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ETIOPATHOGENESIS, DIAGNOSTICS AND TREATMENT OF ACUTE COMPARTMENT SYNDROME – CURRENT STATE OF KNOWLEDGE

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ABSTRACT

Introduction and purpose: Acute compartment syndrome (ACS) is a surgical condition marked by a critical elevation of intracompartmental pressure within a confined osseofascial space, resulting in impaired perfusion and subsequent risk of ischemic tissue injury. This article seeks to present a comprehensive overview of the contemporary understanding of ACS, including its pathophysiology, diagnostic evaluation, and treatment, with particular focus on associated clinical challenges and recent developments.

A brief description of the state of knowledge: Although ACS most frequently occurs secondary to traumatic injury, it may also develop in the absence of trauma, e.g., infections, bites, and prolonged limb compression. While classic clinical manifestations—such as disproportionate pain and swelling—are widely recognized, they lack specificity. Continuous measurement of intracompartmental pressures remains the diagnostic tool of greatest clinical utility. At present, no laboratory biomarker demonstrates adequate sensitivity or specificity to reliably confirm or exclude the diagnosis, though research is ongoing. The extent and duration of ischemia are the primary determinants of functional outcome in ACS. Accordingly, timely recognition and intervention—most critically surgical fasciotomy—are essential to prevent irreversible neuromuscular ischemic injury.

Conclusions: Comprehensive understanding of the risk factors, diagnostic tools, and therapeutic strategies for ACS facilitates prompt evaluation and intervention. Increased clinical vigilance is imperative to mitigate complications and limit irreversible tissue injury.

KEYWORDS

Acute Compartment Syndrome, Fasciotomy, Pediatric Compartment Syndrome, Pressure Monitoring

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1. Introduction and purpose

Acute compartment syndrome (ACS) is a rare, time-critical condition that can lead to irreversible loss of function and, in severe cases, death. Initially described by Volkmann in the late 19th century, ACS is defined by the detrimental elevation of pressure within a confined anatomical space that exceeds local perfusion pressures, thereby restricting oxygen delivery to enclosed tissues. Although ACS can involve any fascial compartment in the body, whether in the extremities or trunk, this article focuses on ACS affecting the limbs (Walls, 2017). Acute compartment syndrome most frequently arises in the context of fractures or vascular insults because muscle swelling following injury, can lead to an increase in intracompartmental volume due to the accumulation of blood and interstitial fluid within the fascial compartment (Hansen et al., 2021).

The aim of this article is to highlight rare etiologies of acute compartment syndrome (ACS). Although the vast majority of ACS cases are trauma-related, awareness of atypical causes is crucial, as early recognition can be life- and limb-saving. Timely diagnosis remains a key determinant of successful treatment outcomes in ACS.

Despite the well-documented and limb-threatening nature of acute compartment syndrome (ACS), its diagnosis remains challenging due to the lack of a universally accepted definition of onset. While hallmark signs such as pain and swelling are commonly recognized, these symptoms are nonspecific and frequently present in patients without ACS (Schmidt, 2016). Therefore the purpose of this article is to broaden the knowledge on acute compartment syndrome in accordance with current literature, thus leading to more precise diagnosis, successful treatment and prevention of complications. The authors of this article aim to gather the most up to date knowledge from various scientific research papers, in order to summarize available information on ACS including its risk factors, etiology treatment and therapeutic options.

2. Description of the state of knowledge

Anatomy and pathophysiology

Anatomy

Skeletal muscles are organized into compartments - distinct anatomical structures encased by fascia, a non-elastic connective tissue that surrounds structures like blood vessels, nerves, tendons, and ligaments (Walls, 2017). Fascia serves three primary biomechanical roles: it provides anchorage for muscles, supports organized muscle groupings, and enhances mechanical efficiency during muscular contractions (Jones et al., 2020). Fascial layers vary in thickness depending on anatomical location (Hansen et al., 2021). The lower leg for example consists of four muscle compartments (deep posterior, lateral, anterior, and superficial posterior) that contain the posterior tibial artery and vein, the tibial nerve, and 11 different muscles. Fascia does not stretch, therefore any accumulation of blood or muscle injury leading to swelling within a compartment will result in increased pressure inside it (Walls, 2017).

Pathophysiology

Acute compartment syndrome (ACS) occurs when the pressure within a muscle compartment increases to a level that impairs local circulation. Initially, rising pressure compromises capillary perfusion, followed by venous congestion and eventually arterial occlusion (Hansen et al., 2021). Reduced perfusion results in tissue ischemia, which subsequently causes nerve damage and muscle necrosis. The damage begins even before arterial flow is significantly reduced (Livingston et al., 2017).

