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Current thinking about acute compartment syndrome of the lower extremity

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Acute compartment syndrome of the lower extremity is a clinical condition that, although uncommon, is seen fairly regularly in modern orthopedic practice. The pathophysiology of the disorder has been extensively described and is well known to physicians who care for patients with musculoskeletal injuries. The diagnosis, however, is often difficult to make. In this article, we review the clinical risk factors of acute compartment syndrome of the lower extremity, identify the current concepts of diagnosis and discuss appropriate treatment plans. We also describe the Canadian medicolegal environment in regard to compartment syndrome of the lower extremity.

Le syndrome compartimental aigu des membres inférieurs est un état clinique qui, même s'il n'est pas courant, se manifeste assez régulièrement en pratique orthopédique moderne. La pathophysiologie de ce trouble a été décrite en détail et elle est bien connue des médecins qui s'occupent de patients atteints de traumatismes de l'appareil locomoteur, mais le diagnostic est souvent difficile à poser. Dans cet article, nous passons en revue les facteurs de risque cliniques du syndrome compartimental aigu des membres inférieurs, nous décrivons les concepts courants du diagnostic et nous discutons des plans de traitement appropriés. Nous décrivons aussi le contexte médico-légal canadien en ce qui concerne le syndrome compartimental des membres inférieurs.

Acute compartment syndrome (ACS) of the lower extremity is a clinical condition that is seen fairly regularly in modern orthopedic practices. Although the pathophysiology of the disorder is well known to physicians who care for patients with musculoskeletal injuries, the diagnosis is often difficult to make. A delay in diagnosis that leads to a delay in treatment can result in permanent disability. Delayed or missed diagnosis can be devastating to both patients and members of the health care team.

RISK FACTORS

The most frequently quoted data about the predisposing factors for ACS come from the Royal Infirmary of Edinburgh.¹ The average annual incidence is 3.1 per 100 000 people (7.3 per 100 000 men and 0.7 per 100 000 women). The most common cause of ACS is fracture of the tibial diaphysis. About 36% of all cases of ACS are associated with tibial fracture. The second most common cause is blunt soft-tissue injury, which was reported in 23.2% of all ACS cases in the Scottish series.¹ The reported incidence of ACS following tibial fracture varies from 2.7% to 11%. The Edinburgh group found an incidence of 7%.¹

The pathophysiology of ACS involves an insult to normal tissue homeostasis within a compartment, which leads to increased tissue pressure, reduced capillary blood flow, local tissue hypoxia and local tissue necrosis. It can help to think of the possible causes of compartment syndrome as factors that can increase the contents of a compartment, those that can decrease the fascial volume of the compartment and metabolic insults that can disrupt the microvasculature (Box 1).

Age

Acute compartment syndrome occurs most frequently among young people. Physicians often believe that younger individuals are more likely than older individuals to have tight, strong fascia, that there is more muscle filling the compartment of younger people and that younger people are more likely to sustain an injury of significant energy and are thus at greater risk of ACS. These are all theories that may be true but have not been proven.

There seems to be a much higher incidence of ACS following tibial fracture in people under 35 years of age than in those aged greater than 35 years (Fig. 1). In the Edinburgh cohort,¹ there was a 30-fold increase in the likelihood of compartment syndrome following tibial fracture in patients under 35 compared with those over 35.

Open fractures

It was previously believed that an open fracture, by the nature of the disrupted skin and openings in the fascia, would decompress the compartment and prevent compartment syndrome. There is logic to this idea; however, the evidence does not support it. A series published in 1990 by McQueen and colleagues² prospectively reviewed 67 tibial fractures treated by intramedullary nailing and monitored the compartment pressure. In this and other series, there was no difference in pressure profiles between open and closed injuries.

Box 1. Possible causes of compartment syndrome

Conditions that increase compartment contents

- Fracture
- Soft-tissue injury
- Crush syndrome
- Revascularization
- Exercise
- Fluid infusion
- Arterial puncture
- Ruptured ganglia/cyst
- Osteotomy
- Snake bite
- Nephrotic syndrome
- Leukemia infiltration
- Viral myositis
- Acute hematogenous osteomyelitis

Conditions that reduce compartment volume

- Burns
- Repair of muscle hernia
- Circumferential dressings
- Casts

Medical comorbidities

- Diabetes
- Hypothyroidism
- Bleeding diathesis/anticoagulation

Traction

Traction has been reliably shown to increase the intracompartmental pressure (ICP) in injured limbs but this has not resulted in a consistent increase in ACS. Many groups have looked at ICP as a surrogate for ACS. However, ACS remains a clinical diagnosis: a conglomerate of signs and symptoms, one of which may be an increase in ICP.³

CLINICAL DIAGNOSIS OF ACS

Acute compartment syndrome can develop in any of the 4 compartments of leg; however, the anterior compartment is the most commonly affected and probably the most easily recognizable one. From an anatomic standpoint, this makes sense because it is the tightest compartment.

