Acute Lower-leg Compartment Syndrome

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Abstract: Acute compartment syndrome remains a challenging problem for orthopedic surgeons because its diagnosis is not always straightforward and it has a high risk of associated limb morbidity if left undiagnosed or untreated. Failure to diagnose and treat acute compartment syndrome is one of the most common causes of successful medical liability claims. The authors review the current literature concerning the diagnosis of acute compartment syndrome and discuss new non-invasive technologies that may allow for earlier and more accurate diagnosis of impending acute compartment syndrome.

Acute lower-leg compartment syndrome is an evolving pathologic process. Early fasciotomy is of the utmost importance because definitive treatment through early and correct diagnosis is key in preventing morbidity. Classic cardinal signs for acute compartment syndrome have demonstrated less than optimal sensitivity but excellent specificity. The limiting envelope is most often the fascia but can be the epimysium or skin. Fascial compartments are inelastic and do not change in size after development is complete, but the muscle within is not limited in growth. Patients with increased muscle mass have less space for swelling after muscle injury.

The basic principle of ischemia stems from inadequate perfusion relative to demand. The ischemia produced by compartment syndrome is a self-perpetuating cycle involving increasing edema, pressure, and ischemia. Muscles and nerves tolerate ischemia for up to 4 hours with limited sequelae; however, 8 hours of ischemia results in irreversible damage. Fasciotomy allows for normalization of capillary blood flow and clears the local accumulation of anaerobic metabolite build-up via re-
trauma update

Etiology

The mechanism of injury for acute compartment syndrome ranges from high- and low-energy trauma to nontraumatic causes. The reported incidence of high- and low-energy trauma leading to acute compartment syndrome is approximately equal. McQueen et al reported that routine traffic accidents (involving both vehicle vs vehicle and vehicle vs pedestrian) were the most common causes of acute compartment syndrome, followed by sport-related injuries. Tissue-crushing injuries, falls, direct blows, burns, and penetrating injuries were among other reported traumatic mechanisms.

Epidemiology and Etiology

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Age and sex distributions indicated that men in their thirties have the highest likelihood of developing acute compartment syndrome, which may be explained by the relatively larger muscle mass in men within a fixed compartment size after growth is complete. McQueen et al reported that the average annual incidence of compartment syndrome for men was 7.3 per 100,000, with a mean age of 32 years, whereas the average annual incidence for women was 0.7 per 100,000, with a mean age of 44 years.

Fracture is the reason for initial presentation and a major contributing factor in approximately 75% of cases of acute compartment syndrome. Specifically, in a review of 164 patients with acute compartment syndrome, 113 (70%) patients had an associated fracture, with the most common fracture being tibial diaphyseal fractures in 59 (36%) patients.

Lower-leg acute compartment syndrome has been reported in 2% to 9% of tibial fractures. Park et al found that, depending on the location of tibial fractures, the incidence of acute compartment syndrome varied from 1.8% in proximal tibial fractures to 8.1% in diaphyseal fractures and 1.4% in distal fractures, with the predominance at the diaphysis because the majority of muscle mass surrounds this area. McQueen et al reported 68 tibial fractures with acute compartment syndrome, of which 59 were diaphyseal fractures, 5 were tibial plateau fractures, and 4 were tibial pilon fractures.

Hope and McQueen reported the first series of patients to develop acute compartment syndrome in the absence of a fracture and excluded crush syndrome as a diagnosis. They showed that these patients are typically older, have more comorbidities, and...
have an increased chance of delay to fasciotomy, leading to increased muscle necrosis at the time of fasciotomy, citing a low awareness for risk of acute compartment syndrome in an isolated soft tissue injury. Posterior compartment involvement is more common in acute compartment syndrome without a fracture.26

**Clinical Features and Diagnosis**

The “6 Ps” (pain, pallor, paresthesia, paralysis, and pulselessness), which were initially developed to describe the findings seen in vascular injuries, have been used to describe clinical signs associated with compartment syndrome. However, these clinical symptoms are subject to large variability and inconsistencies.27 Pulselessness and pallor are rarely associated with compartment syndrome unless an associated vascular injury or systemic hypotension occurs. Practitioners who are unfamiliar with the pathophysiology of compartment syndrome often place emphasis on the presence of pulses to incorrectly rule out compartment syndrome. The presence of paresthesia and paralysis represent late findings after acute compartment syndrome has likely been present for 4 hours or more.