Cells shift to anaerobic metabolism, consuming excess glucose and leading to hypoglycemia. The resulting hypoxia, oxidative insult, and glucose depletion cause a decline in adenosine triphosphate (ATP) production, ultimately impairing sodium-potassium pump function. As ATP-dependent ion channels fail, cellular homeostasis collapses. The sodium-potassium pump shutdown disrupts the membrane potential, allowing influx of chloride ions, which increases intracellular osmotic pressure and induces cellular swelling and lysis. The rupture of these cells exacerbates local inflammation and tissue injury. As compartmental pressure escalates further, vascular perfusion becomes critically compromised, perpetuating the cycle of ischemia and cellular damage (Jones et al., 2020). Research has demonstrated that tissue injury typically starts after 4 hours of ischemia, and irreversible damage may occur by 8 hours (Livingston et al., 2017).

Oxygen deprivation leads to muscle cell necrosis and rhabdomyolysis, releasing myoglobin into the bloodstream, which may result in acute kidney injury (Hansen et al., 2021).

Etiology and risk factors

Recent analyses suggest that young age is the strongest predictor of acute compartment syndrome (ACS), particularly in trauma patients. However, ACS can also occur in the absence of a fracture, and in such cases, patients are generally older and tend to have more medical comorbidities compared to those who develop ACS following a fracture. This highlights the need for clinical vigilance across a broad patient demographic, not just in the traditionally high-risk, young trauma population. In addition, studies showed that ACS occurs in 53% of patients with medial knee fracture dislocations, and in approximately 18% of patients with bicondylar tibial plateau fractures treated with knee-spanning external fixation (Hansen et al., 2021; Schmidt, 2016). Individuals receiving anticoagulation therapy are particularly vulnerable during the early postoperative period. In such cases, ACS is likely triggered by hematoma formation and surgical edema, which increase intracompartmental pressure, leading to capillary collapse and subsequent ischemia of neural and muscular tissues (Kooner et al., 2020).

Trauma

Trauma is the leading cause of ACS. The anterior distal region of the lower extremity is the most commonly affected area in compartment syndrome. Analysis showed that over two-thirds cases of compartment syndrome diagnoses were associated with fractures, with half of those involving the tibia. Open tibial fractures were involved in ACS three times more often than closed fractures. Available statistics may not fully reflect the true incidence, as compartment syndrome can often go undiagnosed in patients with severe multisystem trauma (Walls, 2017). The highest incidence of ACS following tibial fractures occurs in individuals aged 12–29 years, with males having up to 10 times higher risk than females—likely due to differences in trauma exposure patterns between sexes. ACS typically develops within 1–2 days of the inciting event but can occur several days later (Hansen et al., 2021). Prompt recognition and intervention are critical to prevent irreversible damage (Kooner et al., 2020).

Upper limb

Recent study aimed to describe the demographic and characteristics of patients with acute hand compartment syndrome showed that the most common causes were crush injury, prolonged decubitus, and infection (Williams et al., 2023).

Acute compartment syndrome of the upper extremity accounts for a significant proportion of cases related to orthopedic injuries. It occurs in approximately 10% of distal radius fractures and over 2% of hand fractures (Meshram et al., 2021).

Over the past 25 years, the demographics of acute hand compartment syndrome have shifted. This evolution is partly attributed to the opioid epidemic, which has led to an increased incidence of "found down" compartment syndromes—cases where patients are discovered unconscious or immobilized for prolonged periods. More than 20% of cases were associated with substance abuse (Williams et al., 2023).

There are cases of deltoid and hand ACS in young men following prolonged syncope because of polysubstance use. Young adult males appear to be particularly predisposed to developing deltoid compartment syndrome. The most frequently reported etiologies are iatrogenic injury or prolonged immobilization, together accounting for approximately 90% of cases (Kooner et al., 2020).

Rare causes

Acute limb compartment syndrome (ALCS) has been reported in a wide array of clinical contexts, including uncommon causes such as penetrating trauma from large animal or human bites. There are documented cases of ALCS wasp stings and other insect bites, primarily in children (Steadman et al., 2022).