Optimum management of ACS following tibial fracture relies on early diagnosis and prompt surgical fasciotomy. Delayed diagnosis even for a couple of hours may result in serious irreversible complications, loss of limb or even death.

The initial diagnosis of ACS is based on the clinical presentation of the syndrome. This is very important but is not always enough or even possible for an early accurate diagnosis.

It is advocated that the main clinical symptom of a developing ACS is pain. Palpable tenseness, paresthesia, paresis and pulselessness may also be associated with compartment syndrome. The pain associated with a developing ACS is described as severe and out of proportion with the apparent injury, increasing with time and often resistant to analgesic medications. Although this distinctive pain can be an important and leading hallmark feature, it may not be useful in children who are unable to provide feedback and in adults who have an altered level of consciousness. Following an injury to the lower leg, pain from a tibial fracture and associated injuries may mask the pain of an impending compartment syndrome. Paresthesia and paresis do not develop until a significant compromise of flow and compartmental ischemia have already developed.

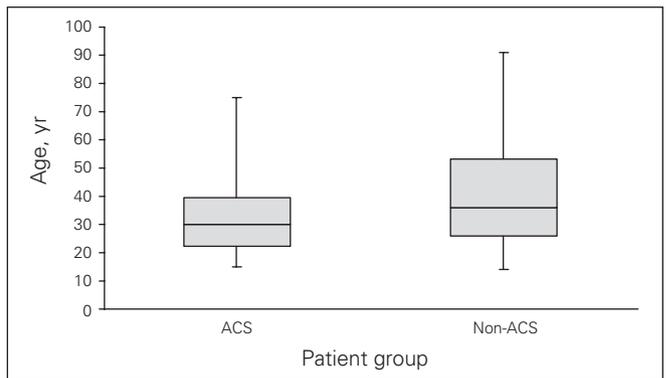


Fig. 1. Age distribution of patients with tibial fracture with and without acute compartment syndrome involvement (P.J. O'Brien, unpublished data, 2009). ACS = acute compartment syndrome.

Pulselessness and slow capillary refill are late signs of ACS and usually imply vascular injury rather than compartment syndrome.

Several studies have shown that the absence of clinical findings is more useful in excluding ACS than their presence is in confirming the diagnosis.^{1,3} Following a tibial fracture, evidence of progressive pain, paresthesia and functional losses are particularly important signals of a developing compartmental syndrome because all other symptoms and signs may be altered by direct trauma. Detection of this progression is obviously dependent on careful and sequential clinical examinations, preferably performed by the same clinician.

To confirm the clinical diagnosis of ACS, especially in difficult clinical situations, different diagnostic interventions have been used over the last few decades. In ACS, increased pressure within a compartment causes impairment of local blood flow, intracompartmental ischemia and hypoxia that eventually result in neuromuscular injury and dysfunction. Therefore, for diagnosis of ACS, any finding of increased ICP, intracompartmental ischemia, neuromuscular hypoxia and dysfunction should be taken into consideration. In fact, the diagnosis of ACS following tibial fracture requires a high index of suspicion and careful clinical evaluation as well as the monitoring of intracompartmental conditions by a reliable diagnostic method during the first 48 hours after fracture or surgical fixation.

The cascade of ACS following a tibial fracture starts with abnormal elevation of ICP in 1 or more compartments of the limb, most commonly the anterior compartment. The normal resting ICP is 0–8 mm Hg. A gradual increase of ICP results in a gradual decrease of local blood perfusion within the compartment. At a certain level of ICP, when it rises above the capillary blood pressure, intracompartmental blood circulation ceases. From this point, intracompartmental ischemia and hypoxia rapidly progress and compromise the viability of the muscles within the compartment. The first clinical symptoms of ischemia appear at an ICP of 20–30 mm Hg. At an ICP of 30–33 mm Hg, the fascial membranes reach their maximum tolerance of stretch, which limits the compliance of the compartment. Experts have advocated fasciotomy for absolute compartment pressures from 30 to 45 mm Hg.³ If the clinical observations are inconclusive, ICP measurement can help to confirm or exclude the diagnosis.

Compartment pressure measurement at a single point in time is the most widely used technique to assist in the diagnosis of ACS if the clinical diagnosis is in question. Currently, an ICP that is within 30 mm Hg of the patient's diastolic blood pressure ($\Delta p \leq 30$) is the most commonly accepted threshold for surgical intervention. There is some evidence that continuous monitoring of ICP is a safe and effective way of assessing patients who are at risk of developing ACS. Again, a $\Delta p \leq 30$ is the surgical threshold in the continuous monitoring protocol.³

RECENT ADVANCES IN THE DIAGNOSIS OF ACUTE COMPARTMENT SYNDROME

A coenzyme or biomarker specific to skeletal muscle ischemia has not yet been identified. Tibial fracture and ACS both result in early inflammation. Therefore, inflammatory biomarkers such as an elevated white blood cell count, erythrocyte sedimentation rate, creatine kinase, myoglobin, troponin I and fatty acid binding protein levels cannot specify the occurrence of compartment syndrome. Detection of a sensitive and specific biomarker for skeletal muscle ischemia would be a major advance in the diagnosis of ACS. Unfortunately, none are currently available.

