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Acute compartment syndrome in the limb

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Abstract

Compartment syndrome of the limb is a true orthopaedic emergency that warrants prompt evaluation and treatment. Acute compartment syndrome of the limb is not uncommon and has the potential to cause devastating morbidity and mortality. Failure to provide urgent surgical intervention once established can lead to irreversible tissue damage within hours of onset.

Compartment syndrome can occur across all limbs, the buttocks and even the abdomen, but this article focuses solely on the diagnosis of acute compartment syndrome of the limb. Acute compartment syndrome can have a wide range of causes, with trauma representing approximately 70% of cases.

Key words: Compartment syndrome; BOAST guidelines; Trauma; Orthopaedics; Orthopaedic emergency; Fasciotomy

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Introduction

Compartment syndrome of the limb is a true orthopaedic emergency that warrants prompt evaluation and treatment (Donaldson et al, 2014). Acute compartment syndrome of the limb is not uncommon and has the potential to cause devastating morbidity and mortality. Failure to provide urgent surgical intervention once established can lead to irreversible tissue damage within hours of onset.

The earliest mention of acute compartment syndrome was first given by German surgeon Richard von Volkmann in 1881 who described (Volkmann's) paralytic contracture secondary to ischaemia in elbow fractures (von Volkmann, 2007). Since then, research has shown that this catastrophic process can occur across all limbs, the buttocks (Liu and Wong, 2009) and even the abdomen (Patel et al, 2007). However, this article focuses solely on the diagnosis of acute compartment syndrome of the limb. In the UK, the British Orthopaedic Association has created a framework, alongside the British Association of Plastic, Reconstructive and Aesthetic Surgeons and the Royal College of Nursing, that guides the initial assessment and management of this condition (British Orthopaedic Association et al, 2014).

Acute compartment syndrome can occur as a result of a wide array of aetiologies, with trauma representing approximately 70% of cases. The average incidence of acute compartment syndrome is 3.1 per 100 000. The incidence in men is 10 times higher than in women (7.3:0.7 respectively) (Wright et al, 2011). This discrepancy is mainly attributed to a high prevalence in men below 35 years of age (McQueen et al, 2000).

Pathophysiology

Although it remains contentious, the prevailing theory is the arteriovenous pressure gradient theory (Via et al, 2015), whereby excessive interstitial pressure leads to a reduction in capillary or tissue perfusion and ultimately tissue ischaemia.

Tissue perfusion is dependent on the gradient between capillary perfusion pressure, which is usually 8 mmHg, and the surrounding tissue pressure; adequate arterial oxygen delivery and subsequent venous drainage is determined by this gradient. Acute compartment syndrome occurs when this pressure gradient is disrupted by internal factors within a non-elastic compartment, such as bleeding, or external compression of the tissue, for example constricting casts or bandages.

Within the limb, skeletal muscle is separated into anatomical segments and is enveloped by a dense fibrous sheath known as the fascia. Each segment contains a muscle group and

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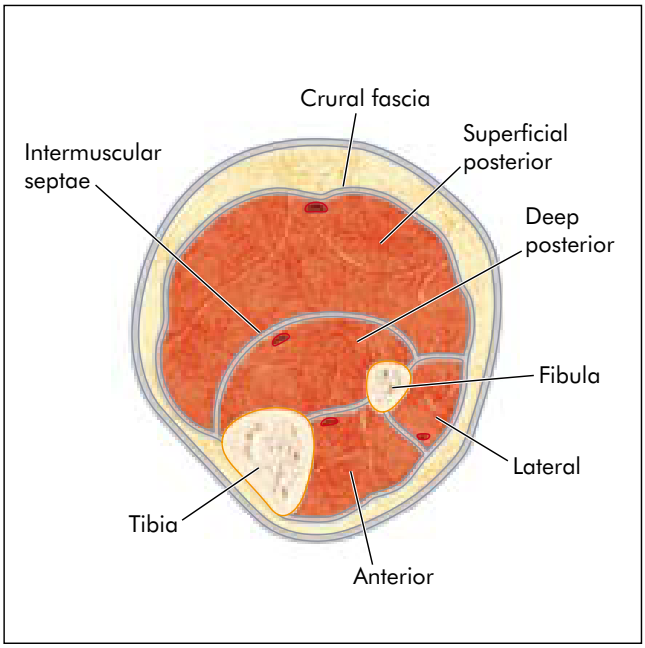

Figure 1. The four compartments of the lower leg: anterior, superficial posterior, deep posterior and lateral.

associated neurovascular structures to form a closed non-elastic compartment (Figure 1). The number of compartments in each limb is as follows:

- Brachium (3: anterior, posterior, deltoid)
- Forearm (3: volar (most commonly affected), dorsal mobile wad, lateral)
- Hand (10: hypothenar, thenar, adductor pollicis, dorsal interosseous (x4), volar (palmar) interosseous (x3))
- Thigh (3: anterior, posterior, adductor)
- Foot (9: medial, lateral, interosseous (x4), central (x3)) (Donaldson et al, 2014).