Exercise-induced compartment syndrome

Acute exercise-induced compartment syndrome, sometimes referred to as “march gangrene,” is a rare but serious variant of compartment syndrome. It results from intense activity leading to sufficient swelling within a muscle compartment to impair perfusion and cause ischemic injury. Unlike chronic forms, this variant does not resolve with rest and may lead to tissue loss. There are cases of active duty servicemen who developed acute exercise-induced compartment syndrome of the lower leg following a military fitness assessment (Guenther et al., 2020).

Hemostatic imbalances

Hemostatic imbalances are important contributing factor, with both pro- and anticoagulant states implicated. These include spontaneous hemorrhages in patients with hemophilia, clotting factor deficiencies, hematological malignancies, and platelet disorders. Extreme thrombocytosis has also been associated with spontaneous bleeding that can precipitate ACS. Anticoagulant therapy itself may trigger ALCS, even in response to relatively minor trauma, such as venepuncture or acupuncture. Hypercoagulable conditions, such as protein S deficiency, have been linked to ACS secondary to thrombosis. There are reports of ACS related to deep vein thrombosis, typically occurring before treatment initiation, though cases have also been observed during anticoagulation and in unprovoked DVT (Steadman et al., 2022).

Intoxication

Patients who lost consciousness especially due to intoxication may remain in a position that places prolonged pressure on an extremity until they are discovered or regain consciousness. In these cases, the affected limb is compressed beneath the body weight of the individual, and they are unable to alleviate the pressure by repositioning themselves. Once the pressure from the patient’s body weight is relieved, an inflammatory response can be triggered, causing edema and, in some instances, hemorrhaging. This results in an increase in pressure within the affected muscle compartments, potentially leading to acute compartment syndrome (Jones et al., 2020).

Cocaine

Although cocaine abuse is a well-recognized risk factor for the development of rhabdomyolysis, the progression to compartment syndrome remains uncommon. Cocaine exerts its vascular effects through multiple pathophysiological pathways such as inhibition of the norepinephrine and dopamine reuptake at synaptic junctions and impairment of norepinephrine reuptake at the neuromuscular junction. Additionally, cocaine increases the production of endothelin and suppresses endothelial nitric oxide synthesis, contributing to vasoconstriction. Due to these combined mechanisms, cocaine use has been linked to various ischemic complications that may lead to ACS (Iftikhar et al., 2022).

Infection

Severe local infections can also lead to ALCS, especially in the context of necrotizing fasciitis. Other infectious contributors include non-necrotizing fasciitis, cellulitis, and sepsis. Most reported cases involve Group A Streptococci or *Staphylococcus aureus*, though less common pathogens such as *Vibrio*, *Clostridium*, and *Proteus* species have also been implicated (Steadman et al., 2022).

Compartment syndrome related to viral infection is considered atypical, but long-term COVID-19 can cause myosis either through direct invasion of myocytes by the virus or via autoimmune mechanisms and eventually lead to ACS. Early symptoms can vary widely, from dermatomyositis to weakness or pain to classic with MRI revealing muscle involvement (Moreira Machado et al., 2023).

Intra-arterial injection

ACS can be caused by intra-arterial injection of drugs such as oral methadone. A few cases have been documented and most of them involved methadone tablets dissolved in water often alongside crushed benzodiazepine tablets. Both the drug itself and its insoluble excipients—such as cellulose microcrystals—can cause endothelial damage and distal microvascular embolization. Other possible mechanisms include direct endothelial cytotoxicity leading to vasospasm, chemically induced endoarteritis, and tissue damage resulting from osmotic imbalance (Ruiz-Carmona et al., 2022).

Genetic predispositions

There are cases of recurrent compartment syndrome associated with a heterozygous dominant gain-of-function mutation in the skeletal muscle ryanodine receptor gene (*RYR1*)—a well-known genetic contributor to exertional rhabdomyolysis and myalgia. Recurrent CS can be also linked to PYGM-related McArdle disease as metabolic disturbances characteristic of McArdle disease may contribute to the pathophysiological mechanisms underlying CS. It is worth mentioning, that genetic predisposition to increased risk of trauma related acute compartment syndrome (ACS) is yet to be determined (Famili et al., 2023).

Revascularization

Acute compartment syndrome can be a severe and insidious complication following revascularization procedures for acute lower limb ischemia (ALI). Its onset during endovascular catheter-directed thrombolysis (CDT) poses a particular diagnostic challenge. ACS may not manifest until several hours after the surgical intervention. This delayed onset has sparked ongoing debate regarding whether it should be managed through prophylactic decompression fasciotomies or treated only upon clinical presentation. Currently, there are no definitive guidelines supporting routine prophylactic fasciotomy in cases of acute limb ischemia (Coccolini et al., 2020).