The diagnostic value of imaging interventions in early detection and monitoring of ACS is limited. Magnetic resonance imaging (MRI) is able to detect soft-tissue edema and swollen compartments but cannot differentiate between the edema of affected muscles in compartment syndrome and that of soft-tissue injury after trauma.⁴ Scintigraphy is a radionuclide imaging intervention used to evaluate regional perfusion. There are some reports of the successful use of this method in the diagnosis of chronic compartment syndrome;⁵ however, its application in ACS is limited by the time required to perform this type of investigation, the potential lack of specificity in the traumatized limb and the inability to perform repeated or continuous examinations. Conventional and Doppler sonography of the geometry, echogenicity and perfusion of the affected muscles in the early diagnosis of ACS have not yet been successful.

There are some initial reports of the promising value of near-infrared spectroscopy in the detection of muscle deoxygenation in ACS.^{6,7} This is a noninvasive optical technique that can monitor the local muscle oxygenation and perfusion in a real time. This method uses a transcutaneous optode interface for the transmission of photons in the near-infrared spectrum into tissue and detection of those returning unabsorbed or unscattered by a receiver optode in order to monitor change in concentration of the chromophores oxyhemoglobin and deoxyhemoglobin, which have different absorption characteristics across the near-infrared spectrum.⁸ The interoptode distance determines the depth of penetration of near-infrared light. For a given interoptode distance, the region of maximum sensitivity for tissue interrogation is approximated as half the interoptode distance, which makes penetration to a depth of 30–40 mm possible, with 60 mm being the approximate limit for oxygen monitoring in the microcirculation.

With spatial configuration, this method can measure changes in local muscle oxygen saturation⁹ and thus has the potential to detect and provide continuous monitoring of intracompartmental ischemia and hypoxia. Near-infrared spectroscopy may, in the future, provide the benefit of a rapid, continuous, noninvasive tool that is sensitive and specific for the early detection of ACS. Technical

development and clinical validation of this diagnostic method are currently underway.

The early diagnosis of ACS is important. In spite of the drawbacks, clinical assessment is still the basis of the diagnosis of ACS. Measurement of ICP can assist in the diagnosis in suspected cases and may have a more important role in the diagnosis of this condition in unconscious patients and children. Access to an accurate, reliable and noninvasive method for early diagnosis would be a great development in orthopedic and emergency medicine. This requires refinement of the current gold standards as well as further investigations for developing new reliable methods.

LOWER LIMB FASCIOTOMY: TECHNIQUES

Once the diagnosis of ACS has been established, the treatment is urgent surgical decompression of the affected osseofascial compartments.¹⁰ The objective is to relieve the increased pressure, thereby reestablishing normal soft-tissue perfusion, and to debride any clearly necrotic tissue.

Two options are available for 4-compartment decompression of the leg: a single incision (parafibular) approach¹¹ or a 2-incision approach.

Double incision

This is the most commonly used technique and involves an anterolateral and posteromedial incision. The anterolateral incision is made midway between the anterior tibial crest and the axis of the fibula. It extends 5 cm from the fibular head to 5 cm from the lateral malleolus. The anterior compartment fascia is divided the full length of the leg. The anterior intermuscular septum is identified by palpation or by transverse incision of the fascia. The lateral compartment is then released over its entire length, avoiding injury to the superficial peroneal nerve as it exits through the fascia in the distal portion of the wound. The posteromedial incision is made 2 cm posterior to the posteromedial border of the tibia. Care must be taken to avoid injury to the saphenous vein and nerve in the subcutaneous tissue. The superficial and deep posterior compartments are decompressed by longitudinal incisions to the length of the tibia. The bulk of the musculature of the superficial compartment is in the proximal leg, in contrast to that of the deep compartment where it is distal. The soleus insertion must be released proximally to adequately decompress the proximal deep compartment (especially the tibialis posterior muscle) as needed.

Single incision

A single long incision is made over the fibular axis extending 5 cm from the fibular head to 5 cm from the lateral malleolus. The fascia is identified, and full-thickness cutaneous flaps are created anteriorly and posteriorly to access

the anterior, lateral and superficial posterior compartment fasciae. Care is taken to preserve perforating vessels to maintain perfusion to the skin flaps. The 3 compartments are decompressed with longitudinal fasciotomies, avoiding the superficial peroneal nerve as it exits the lateral compartment distally. The peroneal muscles are then raised off the posterior intermuscular septum until the latter's insertion on the posterolateral surface of the fibula. The intermuscular septum is then incised along this insertion over its full length to decompress the deep posterior compartment. The peroneal vein and artery are in proximity and are at risk of injury unless the dissection is kept close to the fibula.

