Acute compartment syndrome of the extremities: an update

Ioannis V Papachristos

Peter V Giannoudis

Abstract

Acute compartment syndrome (ACS) remains a significant clinical problem with multiple health-related implications to the patient. It also carries intractable social and healthcare cost. Extensive research over the years has clarified its pathophysiology, however delays in diagnosis still persist leading to devastating consequences. Prompt and adequate fasciotomies remain the cornerstone of treatment. While diagnostic methods continue to evolve, clinical signs continue to be the most commonly used diagnostic tool for this condition. Evidence suggests that some cases are missed while many others may be overtreated. All trauma and orthopaedic surgeons will deal at least once in their career with this clinical entity affecting the extremities. Consequently, up-to-date awareness of current diagnostic and treatment developments and ongoing vigilance not to miss ACS is of paramount importance.

Keywords acute compartment syndrome; compartment syndrome; fascial envelope; fasciotomy; intracompartmental pressure

Definition

Compartment syndrome is characterized by an increase in the resting pressure in a contained fibro-osseous compartment, such as the forearm or leg, resulting sequentially in decreased lymphatic and venous drainage, loss of arterial inflow and subsequently diminishing of perfusion pressure. This leads to neuromuscular hypoxia and death of the contained structures. According to the above definition possible causes of compartment syndrome are those that increase the contents of a compartment (fractures, soft-tissue injury, crush syndrome, revascularization, fluid infusion, osteotomy, snake bite, acute haematogenous osteomyelitis and leukaemia) and those that can decrease the fascial volume of the compartment (casts, tight circumferential dressings, burns). Volkman described this entity 137 years ago but it still remains challenging to diagnose and effectively treat.[1] Established compartment syndrome if left untreated leads to contractures, sensory deficits, paralysis, permanent disability, amputation and even death. In order to minimize morbidity and optimize treatment of a patient at risk for compartment syndrome, clinicians need a clear understanding of the pathophysiology, means (and problems) of diagnosis, and treatment of compartment syndrome.

Incidence

According to The Royal Infirmary of Edinburgh series the annual incidence of acute compartment syndrome (ACS) is 3.1 per 100 000 people (7.3 per 100 000 men and 0.7 per 100 000 women, i.e. a 10-fold increase in men).[2] Tibial fractures accounted for 36% of cases with soft-tissue injuries being the second cause. In other series the most common fracture site associated with ACS in adults was documented to be the tibial diaphysis with a prevalence varying from 2.7% to 11% and with the forearm following. Tibial plateau and distal tibia fractures can also be complicated by ACS.[3] Age is a major risk factor for developing ACS. Patients younger than 35 years of age are more likely than older patients to develop ACS following the same type of injury. This can be explained as younger people have more muscle in a given compartment whereas older people usually have smaller musculature and also the protective effect of hypertension. Adolescents have a high rate of ACS (8.3%) after tibial fractures.[4] In this age group, individuals older than 14 years seem to carry the biggest risk.[5] Conversely, soft-tissue injuries lead to ACS in older ages as they tend to have more medical co-morbidities and to be anticoagulated.[6] It is also interesting that 61% of cases of non-fracture-caused ACS in children are iatrogenic (administration of infusions).[7] One could expect that open fractures would not be complicated by ACS as the compartment boundaries are disrupted. However, this is not true as open tibial diaphyseal fractures in particular can develop ACS with a reported incidence of approximately 3%.[2] Notably, it is believed that lower energy fractures are more vulnerable possibly because higher energy mechanism results in an 'autodecompression effect'.[8]

Summarizing, risk factors for developing ACS are considered male gender, young age, tibial fracture, high-energy forearm fracture, high-energy femoral diaphyseal fracture and anticoagulation.

Pathophysiology

Muscle injury causes precapillary arterioles to vasodilate and venules to collapse leading to increased permeability of the capillary bed, increased net filtration and raised interstitial fluid pressure. Physiological intra-compartmental pressure (ICP) is 8–10 mmHg in adults and 10–15 mmHg in children. Compartment syndrome occurs when the interstitial pressure within the compartment exceeds the perfusion pressure at the level of the capillary beds. Elevated hydrostatic pressure increases further the ICP leading to arteriole compression.[9] Once perfusion pressure reaches a critically low level, then tissue hypoxia ensues. Hypoxia, oxidative stress and tissue hypoglycemia cause shortage of ATP and cell oedema due to shutdown of the sodium-potassium ATPase channel which maintains physiological cellular osmotic balance. At the cellular level shortage of ATP correlates closely with worsening of muscle necrosis.[10] Chloride ions enter the cell due to the resultant loss of cell-membrane potentials leading to worsening of hypoxic state and eventually to a vicious circle.

Ioannis V Papachristos *MD Research Fellow, Academic Department of Trauma & Orthopaedics, School of Medicine, University of Leeds, UK. Conflicts of interest: none declared.*

Peter V Giannoudis *MD FACS FRCS Professor, Academic Department of Trauma & Orthopaedics, School of Medicine, University of Leeds and NIHR Leeds Biomedical Research Unit, Chapel Allerton Hospital, Leeds, UK. Conflicts of interest: none declared.*

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Please cite this article in press as: Papachristos IV, Giannoudis PV, Acute compartment syndrome of the extremities: an update, Orthopaedics and Trauma (2018), https://doi.org/10.1016/j.mporth.2018.05.005

On the other hand in the case of reperfusion injury once vascularity is restored after an extended period of ischaemia the production of oxygen radicals, lipid peroxidation and calcium influx lead to disturbances of mitochondrial oxidative phosphorylation and eventually cell-membrane destruction. Release of acidic and hyperkalaemic blood can lead to kidney failure, cardiac arrhythmias and even multiple organ failure and death.