Studies documented rates ranging from 10% to 30% following revascularization for ALI. It is worth noting that a low threshold for performing fasciotomy was observed among surgeons, reflecting serious consequences of missed or delayed ACS diagnosis. Patients who developed ACS during CDT typically exhibited more severe preoperative ischemia, a higher prevalence of occluded popliteal artery aneurysms, and experienced increased amputation rates within the first year compared to controls (Olivia et al., 2022).

Snakebites

Acute compartment syndrome (ACS) is one of the most concerning complications associated with snakebite envenomation. Estimating the actual incidence of true ACS in the context of snakebite is challenging, as misdiagnosis is common. While some studies from report ACS in 4% to 15% of snakebite cases, others suggest a much lower incidence of approximately 1%. It is worth noting that the applicability of established diagnostic and treatment protocols for trauma-induced ACS to snakebite-associated cases remains controversial. The pathophysiological mechanisms underlying local tissue damage following bites from venomous snakes are largely enzymatic in nature, leading to progressive tissue injury that may mimic or, in rare instances, result in true ACS. Importantly, the management of snakebite-induced ACS should differ from that of traumatic ACS. Further preclinical and clinical research is essential to establish appropriate diagnostic and therapeutic strategies tailored to envenomation-related compartment syndrome (Cañas, 2024).

Well-leg compartment syndrome

Well-leg compartment syndrome (WLCS) is a subset of compartment syndrome that occurs due to abnormal positioning during surgery. Despite being a known complication, WLCS remains underdiagnosed and underreported. There is a case of a patient positioned in the lithotomy position during 2.5 hour surgery. The use of leg holders, probably contributed to the development of compartment syndrome. Lithotomy position caused initial ischemia, followed by reperfusion after surgery, and the effects of epidurally administered bupivacaine, which increased blood flow, leading to the development of WLCS (Anwer et al., 2020).

Extravasation

There are reports of ACS caused by peripheral phenylephrine and dopamine intravenous extravasation. As critical care practices evolve, vasoactive agents are increasingly being administered via peripheral lines in acute settings. Consequently it is important to remain vigilant for this complication and be prepared to implement emergent fasciotomy and pharmacologic intervention to prevent irreversible damage (Fisher & Jarrett, 2021).

Compartment syndrome is a rare but serious complication of contrast medium extravasation. While extravasation of contrast medium during computed tomography typically causes only minor injuries, there are instances—even in the absence of traditional risk factors—where it can lead to the development of compartment syndrome. Despite its infrequency, awareness of this potential complication is crucial for both physicians and radiology technicians (van Veelen et al., 2020).

Comorbidities

Several groups appear to be at heightened risk for ACS. For instance, patients with limb malignancies, especially sarcomas, have presented with a palpable mass and subtle signs of ACS. In such cases, the compartment syndrome may be masked by the primary pathology, necessitating a high index of suspicion. In patients with type 1 diabetes mellitus, ACS has been documented in a few cases, without a clearly defined mechanism. These occurrences involved both long-standing and newly diagnosed diabetics. Some researchers suggest a possible link between diabetes and spontaneous muscle infarction, though definitive proof remains elusive (Steadman et al., 2022).

Diagnostics

Clinical Presentation

ACS is traditionally characterized by the "Five Ps": Pallor, Pain out of proportion, Pulselessness, Paresthesia and Paralysis. It is worth noting however, that pallor and pulselessness indicate late-stage arterial compromise, which means irreversible damage may already have occurred. The presence of multiple “Ps,” severe pain on passive muscle stretch, or a tense, swollen compartment on palpation are more sensitive indicators (Hansen et al., 2021).

Pain

Pain is universally recognized as the most prominent and reliable symptom, typically presenting as the earliest sign (Schmidt, 2016; Walls, 2017). In conscious patients with ACS, the clinical hallmark is simply "pain, pain, pain, pain, and pain." (Hansen et al., 2021).

While pain is often linked to the injury itself, it is crucial to consider the possibility of increasing compartment pressure if the pain is disproportionate to the injury. The pain associated with compartment syndrome is often described as persistent, intense, deep, and hard to localize. Pain that intensifies with passive muscle stretching within the affected compartment should raise suspicion for compartment syndrome (Walls, 2017).

