly over the tibia, however, is more characteristic of a stress fracture, tibialis posterior tendonitis or periostitis.

Passive stretching of the involved muscle after exercise may increase pain. The key to diagnosis is compartment pressure measurements. A resting pressure must be measured first which is then followed by exercise, during which sequential measurements are taken at specific time intervals, culminating in final readings 5, 10 and 20 minutes after exercise to ensure pressure has returned to baseline. It is very important that symptoms are elicited during this process and measurements taken at intervals until they subside. A positive indication of CCS is post-exercise pressures in excess of their insertional pressure and/or pressures in excess of 15 mmHg persisting for longer than 15 minutes. Definitions for elevated pressures are pre-exercise, >14–15 mmHg; 1 minute post-exercise, >30 mmHg, 5 minutes post-exercise >19–20 mmHg.

Treatment

Although there have been reports of successful treatment of CCS by conservative means, massage and physiotherapy alone are rarely satisfactory and fasciotomy is the treatment of choice (Brennan and Kane, 2003; Fraipont and Adamson, 2003).

Surgical intervention has a generally satisfactory outcome (Turnipseed, 2002) with some 70–85% of patients able to return to pre-treatment levels of activity without symptoms. The success rate depends largely on the compartment concerned, and patients with lower limb CCS in whom the deep posterior compartment is affected respond less well than those whose anterior or lateral compartment is involved. Cases of recurrent CCS have been reported, almost certainly attributable to scarring and closure of the initial compartment release.

Complications

Complications including inability to return to premorbid performance levels, weakness in muscles of the affected compartment and recurrence of symptoms may be noticed.

Conclusions

ACS and CCS may have linked pathophysiology but they occur in very different clinical settings. ACS is usually associated with a fracture but not exclusively. It is a serious limb-threatening condition and delay in treatment of ACS increases morbidity and complications. ACS can occur in open fracture injuries.

CCS most commonly affects the lower extremities in competitive athletes, and is probably caused by raised pressure within a non-compliant myofascial compartment as a result of repetitive muscle activity, causing symptoms during and immediately after exercise. Diagnosis is more complicated but less urgent than ACS. BJHM

Conflict of interest: none.

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KEY POINTS

- Acute compartment syndrome is a condition which occurs when increased tissue pressure within a myofascial compartment compromises the vascular supply and the function of structures within that space.
- Acute compartment syndrome can affect the hand, foot, forearm, thigh, lower leg and rarely the abdomen; fractures are the cause in some 75% of cases.
- Diagnosis of acute compartment syndrome is mainly a clinical one which may be supported by compartment pressure measurement
- Chronic compartment syndrome is characterized by exertional limb pain, swelling and dysaesthesia during and immediately after exercise.
- Diagnosis of chronic compartment syndrome is both clinical, mainly from the history and physical, from compartment pressure measurements before, during and after exercise.

Pseudosubluxation of the cervical spine

A 5-year-old boy presented to accident and emergency with torticollis and painful swallowing. There was a recent history of an upper respiratory tract infection (URTI) and an unwitnessed fall from a ladder. A lateral X-ray of the cervical spine showed apparent subluxation at C2/3 and a widened pre-vertebral space. A fracture-dislocation of the cervical spine was suspected, however, a computed tomography scan revealed a retropharyngeal abscess with no evidence of cervical spine abnormality. The abscess was drained transorally with complete resolution of the torticollis and odynophagia.

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Pseudosubluxation is a recognized radiological finding in children and may lead to diagnostic uncertainty. It most commonly affects the C2/3 junction and occurs as a result of ligamentous laxity and relative horizontal alignment of the apophyseal joints.

The posterior cervical line is used to help differentiate pseudosubluxation from the displacement seen with a hangman's fracture of C2 (Swischuk, 1977). This is a line drawn from the cortex of the posterior arch of C1 to the posterior arch of C3 (arrowheads in *Figure 1*). In pseudosubluxation the line passes within 1 mm of the cortex of the posterior arch of C2. In this case the primary pathology was the retropharyngeal abscess, presumably secondary to the URTI, and the trauma was irrelevant. BJHM

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Figure 1. Note anterior displacement of the body of C2 on C3, in the presence of a normal posterior cervical line (arrowheads).