Two points bear highlighting for both the single- and double-incision techniques. The first is that the length of the dermatomy should extend the length of the muscle bulk of the affected compartments. Skin acts as a circumferential restraint to expansion and has been shown to maintain increased ICP, despite full-length fascial release.¹² Second, adequate decompression must be ascertained by direct visualization of the muscle groups while passively moving the ankle and toes.

SKIN CLOSURE

Skin closure can begin once peak swelling has occurred and is resolving and all necrotic soft tissue has been excised following repeated débridement as necessary. This will vary from patient to patient, and clinical judgment is necessary. A number of options exist, but none have been shown superior in prospective comparative studies. Delayed primary closure is possible but often requires an extended delay to allow reapproximation with minimal or no skin tension. Split thickness skin grafting is a popular technique that, at times, requires eventual scar revision or resection for cosmetic reasons. Dermatotractor using skin staples along the wound margin and elastic cords (e.g., vessel loops) to gently bring the skin margins together as swelling resolves is another useful technique. Typically, once the margins are within 1 cm, elastics are no longer effective, and delayed primary closure can be accomplished.¹³ Lastly, vacuum-assisted wound closure has gained wide popularity in the management of these wounds with some suggestion that it hastens swelling resolution and skin closure.¹⁴ This technique may increase the probability of obtaining delayed primary wound closure or may facilitate successful split thickness skin grafting in cases that cannot be closed (Fig. 2).

MEDICOLEGAL ASPECTS OF ACUTE COMPARTMENT SYNDROME

A total of 91 (64 closed, 27 open) legal civil cases involving lower limb ACS occurring over a 10-year period (1998–2008) were identified in a review of Canadian medicolegal

cases performed for this article. These cases were divided into traumatic ACS (59 cases: 41 completed, 18 still in progress) and postprocedure ACS (32 cases: 23 completed, 9 still in progress). Traumatic cases were defined as cases in which compartment syndrome was related to a sports injury, fall or motor vehicle collision. Postprocedure cases were defined as those in which compartment syndrome developed following a surgical intervention unrelated to compartment syndrome or traumatic injury. Risk assessment was completed for each grouping.

Regardless of the mechanism of injury, ACS cases pose a high risk for members of the medical team and for patients. Fifty-five percent (35/64) of legally completed cases had an unfavourable outcome for the doctors involved with settlement (34) or judgment for plaintiff. Seventy-seven percent (63/82) of patients had a permanent physical disability (minor or major). Orthopedic surgeons were assigned responsibility with the highest frequency in both groups.

Young men (11–30 yr) involved in contact sports were identified as the highest risk group for ACS. Diagnostic issues related to other health care professionals and failure or delay in performing diagnostic tests or procedures were the most frequent clinical issues. Nurses failed to monitor patients, perform neurovascular checks and communicate changes to physicians. Doctors delayed or failed to perform fasciotomies, compartment pressure measurements, surgical repair of fractures and other diagnostic strategies.

There was no significant age or sex grouping for risk of postprocedure ACS. Orthopedic procedures (e.g., total knee replacement, osteotomies) and vascular surgeries (e.g., bypass grafts, abdominal aortic aneurysm repair and aortic valve replacement) were the most frequently documented interventions. Both performance and diagnostic clinical issues were identified. About 31% (5/16) of cases had both a diagnostic and performance critical incident documented; 25% (4/16) of cases had a diagnostic, perfor-

mance and communication critical incident. Patients in this group had documented comorbidity factors of smoking, cardiac disease, obesity, lung disease and use of anticoagulants.

Personnel caring for patients at risk of ACS must have a high level of suspicion, and systems must be in place whereby nursing staff regularly monitor patients and communicate any changes to the physicians. Physicians must carefully assess patients when symptoms develop, measure compartment pressures when necessary and proceed immediately to surgical treatment as soon as the diagnosis is made.

CONCLUSION

Acute compartment syndrome continues to be a serious complication of lower extremity trauma. Early diagnosis is essential and requires a high degree of vigilance by the care team. Clinical features remain the mainstay of diagnosis. Immediate fasciotomy once the diagnosis has been made is necessary to provide the patient with the best chance for a favourable clinical result. Despite awareness, delayed diagnosis and treatment continue to occur in modern orthopedic practice. There is a need for more research into new methods for early and reliable diagnosis of this potentially devastating condition.

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Fig. 2. Lateral fasciotomy wound temporary coverage with negative pressure wound therapy.

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