Paresthesia

The sensation of "pins and needles" in the cutaneous nerves of the affected compartment is a common symptom of compromised circulation. This sensation can be an early sign of compartment syndrome, which may still be reversible at this stage while fixed hypoesthesia or anesthesia typically indicates more advanced stages of the condition (Hansen et al., 2021; Walls, 2017).

Pallor

Shiny, tense, swollen skin distal to the injury prompt suspicion of ACS. Pallor occurs due to increased capillary pressure and reduced blood flow to capillary beds, typically following arterial occlusion. The affected limb may also exhibit cyanosis or mottling (Schmidt, 2016; Walls, 2017).

Paralysis

Paralysis is generally more common in crush injuries. The loss of motor function is typically the first sign of nerve impairment when ischemia occurs in the affected extremity. If the extremity appears lifeless, it suggests significant muscle and nerve damage, and acute compartment syndrome should be strongly considered (Walls, 2017).

Pulselessness

Hypokinetic or weak pulse can results from the compressive effect that occludes arterial blood flow to the injured extremity at or below the injury site. Complete absence of pulse is a relatively late and severe sign, indicating that compartment pressure exceeds the arterial pressure, compromising the pulse only when the

artery is contained within the affected compartment. The complete absence of a pulse is a grave clinical finding, signaling that the compartment pressure is greater than the systolic blood pressure (Walls, 2017).

Intracompartmental Pressure Measurement

Intracompartmental Pressure Measurement is the most widely used diagnostic tool for ACS, although it requires skill and experience to perform accurately. The needle must be correctly positioned within each affected compartment, and two measurements per compartment are recommended. Estimated normal pressures are: 8–10 mmHg in adults and 10–15 mmHg in children (Hansen et al., 2021).

Measurement approaches

There are two main measurement approaches.

Absolute pressure - pressures exceeding 30–40 mmHg are suspicious for ACS. Some use thresholds up to 70 mmHg to reduce overdiagnosis.

Delta pressure (perfusion pressure) - calculated as diastolic BP – compartment pressure. A delta is considered more clinically relevant and indicates impaired tissue perfusion (Hansen et al., 2021).

It is worth noting that, single pressure measurements may not accurately reflect dynamic changes in tissue pressure over time. Therefore, serial or continuous pressure monitoring—particularly trends showing rising compartment pressures or declining perfusion pressures—is considered more specific for diagnosing compartment syndrome.

Some clinicians advocate measuring near the fracture site to capture the peak pressure, while others recommend measurement at a distance from the fracture to reflect pressures more representative of the entire compartment (Schmidt, 2016).

Limitations

Relying solely on isolated intracompartmental pressure (ICP) thresholds to diagnose acute compartment syndrome (ACS) carries a risk of leading to unnecessary fasciotomies especially in late presentation ACS. Some experts advise against basing the diagnosis exclusively on pressure measurements (Osborn & Schmidt, 2020). On the other hand, research has demonstrated that compartment pressure monitoring can facilitate diagnosis prior to the onset of overt clinical symptoms, enabling earlier fasciotomy compared to reliance on clinical signs alone. This technique is both safe and minimally invasive, utilizing readily available equipment commonly found in acute care settings. Importantly, current evidence does not indicate an increased risk of complications associated with its use. Additionally, the literature suggests that routine use of compartment pressure monitoring does not correlate with higher fasciotomy rates, with outcomes from our institution aligning with these published findings (Keating & Duckworth, 2021).

Laboratory tests

There are no specific biomarkers for diagnosing ACS. However, elevated creatine kinase and myoglobin levels may indicate rhabdomyolysis, and renal function tests can detect secondary complications. Troponin levels have also shown potential as an early marker (Hansen et al., 2021).

Elevated systemic lactate levels are recognized as a predictor of impending abdominal compartment syndrome. However, it remains uncertain whether hyperlactatemia serves as an independent early indicator of compartment syndrome or if it simply correlates with other contributing factors—most notably, aggressive fluid resuscitation, which has been independently associated with the development of this complication in multivariate analyses. A similar pathophysiological mechanism has been observed in the extremities, where hypovolemic shock combined with high-volume fluid resuscitation has been shown to increase compartment pressures, even in anatomically uninjured lower limbs (Vaynrub et al., 2021).

Imaging

Imaging studies like X-rays or CT scans are typically not diagnostic for ACS itself but can help identify underlying injuries. In unconscious or sedated patients, imaging can assist in detecting trauma that may have triggered ACS.