The time from the initiating event to acute extremity compartment syndrome can vary from minutes to hours. Peripheral nerve tissue is affected early in ACS. Ischaemia of 2 hours leads to reversible neurapraxia, whereas 8 hours can lead to irreversible axonotmesis.[11] The amount of muscle necrosis is influenced by available residual blood flow, temperature and predominantly by type of muscle fibres and ischaemic duration. Increased collateral blood flow and decreased ischaemic temperature lead to decreased amounts of muscle necrosis. Red muscle fibres (aerobic metabolism) are more vulnerable to ischaemia (e.g. anterior compartment of leg) rather than white muscle fibres (anaerobic metabolism) (e.g. gastrocnemius-soleus complex).[12]

When ischaemia due to ACS persists for more than 6 hours, irreversible changes occur initiating an irreversible inflammatory cascade leading to fibrosis in necrotic muscle tissue. In summary, the degree of skeletal muscle injury correlates directly with the severity and duration of ischaemia. (Table 1).

Risk factors for delay in diagnosis are patient-controlled analgesia, regional anaesthesia, unconscious patient, children and associated nerve injury. In critically ill patients high base deficit, high lactate levels and high transfusion levels have been found to be correlated with ACS presentation.[13]

Methods of diagnosis

A coenzyme or biomarker specific to skeletal muscle ischaemia has not yet been identified. Moreover, inflammatory biomarkers such as creatine kinase, myoglobin, troponin, erythrocyte sedimentation rate (ESR) and interleukin-6 (IL-6) cannot specify the occurrence of compartment syndrome. Diagnostic options include the development of specific clinical signs, ICP measurements, infrared spectroscopy, pH monitoring, ultrasound and MRI.[14]

A prompt clinical diagnosis requires serial examinations by an experienced examiner. It is essential to look for the classic five 'Ps': pain out of proportion, paraesthesia, paralysis/paresis, pulselessness and pallor.[15] Pain criterion involves severe pain out of proportion for the given injury, pain that does not respond to analgesia, resting pain and pain on passive stretching of affected muscles. Pain may not be deemed reliable as its severity depends on psychological condition, variability of threshold in each individual, different expectations as well as cultural variety. It cannot be evaluated in unconscious or obtunded patients and where regional anesthesia was used. Its detection can also be troublesome in children or people with learning disabilities. In children the ACS presents with the three 'As': increased need for analgesia, anxiety and agitation.[16] Pain has a sensitivity of 19% with a specificity of 97% in diagnosis of ACS.[17] Paraesthesia is often the first indication of nerve ischaemia. Paraesthesia in the first dorsal foot webspace indicates ischaemia of the deep peroneal nerve due to increased pressure in the anterior tibial compartment. As time passes paraesthesia progresses to hypoesthesia and finally anaesthesia. Paraesthesia demonstrated a sensitivity of 13% and a specificity of 98% in diagnosing ACS.[17] Paresis/paralysis are often present in ACS but may be due to guarding secondary to pain or due to nerve injury. It is usually a late finding after prolonged ACS and irreversible muscle damage. However, Robinson et al published a series of 208 patients treated with intramedullary nailing of tibia where 5% developed dropped hallux and numbness in the first dorsall foot webspace but none of them developed ACS.[18] Pulselessness is a late finding of established ACS. ICP rarely is merely so high to occlude arterial inflow. When pulselessness and pallor are both present an arterial injury seems to be likely. Combination of the four 'Ps' (pain, pain with passive stretch, paraesthesia and paresis) has been associated with specificity and negative predictive value of over 97% but low sensitivity and low positive predictive value.[17] This means that the absence of symptoms is more useful in excluding ACS, than the presence of symptoms is for diagnosing ACS. Firmness of the compartment in manual palpation is another clinical sign but it is not considered reliable in diagnosing compartment syndrome. All the above show that even if we judge from clinical appearance we may not be able to diagnose an ACS when it is present or we may end up in delayed diagnosis. The need for diagnostic adjuncts has been obvious for some time. MRI can detect intracompartmental swelling but the finding is non-specific as it can appear in any injury. Scintigraphy and ultrasound also failed to prove useful diagnostic tools.[14] Realizing that the diagnostic approach to ACS should involve the measurement of the ICP as this is the originating cause, Whitesides et al in 1975 measured elevated pressure in a case.[19] Since then equipment has evolved and a number of different techniques have been described for ICP monitoring including the slit catheter, the side portal needle (Stryker needle), and a regular 18-gauge needle with a setup similar to an arterial line (Figure 1). When compared, there was no significant difference between compartment pressures measured by slit catheters and side portal needles. There was, however, on average a 20 mmHg increase in measured compartment pressures when using an 18-gauge needle.[20] Measurements should be taken in all affected compartments and at various timeframes and it is important to measure the peak pressure of the limb which usually occurs 5 cm distal and proximal to the fracture site (two measurements).[21] Great debate exists regarding the compartment pressure thresholds as indicators for decompressive fasciotomy. Some authors used as indicator absolute values of 30 or 45 mmHg but this does not seem reliable as mean or diastolic blood pressures vary and what matters is the perfusion gradient in the compartment rather

Time elapse and tissue survival

Tissue	Time elapse (hours)	Survival
Muscle	3–4	Reversible changes
	6	Variable damage
	8	Irreversible changes
Nerve	2	Loses nerve conduction
	4	Neurapraxia
	8	Irreversible changes

Table 1

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Please cite this article in press as: Papachristos IV, Giannoudis PV, Acute compartment syndrome of the extremities: an update, Orthopaedics and Trauma (2018), https://doi.org/10.1016/j.mporth.2018.05.005

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