

Acute Compartment Syndrome of the Lower Extremity: Update on Proper Evaluation and Management

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Abstract

Acute compartment syndrome (ACS) of the lower extremities is a common complication in patients with lower extremity trauma or ischemia. It has been associated with significant morbidity and complications, especially in cases with missed ACS or delayed treatment. This editorial aims to present a summary of current knowledge on epidemiology, diagnostics and management. Recent progress in intra-compartment pressure measurement as well as pooled results on major outcomes will be reported to assist future clinical practice.

Keywords: Acute; Compartment syndrome; Lower extremity

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Editorial

The incidence of acute compartment syndrome (ACS) of the lower extremities depends on the inciting event. Tibial fractures are the most common precipitating factor, accounting for 2-12% of all compartment syndrome cases, according to literature [1]. However, DeLee and Stiehl [2] have found that 6% of patients with open tibial fractures developed ACS compared with only 1.2% of patients with closed tibial fractures. The incidence of ACS is much higher in patients with an associated vascular injury. Feliciano et al. [3] reported that 19% of patients with vascular injury required a fasciotomy although the true incidence may not be always clear as a prophylactic fasciotomy is usually performed in such patients at the time of vascular repair.

Regarding pathogenesis, the pressure gradient from the pre-capillary arteriole to the post-capillary venule (ΔP) seems to play a primary role in causing an elevated intra-compartmental pressure (ICP), according to Poiseuille's law [1]. A diminished ΔP has two main implications, namely a decrease in the rates of both delivery of oxygenated arterial blood and drainage of deoxygenated venous blood. This establishes a vicious cycle of fluid extrusion into the muscle interstitium, tissue edema, collapse of lymphatics and ultimately tissue ischemia and necrosis [4]. Although Matsen and Krugmire [5] suggested almost 40 years ago that there is a 'critical closing pressure' above which capillaries collapse from transmural pressure and blood flow is arrested, Hastock et al. [6] have found that a pressure gradient between ICP and mean arterial pressure (MAP) arrests capillary blood flow. Therefore, the 'critical closing theory' has been disproved and it has been

suggested that the arterial-venous pressure gradient is the critical determinant of capillary blood flow. Finally, Heppenstall et al. have found that uninjured muscle in dogs develops evidence of tissue ischemia on magnetic resonance spectroscopy when the difference between MAP and ICP (MAP-ICP) drops below 30 mm Hg [7]. Comparing ICP criteria, McQueen and Court-Brown found that an absolute ICP threshold of 30 mmHg would have resulted in fasciotomy in 43% of patients, whereas a dynamic ICP threshold of 30 mmHg less than diastolic pressure resulted in only three fasciotomies [8]. These studies provide compelling data suggesting that a dynamic ICP threshold relative to MAP or diastolic pressure is a more appropriate criterion for selecting patients for fasciotomy.

ICP is elevated by conditions that either increase compartment volume or produce external compression. ACS may complicate up to 21% of cases of acute ischemia, with the ischemia/reperfusion phenomenon playing a crucial role in the pathogenesis [9]. Papalambros et al. [10] have identified several risk factors for

compartment syndrome after acute arterial ischemia including prolonged ischemia time (>6 h), young age, insufficient arterial collaterals, acute time course for arterial occlusion, hypotension, and poor back-bleeding from the distal arterial tree at embolectomy. Additionally, vascular trauma may produce ACS. In a recent meta-analysis of prognostic factors for amputation following surgical repair of lower extremity vascular trauma, ACS was found to be one of major factors leading to secondary amputation in such patients (Odds Ratio=5.11). Furthermore, the incidence of fasciotomy for trauma varies from 11.3% for blunt trauma to 28% for penetrating vascular trauma [11]. Iatrogenic interruption of venous flow, such as when harvesting the superficial femoral vein for use as a conduit for arterial reconstruction, is associated with the development of CS in 17.8% of limbs [12]. Concerning fractures, the incidence of ACS ranges from 1% to 29%, with age, male gender, blue-collar occupation, sporting injury, fracture classification, and treatment with intramedullary nails being major predictors for ACS [13]. Finally, crush injuries as well as iatrogenic causes have been implicated as potential triggers for ACS.