Experimental methods like near-infrared spectroscopy (NIRS) and ultrasound show promise for non-invasive ACS detection, but are not yet standard clinical tools (Hansen et al., 2021). Doppler ultrasound, can be employed in the acute setting to rule out vascular causes of swelling and pain, such as deep vein thrombosis. While helpful in assessing arterial flow, its role in diagnosing compartment syndrome is limited.

MRI, on the other hand, is considered a superior imaging modality due to its ability to grade muscle involvement and suggest the likelihood of reversible ischemia. It also aids in surgical planning by precisely localizing the affected muscle groups and fascial compartments. It is worth noting, that MRI comes with significant drawbacks, including high cost, limited availability, and time requirements that can render it impractical in urgent clinical situations (Miranda-Klein et al., 2020).

Electromyography

While electrodiagnostic assessments are of limited utility in the diagnosis of ACS, they can assist with prognosticating return of function after surgical fasciotomy or in clarifying the injury pattern in cases in which a traumatic injury results in subsequent neuromuscular deficits. Around three to six weeks after the onset of symptoms, electromyography (EMG) can help determine the potential for muscle recovery based on specific electrical patterns. The presence of spontaneous activity, such as fibrillation potentials, is generally considered a favorable prognostic indicator, as it reflects the presence of viable muscle tissue. In contrast, the absence of such activity typically suggests fibrosis or necrosis and is associated with minimal chances for functional recovery (Broadhurst & Robinson, 2020).

Future perspectives

Multiple studies have explored potential diagnostic approaches, including objective assessment of limb firmness and the use of adjunctive technologies such as near-infrared spectroscopy, electromyography, and intramuscular pH measurement. Further research is necessary to evaluate effectiveness of those methods (Osborn & Schmidt, 2020).

Recent studies have identified CCL23 as a potential biomarker distinguishing ACS from tibial fracture cases, and demonstrated the diagnostic significance of combining CSF-1, IL-6, and HGF in differentiating ACS from healthy individuals. Nevertheless, larger cohorts are necessary to validate these findings (Wang et al., 2023).

In Experimental study aimed at identifying a biochemical marker for the early diagnosis of acute compartment syndrome (ACS), several parameters showed significant predictive value. These included total oxidant status (TOS), ischemia-modified albumin, aspartate aminotransferase and neopterin. Among these, TOS was identified as the earliest predictive marker for ACS, suggesting it may play a key role in the early detection and timely intervention of the condition. Although some promising strategies have emerged from experimental research in a rat model, randomized controlled trials are still required to fully assess their diagnostic potential and clinical effectiveness (Yıldırım et al., 2023).

Treatment

Clinicians managing patients at risk for ACS are often faced with a difficult decision between two unfavorable options: proceeding with fasciotomy, thus exposing the patient to potential surgical risks and increased healthcare costs or delaying intervention, thereby risking the detrimental consequences of a missed or late-diagnosed compartment syndrome (Schmidt, 2016).

The initial management focused on shock treatment and preservation of renal function involves the administration of substantial intravenous fluids. Typically 0.9% sodium chloride at a rate of 6 to 12 liters over 24 hours is used unless there are contraindications such as cardiac failure or pulmonary edema are present. The primary objective in the treatment of ACS is to prevent irreversible damage to intercompartmental structures through rapid decompression of the affected compartment to restore perfusion and oxygenation (Walls, 2017).

Initial supportive measures include: removing or loosening tight bandages or casts, elevating the limb to heart level, repositioning bone fractures and maintaining normotension. Negative-pressure wound therapy may reduce the time to wound closure and the need for skin grafts (Hansen et al., 2021).

Pain management

In trauma patients presenting to the emergency department, pain management can be particularly challenging. The presence or risk of ACS complicates analgesic strategies, as excessive analgesia may mask the progression of compartment syndrome. Regional anesthesia, particularly peripheral nerve blocks, has traditionally been regarded as a low-priority analgesic option in high-risk clinical scenarios. It is worth noting however that, recent literature suggests that peripheral nerve blocks are not absolutely contraindicated in patients at risk of ACS, provided that continuous clinical surveillance is maintained and specific precautionary guidelines are followed. Careful pain control must be balanced with vigilant clinical observation to ensure timely diagnosis and intervention (Soulioti et al., 2024).

Fasciotomy

An emergency fasciotomy is considered the treatment of choice for ACS, typically performed by a vascular, orthopedic, or trauma surgeon under general anesthesia. Key principles for performing an effective fasciotomy include ensuring appropriate incision length and depth with accurate anatomical landmarking, achieving complete decompression of all involved muscle compartments, minimizing iatrogenic injury to adjacent neurovascular structures, excising necrotic or ischemic tissue as needed, performing (Jones et al., 2020).