When the intracompartmental pressure rises within the fixed non-elastic tissue, it eventually exceeds the capillary perfusion pressure leading to capillary collapse. Moreover, the decrease in venous drainage leads to pooling and extrusion of fluid into the third space. In addition, tissue hypoxia leads to increased capillary membrane permeability, further increasing the intracompartmental pressure. If left untreated, this vicious cycle continues until intracompartmental pressure exceeds the capillary pressure, venous pressure (30 mmHg) and finally arterial pressure, at which point the tissue becomes completely ischaemic. Irreversible tissue damage can occur within 4 hours of ischaemia.

Assessment

History

Classically, acute compartment syndrome is a rapidly evolving condition associated with recent trauma such as bleeding from a tibial fracture. However, it can be secondary to any condition that causes rapid filling of the compartment (Table 1).

Identification of patients presenting with acute compartment syndrome should not solely depend on symptomatology once it has occurred; a thorough clinical history and examination should be used pre-emptively to stratify high-risk patients. This should include:

- Time and mechanism of injury
- Time of evaluation
- Level of pain (SOCRATES)
- Level of consciousness
- Response to analgesia
- Regional anaesthesia
- Medical history
- Social history.

Table 1. Causes of compartment syndrome

Cause	Example
Orthopaedic	Tibial fracture
	Total knee replacement
	Blunt or penetrating trauma
Vascular injuries	Reperfusion injury
	Arterial puncture
	Haemorrhage
	Deep vein thrombosis
Soft tissue injuries	Burns
	Rhabdomyolysis
	Crush injury
	Contusion
	Envenomisation
Iatrogenic	Casts and bandages
	Anticoagulants
	Medication and fluids
	Prolonged tourniquet use
Other	Intravenous drug use
	Diabetic infarction
	Myositis
	Bleeding disorders

From Mar et al (2009)

Assessment for acute compartment syndrome should be part of the routine evaluation of patients following significant limb injuries, patients who have undergone surgery for limb trauma, and in patients following any prolonged surgical procedure which may result in hypoperfusion of a limb.

Despite being an acute condition, acute compartment syndrome can occur up to 48 hours post-trauma or spontaneously depending on the cause.

Patients with open fractures should not be overlooked, as disruption of the fascia secondary to the fracture does not sufficiently decompress the compartment.

Examination

All patients should undergo complete physical examination, with a detailed inspection of the affected limb.

Conventionally, acute compartment syndrome presents with the six cardinal 'P's:

1. **Pain**. Severe, increasing pain, that is out of proportion with the diagnosis, is the most common and sensitive presenting symptom. This is often the only symptom that occurs before irreversible damage. As compartment pressure exceeds capillary pressure, oxygen perfusion from the capillary to its associated nerve is halted, resulting in nerve ischaemia, which is experienced as excruciating pain. Pain will be exacerbated when the affected compartment is placed under tension, which can usually be achieved by passive stretching. Table 2 highlights the expected examination findings in the lower limb.
2. **Paraesthesia/paresis**. This can be a varied finding depending whether the patient has an acute nerve injury or any pre-established neurological changes. Light touch and

Table 2. Examination findings in below-knee acute compartment syndrome

	Anterior	Lateral	Superficial posterior	Deep posterior
Muscles	Tibialis anterior extensors: ■ Hallicus longus ■ Digitorum longus	Fibularis longus Fibularis brevis	Superficial flexor: ■ Soleus ■ Gastrocnemius ■ Plantaris	Deep flexor: ■ Fibialis posterior ■ Fibularis: hallicus longus ■ Digitorum longus
Artery	Anterior tibial	Peroneal	Posterior tibial	Posterior tibial and peroneal
Nerves	Deep peroneal	Superficial peroneal	Tibial	Tibial
Effect of injury	Sensation of great toe web space Dorsiflexion of the foot, lateral four toes and great toe (extensor hallicus longus test) Foot inversion: dorsalis pedis pulse	Sensation: anterolateral, shin, dorsum foot Foot plantar Flexion and eversion	Foot plantar flexion	Sensation: plantar aspect of the foot Plantar flexion of the great toe and lateral four toes Foot inversion Posterior tibial pulse

From Pechar and Lyons (2016)

two-point discrimination are usually the first to be affected, and further neurological change or paralysis is usually indicative of late stage acute compartment syndrome.