Diagnosing compartment syndrome is difficult in clinical practice, even with the availability of intracompartmental pressure measuring, and it has been argued that there is no way to determine the true rate of compartment syndrome.28 In addition, the gold standard for diagnosis via measurement of compartment pressures has recently been questioned. Within a single Level I trauma institution, a significant variation was found in different surgeons’ rates of compartment syndrome diagnosis. Interestingly, when looking at individual surgeons, the “more commonly a surgeon decided to check compartment pressures, the more likely the surgeon was to perform fasciotomy,”28 which introduces the hypothesis that a high false-positive rate may exist when using compartment checks. In addition, Prayson et al29 prospectively looked at compartment pressures in lower-extremity fractures and showed that 84% of fractured extremities qualified for the diagnosis of compartment syndrome based on having a compartment pressure within 30 mm Hg of diastolic pressure. Prayson et al29 argued that “direct compartment measurement with existing thresholds and formulations to determine the diagnosis of compartment syndrome may not accurately reflect a true existence of the syndrome.” Current methods for direct measuring and monitoring of the compartment pressures

<table>
<thead>
<tr>
<th>Lower-leg Compartment</th>
<th>Muscle</th>
<th>Nervous</th>
<th>Vascular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>Extensor hallucis longus, extensor digitorum communis, tibialis anterior, peroneus tertius</td>
<td>Deep peroneal nerve</td>
<td>Anterior tibial artery</td>
</tr>
<tr>
<td>Lateral</td>
<td>Peroneus brevis and longus</td>
<td>Superficial peroneal nerve, proximal portion of deep peroneal nerve</td>
<td>Peroneal artery</td>
</tr>
<tr>
<td>Posterior superficial</td>
<td>Gastrocnemius, soleus, plantaris</td>
<td>Tibial nerve branches</td>
<td>Posterior tibial artery, popliteal artery, peroneal artery, sural arteries</td>
</tr>
<tr>
<td>Posterior deep</td>
<td>Poplitoeus; tibialis posterior, flexor hallucis longus, flexor digitorum longus, popliteus</td>
<td>Tibial nerve</td>
<td>Posterior tibial artery, peroneal artery</td>
</tr>
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**Figure:** Axial section of the lower leg. Abbreviations: AIS, anterior intermuscular septum; ATA, anterior tibial artery; ATV, anterior tibial vein; DPN, deep peroneal nerve; F, fibula; GSaV, great saphenous vein; IM, interosseous membrane; LSuCN, lateral sural cutaneous nerve; MSuCN, medial sural cutaneous nerve; PA, peroneal artery; PIS, posterior intermuscular septum; PTA, posterior tibial artery; PTV, posterior tibial vein; PV, peroneal vein; SaN, saphenous nerve; SPN, superficial peroneal nerve; SsaV, small saphenous vein; T, tibia; TIS, transverse intermuscular septum; TN, tibial nerve.
include slit catheters, side-port needles, and ultrafiltration catheters. Pressure measurement is done either by an arterial line set or another pressure-monitoring device. Contrary to previous studies questioning the usefulness of compartment pressure measurements, McQueen et al estimated the sensitivity and specificity to be high. In a large retrospective review of patients who sustained a tibial diaphyseal fracture and underwent documented continuous anterior compartment pressure monitoring, the sensitivity and specificity were estimated as 94% and 98%, respectively, and the positive and negative predictive values were 93% and 99%, respectively. The study diagnosed acute compartment syndrome only after 2 hours of continuous anterior compartment measurement with the differential pressure (diastolic-intracompartmental pressure) remaining higher than 30 mm Hg, and the authors recommended continuous monitoring over 1 single measurement.

Ulmer reviewed prospective studies on compartment syndrome and, despite variability in the clinical history and examination, was able to draw conclusions about the usefulness of the clinical findings. He looked specifically at pain, pain with passive stretch, paresthesias, and paresis in the literature and was unable to determine a consensus regarding which sign was of the greatest value due to a paucity of data. However, regarding use of these clinical findings in diagnosing compartment syndrome, the sensitivity was low (13% to 19%), the positive predictive value was low (11% to 19%), and both the specificity and negative predictive values were high (both 97% to 98%, respectively).