Fasciotomy techniques

Fasciotomy techniques include open fasciotomy, fasciotomy with partial fasciectomy, or a minimally invasive (endoscopic) approach. The number of compartments released during the procedure (ranging from 1 to 4) may vary. In most studies both 2-compartment and 4-compartment fasciotomies have demonstrated similar outcomes in terms of functional recovery patients' satisfaction and residual symptoms. In conclusion the fasciotomy technique is less important than ensuring complete decompression of the affected extremity's compartments. (Weiss et al., 2022).

Postoperative management

The surgical wounds are left open, with delayed closure expected within 7 to 10 days. It is worth noting that this intervention creates new surgical wounds, which may predispose the patient to complications such as infection, delayed wound healing, bleeding, and cosmetic concerns (Walls, 2017). Therefore postoperative management such as regular dressing changes every 24 to 72 hours and careful timing for eventual wound closure is very important (Jones et al., 2020). Surgical decompression must be accompanied by continuous assessment of the patient's clinical status, including close monitoring, fluid resuscitation, and hemodynamic stabilization to optimize outcomes and prevent systemic complications (Soulioti et al., 2024).

Historically, wet-to-dry gauze dressings were the standard for managing fasciotomy wounds until delayed primary closure or skin grafting could be performed. However, in recent years, negative-pressure wound therapy (NPWT) has become increasingly favored (Schmidt, 2016).

Contraindications

Coagulopathy is one of the few absolute contraindications to fasciotomy, due to the risk of uncontrolled hemorrhage. A relative contraindication is late diagnosis, when irreversible muscle necrosis has already occurred. However, determining the point at which it is "too late" to intervene remains clinically subjective, as no validated guidelines currently exist. Decisions in such cases rely heavily on clinical judgment and experience (Schmidt, 2016).

Outcome and complications

Studies have shown that when fasciotomy is performed within 12 hours of the onset of compartment syndrome (defined as the first appearance of any clinical sign such as motor weakness, stretch pain, or hypesthesia) normal function is restored in approximately 68% of patients (Schmidt, 2016). Complications of ACS include: nerve injury, rhabdomyolysis, amputation and long-term disability (Hansen et al., 2021).

In a recent systematic review of forearm compartment syndrome, the overall complication rate was reported to be 42%, with neurologic deficits being the most frequently observed complication. Other notable complications included gangrene, Volkmann ischemic contracture, crush syndrome and complex regional pain syndrome. Volkmann ischemic contracture is one of the most devastating complications of ACS. It results from muscle necrosis caused by irreversible tissue ischemia and may lead to permanent functional impairment due to fibrosis and shortening of the affected muscles, often resulting in a fixed flexion deformity of the wrist and fingers (Kistler et al., 2018).

Outcome predictors

The duration of ischemia is the strongest predictor of patient outcomes. After one hour of ischemia, nerve dysfunction is typically reversible. By four hours, irreversible nerve damage may begin to occur, and after six hours, muscle necrosis can set in. However, fasciotomy itself carries significant risks. Approximately one-third of patients experience postoperative complications, including infection and tissue necrosis. Additionally, sensory disturbances, scarring, and iatrogenic nerve injuries may develop following the fasciotomy (Hansen et al., 2021).

Delayed or Missed Diagnosis

Failure to recognize or act on ACS in time is the most serious pitfall. Because the exact onset of symptoms can be hard to define, if more than 24–48 hours have passed since symptom onset, different therapeutic approach may be required (Hansen et al., 2021).

Some studies showed that in adult patients with evidence of irreversible intracompartmental damage, fasciotomy is not indicated. What is more, studies suggest that, if fracture stabilization is required in such cases, the surgical approach should avoid disrupting the affected compartment. Techniques such as external fixation or casting should be employed (Osborn & Schmidt, 2020). It is worth noting, that large-scale data analyses employing multivariable regression have demonstrated no association between the method of fracture stabilization and the development of acute compartment syndrome. Therefore some experts believe there is no justification to opt for an alternative fixation method when intramedullary nailing is deemed the most appropriate treatment for a given fracture (Keating & Duckworth, 2021).

