Acute compartment syndrome of the lower extremity: an update

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Abstract

Acute Compartment Syndrome (ACS) represents a limb threatening condition characterised by increased intracompartmental pressure and decreased tissue perfusion leading to cellular anoxia, muscle ischemia, and death. Musculoskeletal trauma, as well as other medical conditions, initiate this syndrome. Most commonly the lower leg is involved. Basic science data shows the involvement of reactive oxygen metabolites in the development of this clinical entity. Diagnosis is principally clinical, reportedly delayed by certain anaesthetic techniques, such as nerve blocks and other forms of regional and epidural anaesthesia. Measurement of the intracompartmental pressure is required for the confirmation of the syndrome. Complete fasciotomy of all compartments involved is mandatory to reinstate perfusion to the affected tissues. Recognising a compartment syndrome requires a high index of suspicion, accurate evaluation. Early treatment.prevents irreversible damage and subsequent disability as well as avoiding medico-legal problems.

Keywords compartment; fasciotomies; intracompartment pressure; tibial fractures

Introduction

Acute compartment syndrome (ACS) is a true orthopaedic emergency representing a unique form of ischaemia that affects a group of muscles enclosed within a relatively non-compliant fascial sheath.[1–3]

ACS can occur as a result of any extremity fracture, crush injuryto a limb, prolonged tourniquet use under anaesthesia, compression by casts or dressings, burns, bleeding from trauma or anticoagulation therapy, soft tissue injuries or, from exercise in chronic compartment syndrome.[1–12] ACS may cause irreversible muscle or nerve damage, leading to a poor functional result.[13,14]

The most frequently affected site is the anterior compartment of the leg following a tibial fracture but it can also occur in the hand, forearm, foot, or proximal portions of the limbs.[15]

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The key element in its pathogenesis is an elevation of tissue pressure within encapsulated muscles as a consequence of increased vascular permeability and interstitial oedema formation. Elevated compartment pressure results in progressive skeletal muscle microvascular ischaemia with subsequent muscle cell death, loss of motor function, or loss of the limb. Evidence suggests that reactive oxygen metabolites may be decisively implicated in the occurrence of this clinical entity.[16]

The clinical diagnosis of compartment syndrome depends on the presence of pain, tenseness, swelling, and diminished motor and/or sensory function in the affected limb.

Even though some authors recommend that the diagnosis of compartment syndrome should rely on clinical symptoms,[17] others have highlighted the significance of intracompartmental pressure measurement.[18–20]

Nevertheless it remains a fact that the subjectivity of symptoms and the ambiguous nature of the clinical signs emphasize the necessity of intracompartmental pressure measurement in the diagnosis or exclusion of compartment syndrome. However there is no specific, reliable and reproducible test that could confirm the diagnosis of ACS.[21]

Delay in the diagnosis or treatment may result in permanent sequelae, including paralysis, painful dysthaesias, contractures, and occasionally loss of the limb. Thus, a systematic evaluation should include a repeatable physical examination and compartment pressure monitoring.

The definitive treatment for acute compartment syndrome is fasciotomy. This involves a skin incision, and splitting the fascia of the compartment to relieve pressure and allow tissue perfusion. Single or double incisions along the limb axis may be required. Failure to recognise the syndrome has significant functional consequences for the patient and may involve the surgeon in a malpractice litigation.[22] Thus, a knowledge of the pathogenesis, methods of evaluation and treatment are extremely important.

In this paper the current concepts of the pathophysiology, diagnosis, and treatment of compartment syndrome of the lower limb are discussed.

Historical aspects

The risks of elevated intracompartmental pressure have long been recognized. Hippocrates (ca. 400 B.C.) recommended: ''apply the linen bandage putting the head of it at the fracture so as to give support, but without much pressure''.[23] In the modern era, compartment syndrome was originally described by Richard von Volkman.[24] in 1881. He described a classic presentation of forearm muscle contractures following the application of a tight bandage for the treatment of an elbow fracture.

Jepson in 1926[25] described the effect of prompt decompression to prevent such a disastrous condition. Sir Robert Jones in 1928[26] concluded that ''pressure from both within or without, or both'' could be the cause. Bywater and Beall in 1941[27] reported on the victims of the London Blitz, highlighting the systemic consequences of severe crush injuries including renal failure and death.

In the early 1900s, interventional devices were used to measure pressure in the compartments. In 1975 Whitesides et al.[28] advocated the use of a simple pressure monitoring device employing a manometer, catheter, and needle. Owen et al.[29] contributed to our understanding of the pathogenesis of ACS in

their elegant description of 11 cases of crush syndrome following a period of prolonged compression. Matsen[30] suggested that the underlying features of compartment syndrome are the same, irrespective of aetiology or location. He described the role of compartment pressure measurement in ambiguous situations and the importance of prompt surgical decompression. The vivid account by Rorabeck and Macnab[31] deserves special reference. They pointed out the four main entities that could lead to the syndrome:post-exercise, post-traumatic, post-ischaemic and post-arterial occlusion.