Pain out of proportion to the injury is often cited as an early sensitive sign, yet pain is often already present at varying levels in trauma patients. Pain with a passive stretch of the muscle in the area of the injury has been reported to be of greatest clinical value but has also been stated to be too subjective. Swelling and palpable tenseness may be early signs of compartment syndrome, but at best they are crude indications of acute compartment syndrome. It is critical to maintain a high index of suspicion in at-risk patients, and repeated serial examinations are necessary.

Magnetic resonance imaging (MRI) has been shown to identify chronic exertional compartment syndrome; however, the use of MRI in trauma settings is limited due to the time commitment of MRI vs the emergent nature of acute compartment syndrome. Edematous changes on MRI are partially due to the initial injury and, although MRI can show changes in late compartment syndrome, it is not effective in diagnosing early acute compartment syndrome.

Ultrasonography techniques are still in the early stages of development for monitoring compartment and perfusion pressures. Noninvasive ultrasound devices and pulse phase-locked loops detect slight movements in the fascia corresponding with arterial pulsation and distinguish between normal and abnormal intramuscular pressures. The benefit of this technology is that it is noninvasive and can be performed serially to produce a trend of intramuscular pressures. This may allow clinicians to detect impending acute compartment syndrome in its early stages. Near-infrared spectroscopy technology is noninvasive, measures local soft tissue oxygenation approximately 2 to 3 cm below the skin, and could provide continuous monitoring for intracompartmental hypoxia. Near-infrared spectroscopy has shown promise to become a sensitive and specific monitor of oxygenation of individual muscle compartments and is inversely correlated with increasing compartmental pressures. However, its limitations include use for patients with total body hypoperfusion and for obtaining measurements over a deoxygenated soft tissue hematoma. Interestingly, a lack of information exists in the literature on this subject, but measuring somatosensory-evoked potentials to detect nerve dysfunction may play a role in future noninvasive monitoring for compartment syndrome.

In a prospective study comparing different compartmental measuring devices, Collinge and Kuper reported that no single measurement should be used for determination for or against performing fasciotomy, recommending the necessity of clinical correlation in the diagnosis of acute compartment syndrome.

**TREATMENT, FASCIOTOMY, AND OUTCOMES**

Definitive treatment for acute compartment syndrome is emergent fasciotomy to decompress the compartments involved and prevent critical ischemia. Soft tissue viability is of immediate concern and has a narrow treatment window. As little as 8 hours of critical ischemia results in irreversible damage to compartmental muscles and nerves.

Both single- and double-incision fasciotomy techniques have been described for releasing the 4 compartments of the lower extremity. In the first analysis of single- vs double-incision fasciotomy techniques for tibial fractures in acute compartment syndrome, Bible et al showed similar infection and nonunion rates between the techniques and left the choice to the surgeon.

Surgical decompression is not always indicated if the compartment syndrome has been evident for more than 48 hours and no evidence exists of retained function of the components within the compartment. Timing of prophylactic fasciotomy is controversial, and outcome data comparing prophylactic vs therapeutic fasciotomy are retrospective and of mediocre quality. In a study of 94 patients (including trauma and vascular patients), Velmahos et al found higher complication and nonclosure rates in...
prophylactic fasciotomy cases. The authors make a valid point that prophylactic fasciotomy is not without major complications, and the risk-benefit ratio should be weighed heavily.

**Conclusion**

Acute compartment syndrome remains a true orthopaedic emergency. Despite a large amount of research and many articles discussing novel diagnostic tools, clinical examination is paramount, and the documentation of findings, discussion with patients and family, and treatment plan are essential. The authors believe that measurements of compartment pressures or other nonclinical diagnostic means should have no bearing on the urgent “get out of bed and take the patient to the [operating room].”

Alternative methods for diagnosing compartment syndromes have been attempted, but none have replaced high clinical suspicion and clinical examination. Review of the literature demonstrates the need for prospective, randomized trials comparing prophylactic and therapeutic fasciotomy, as well as additional investigation into reliable methods of diagnosing acute compartment syndrome.

**References**


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