Special Clinical Challenges

Patients with altered consciousness—whether due to trauma, intoxication, or therapeutic sedation—are unable to communicate pain, which is the primary symptom of ACS. In such cases, objective diagnostic tools such as pressure measurements, blood tests, and imaging should be utilized (Hansen et al., 2021). Repeated or continuous monitoring of ICP is recommended until ACS can be definitively confirmed or excluded (Osborn & Schmidt, 2020).

Preexisting injuries, acute nerve damage, or therapeutic regional anesthesia may all cause sensory loss, thus masking pain and delaying diagnosis. This highlights the importance of close monitoring and a high index of suspicion when regional blocks are used in trauma patients (Hansen et al., 2021).

Neonatal Patients

Neonatal compartment syndrome is quite a rarity, with less than hundred cases reported to date. Proposed etiologies in neonatal patients include oligohydramnios, amniotic band syndrome and thromboembolic events. It is worth noting that the most significant diagnostic clue is sentinel lesion, consistently observed in all documented cases. However, its presentation can vary depending on the lesion's age and ischemic changes in the skin are often mistaken for birth-related trauma (Andey et al., 2021).

In most cases diagnosis has to be made clinically as routine measurement of compartment pressures is not commonly performed in neonates due to the lack of established norms for pressure gradients in this age group (Livingston et al., 2017). Initially after operation limb may appear as pink and well perfused, but in some cases gangrene can progress despite successful pressure release (Andey et al., 2021).

Pediatric Patients

Pain assessment and neurological evaluation can be difficult in children. A practical guide to ACS signs in children includes the three A's: Agitation, Anxiety, and escalating Analgesic demand (Hansen et al., 2021).

Risk factors

Pediatric acute compartment syndrome (PACS) occurs more frequently in males. Some experts suggest that this higher risk may result from an altered muscle-to-fascial ratio, where rapidly growing muscle is constrained by a relatively limited and inflexible fascial envelope (Mortensen et al., 2020). PACS caused vascular injury and infection tend to affect younger children (under 10 years) and may lead to a faster onset of ischemia and result in poorer outcomes (Livingston et al., 2017).

Study focused on operatively managed pediatric Monteggia fractures and equivalents indicated that patients with preoperative vascular compromise, neurological deficits and those treated with intramedullary fixation of the radius demonstrated a significantly increased risk of developing acute compartment syndrome. However, further research with expanded sample size is necessary (Kopriva et al., 2020).

Diagnostics and outcomes

Children have higher normal resting pressures compared to adults and they may be more sensitive to pressure increases due to their lower resting blood pressures, but the difference in fasciotomy thresholds has not yet been conclusively demonstrated in the literature (Hansen et al., 2021; Livingston et al., 2017).

The functional outcomes after PACS are generally positive, with over 90% of patients in most studies reporting excellent recovery. What is more, unlike in adults, fasciotomy wounds in children could be usually closed during a second surgery and do not require skin grafts. It was suggested that muscle necrosis in children might be partially reversible, as there have been instances where muscle tissue initially deemed necrotic during fasciotomy later regained viability (Livingston et al., 2017).

3. Conclusions

Acute compartment syndrome (ACS) remains a challenging clinical emergency that necessitates prompt recognition and surgical intervention to prevent irreversible neuromuscular damage. Although trauma remains the leading cause, ACS can arise from a range of atraumatic etiologies, including coagulopathies, envenomation, infection, or prolonged immobilization. Raising awareness of ACS among healthcare providers across disciplines is crucial.

ACS presents substantial diagnostic challenges, particularly in patients unable to communicate symptoms effectively. Pain—especially disproportionate to the injury and exacerbated by passive muscle stretch—remains the hallmark symptom and should always prompt urgent evaluation. Although it has certain limitations, intracompartmental pressure measurement remains the most important and objective diagnostic tool. At present, no laboratory test offers sufficient sensitivity and specificity to reliably diagnose ACS. While

biomarkers such as serum myoglobin and creatine kinase may support clinical suspicion, they lack diagnostic precision. Nevertheless, promising research is underway to identify more accurate biochemical markers, which may enhance future diagnostic pathways.

Special populations—such as children, unconscious patients, and individuals with altered mental status—are particularly vulnerable due to the difficulty of frequent, reliable clinical assessment.

Ultimately, high clinical suspicion, systematic monitoring, and timely fasciotomy remain the cornerstones of effective ACS management. Prompt diagnosis and treatment remain the most effective strategies to prevent long-term complications and preserve limb function. Future research should aim to optimize diagnostic algorithms and identify reliable diagnostic tools to support early detection and intervention.

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