Mc Queen and Court-Brown[32] more recently reported the role of Delta pressure as the critical determinant of need for decompression.

Incidence and aetiology

Musculoskeletal trauma and various medical conditions are linked to acute compartment syndrome (ACS) (Table 1).[21,32–34]

The commonest fractures associated with ACS are those of the tibial shaft[35] with an incidence of 40%,[21] and mainly involve the anterior and deep-posterior compartments.[36] The incidence after open tibial fractures varies between 1.2 and 10.2%.[8,35,37]

Lower limb and ankle joint position have been reported to contribute to the establishment of ACS by altering compartment pressure.[40]

ACS has been reported to occur also after isolated femoral fractures,[38] and after operative treatment of fractures with intramedullary nailing. However thigh compartment syndrome is seen more commonly in polytrauma patients and can be associated with various soft tissue injuries.[39]

Anticoagulation treatment in joint replacement surgery procedures has been implicated in the occurrence of ACS.[44]

Burns causing eschar formation and intersitial edema have been noted to cause ACS.[41]

Any revascularisation procedure can result in ACS, due to tissue swelling following reperfusion, with its incidence ranging from 0 to 21%.[42]

The use of military antishock trousers (MAST) for abdominal or pelvic haemorrhage has been associated with lower extremity compartment syndromes, although the key factor seems to be the inflation pressure rather than the time duration.[43]

Main aetiologies of acute compartment syndrome of the leg

Closed Fractures[61,77]
Open fractures[8]
Nailing procedures [17,20,21]
Soft tissue injuries[51,75]
Casting material[71]
Lithotomy position[52]
Burns[41]
Revascularization[76]
Military Antishock Trousers[43]
Anticoagulation treatment[44]
Skin traction[81]

Table 1

Pathophysiology of ACS

Phillips[45] defined the pathophysiology of compartment syndrome as an insult to normal local tissue homeostasis that results in increased tissue pressure, decreased capillary blood flow, and local tissue necrosis caused by oxygen deprivation.

Tissue metabolism normally requires an oxygen tension of 5–7 mmHg. This is readily maintained by capillary perfusion pressure (CPP) of 25 mm which is well above the normal interstitial tissue pressure (IP) of 4–6 mm.

The tissue perfusion pressure equals capillary perfusion pressure minus interstitial pressure. As compartment pressure increases, progressive decrease in perfusion leads to ischaemia and necrosis which then triggers a chain of events including an increased capillary permeability due to toxins. Tissue ischaemia, a direct result of increased compartment pressure, is also compounded by:

1. Arterial spasm directly due to increasing interstitial pressure.[46]
2. Effect of critical closing pressures on the arterioles.[47]

Due to a small luminal radius and high mural tension, arterioles naturally have high transmural pressure (arteriolar pressure minus tissue pressure). When the transmural pressure equals zero (due either to increasing tissue pressure or decreasing arteriolar pressure), the arterioles close (critical closing pressure [CCP] is reached) and ischemia ensues.[47]

3. The rising tissue pressure causes collapse of the veins as their walls are thin and susceptible. Initially the unabated arterial flow increases the venous pressure which re-establishes the flow, but the increased venous pressures adversely affect the arterio-venous gradient and result in ischemia.

When interstitial pressure exceeds CCP, capillaries collapse causing reduced perfusion, ischaemia, and cell death.[31]

The hypoxic injury to cells releases vaso-active substances, which increase the endothelial permeability. Subsequently an unabated shift of fluid occurs across the capillary endothelium into the extra-vascular space, causing high tissue pressure.[48]

Nerve conduction slows down as a result of ischaemia. Tissue pH falls and the degradation products contribute to a further increase in the tissue pressure. A vicious cycle of increased tissue pressure and ischemia ensues. Myocyte necrosis produces significant amounts of osmotically active particles (each milli-osmole of such substance has been showed to exert 19.5 mmHg pressure[8]) drawing large quantities of fluid into the tissues.[31]

Rorabeck[31] identified external pressure, trauma, and post-revasularisation swelling as the main causes of the pathological events leading to the compartment syndrome.

The involvement of neutrophils in arterial occlusion models of ischaemic skeletal muscle injury has also been reported.[49]The mechanisms by which neutrophils induce microvascular dysfunction are unclear. It is believed that neutrophils once activated can produce large quantities of oxygen metabolites, which are involved in the occurrence of ACS in rabbits during revascularization.[16] Furthermore, nicotinamide adenine dinucleotide phosphate (NADPH) oxidase and the enzyme xanthine oxidase are believed to be the main sources of oxidants in post-traumatic ischaemia.

Sadasivan et al.[50] showed that neutrophil reduction precludes microvascular dysfunction and muscle neutrophil infiltration. Furthermore, they correlated the occurrence of ACS with