On examination, the most common findings are a tense, swollen compartment with pain elicited by passive movement of the muscles in the involved compartment. The pain is typically not relieved by immobilization or reduction of fractures and responds poorly to analgesic medications. Pain, however, should not be a *sine qua non* of the diagnosis. In severe trauma, such as an open fracture, it is difficult to differentiate between pain from the fracture and pain resulting from increased compartment pressure. Paresthesia represents an early symptom of ischemia of the nerves traversing the muscle compartment in question. However, decreased 2-point discrimination is a more reliable early test and can be helpful for setting the diagnosis [14]. In a meta-analysis by Ulmer concerning the clinical diagnosis of lower extremity CS, the author underlines that clinical findings have a low sensitivity (13% to 19%) for diagnosing CS, and the negative predictive value was high (97% to 98%) [15].

Regarding ICP measurement, clinical suspicion and examination is adequate in most of cases for ascertaining the diagnosis of ACS. Pressure measurement should be utilized for equivocal cases, unconscious patients or pediatric cases suspicious for ACS. Traditionally, several techniques and instruments have been proposed including arterial line manometer, hand-held Stryker system, and Whiteside manometer. According to Booddy and Wongworawat [16] who compared the various devices, side-port needles and slit catheters were more accurate compared to straight needles which tended to overestimate pressures. Lately, several other diagnostic techniques have been introduced and

evaluated in literature. Near-infrared spectroscopy, pressure related ultrasound, magnetic resonance imaging as well as diffusion tensor imaging have shown promising results as ancillary methods although they all need to be evaluated in larger *in vivo* studies for future clinical application [17].

Concerning treatment of ACS, fasciotomy has been the cornerstone of proper management for decades. However, a poor technique could lead to incomplete decompression, with the risk of permanent disability or limb loss. Clinical criteria for fasciotomy include a swollen, tense compartment; pain with passive motion of the muscle groups traversing that compartment; and neurologic findings referable to the compartment. Furthermore, some authors have advocated the 'prophylactic fasciotomy', especially in vascular surgery patients, producing satisfying results [10]. However, fasciotomy has been associated with certain complications such as impaired sensation at the margins of the wound, tethered tendons or recurrent ulcerations. A small subset of patients (7.5%) requires late amputation (between 1 month and 1 year), usually due to neurologically devastated limb or ischemia [18].

Finally, a delayed or missed ACS could have devastated consequences for the patient or the limb, increasing the risk for neurologic deficit and amputation. Potential complications of ACS include myonecrosis causing hyperkalemia, hypocalcemia, elevated liver enzymes, disseminated intravascular coagulation, and myoglobinuria [1]. Renal failure remains a rare complication and it usually occurs in victims of crush injuries. Delay of fasciotomy for more than 36 hours almost invariably results in amputation. In an old study by Sheridan and Matsen [19], the overall complication rate increased dramatically if fasciotomy delayed more than 12 h (54%) compared to early fasciotomy (4.5%). Furthermore, in a recent study by Farber et al. [20], late fasciotomy (>8 h) in patients with extremity vascular injury was found to be associated with higher amputation rate, higher infection rate and longer hospital stay compared to earlier fasciotomy (<8 h). As a result, after 3 to 4 days, decompression of CS is not indicated because the rate of infection and muscle necrosis is prohibitively high.

In conclusion, data so far indicate that clinical suspicion and examination remain the cornerstones for proper diagnosis of lower extremity ACS. However, novel techniques of pressure measurement are being developed to assist diagnosis and management in equivocal cases. Regarding treatment, early fasciotomy and prevention of potential complications of tissue ischemia and myonecrosis remain the recommended first line therapeutic strategy.

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