3. **Paralysis**. Once tissue necrosis and irreversible damage has occurred, this symptom tends to present later in the course of compartment syndrome.
4. **Pulselessness.** Capillary refill rate and distal pulses should be clearly documented, but do not contribute to early diagnosis of the condition. The traditional presentation of pulselessness does not occur until intracompartmental pressure has surpassed arterial pressure, so the presence of a pulse is a poor indicator of tissue perfusion.
5. **Pallor**
6. **Poikilothermia.**

Pitfalls

When initially assessing a patient, there is a risk of compartment syndrome being overlooked in the following:

- An unconscious or uncooperative patient
- Patients who have had a previous neurological or vascular injury
- Patients who have received excessive analgesia.

It is essential to consider compartment syndrome in the above patients who have risk factors but may not present in the traditional manner described above.

Investigations

Blood tests

There are no specific pathology screening tests for acute compartment syndrome, but creatinine kinase levels should be examined as these may be raised in reperfusion injury and rhabdomyolysis. Blood tests should focus mainly on assessing baseline functions and preparing patients for potential surgery. Full blood count, biochemistry, coagulation studies and group and save with cross-match should all be requested.

Radiology

Radiographs should be taken of the afflicted limb. Although acute compartment syndrome cannot be assessed on an X-ray, it is important to try and identify the underlying cause which is often a fracture.

Urine analysis

As mentioned, where rhabdomyolysis is suspected, urinary monitoring may help in the diagnosis and management of the patient. Urinary myoglobin can be measured, fluid status should be diligently documented, and fluid output maintained.

Key points

- Acute compartment syndrome is primarily a clinical diagnosis, clinicians should identify high-risk patients before symptoms and should not delay urgent senior review if suspected.
- Do not be tempted to prescribe excessive analgesia, as pain may be the only overt symptom.
- Suspected patients should be closely monitored. Once diagnosed, the patient should be transferred to theatre immediately for urgent decompression. Delayed fasciotomy is the most important factor in poor outcomes.
- Time is the most critical factor – irreversible tissue damage can occur within 4 hours.

Intracompartmental pressure measurement

If clinical examination yields unreliable results, intracompartmental pressure can be measured using various handheld devices, which can be useful diagnostic tools when available (Köstler et al, 2004). Critical pressure is reached when the difference between the intracompartmental pressure and systemic diastolic blood pressure is below 30 mmHg and warrants urgent senior review. In the absence of other life-threatening conditions, a patient with an absolute intracompartmental pressure over 40 mmHg with clinical symptoms should be considered for urgent decompression.

The sight port needle of the handheld device should first be placed perpendicularly into the suspected compartment, although all compartments in the anatomical segment need testing to give an accurate reading. A small volume of saline is injected, forming a continuous column from tissue to device. Pressure exerted from the compartment is propagated through the column and this pressure is measured by the device.

Management

Patients at risk of acute compartment syndrome should have routine hourly nursing observation and early signs should be recorded. If pain scores remain high or worsen despite judicious analgesia, senior clinical review is vital. Regional and patient-controlled anaesthesia should be avoided, as this may mask early symptoms. Accurate documentation of the exact rate and usage of analgesia is central to good clinical care.

Any form of restrictive bandages, casting or dressing should be removed. The limb should be placed level with the heart and the patient should be reassessed frequently for signs of improvement.

In cases with high clinical suspicion of acute compartment syndrome, the patient should be prepared as if for potential surgery. Intravenous access should be established, recent pathology tests should be reviewed, and blood products should be available. The patient should remain nil by mouth until surgery or until the patient is no longer considered at risk.

If the decision to treat has been made, surgical decompression via fasciotomy alone will suffice. Surgical decompression should not be postponed.

Surgical management involves open fascial decompression of all potentially involved compartments, taking into account possible reconstructive options. Necrotic muscle should be excised. Accurate documentation of the incision and all decompressed compartments must be recorded. All patients should undergo re-exploration at approximately 48 hours, or earlier if clinically indicated. Frequent limb assessment should continue throughout this process.

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Conflicts of interest

The authors declare no conflicts of interest.

Curriculum checklist

This article addresses the following requirements from the specialist training in trauma and orthopaedics curriculum:

- Should demonstrate knowledge of management of vascular injury including compartment syndrome
- Should be able to perform fasciotomies for compartment syndrome
- Recognition and treatment of acute compartment syndrome
- Principles of postoperative management and recognition of developing complications including compartment syndrome.

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