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Compartment Syndrome: A Literature Review

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ABSTRACT

Background: Compartment syndrome is a limb-threatening orthopedic emergency characterized by increased pressure within an osteofascial compartment, leading to impaired tissue perfusion and potential irreversible necrosis. Although most frequently associated with tibial fractures, it can also occur in the hand, foot, forearm, and paraspinal muscles.
Methods: This review synthesizes current evidence on the definition, epidemiology, pathophysiology, clinical features, and diagnostic approach to compartment syndrome. Literature was systematically searched across PubMed, Scopus, Web of Science, and Cochrane Library using Boolean operators. Risk of bias was assessed with the Cochrane Risk of Bias tool for randomized trials and the Newcastle-Ottawa Scale for observational studies.
Results: Approximately 69% of compartment syndrome cases are associated with fractures, with 30% involving the tibia. Pathophysiological mechanisms are explained by the arterio-venous gradient theory and ischemia-reperfusion syndrome, both of which describe a vicious cycle of reduced capillary perfusion, metabolic deficit, ischemia, and necrosis. Clinically, the condition presents with severe pain disproportionate to injury, pain on passive stretch, paresthesia, and tense swelling, summarized by the "6 Ps." Diagnosis remains primarily clinical, but adjunctive investigations such as radiography can identify underlying fractures, while compartment pressure measurement provides objective confirmation in equivocal cases.
Conclusion: Early recognition and timely diagnosis are critical in preventing irreversible damage in acute compartment syndrome. While clinical evaluation remains the cornerstone of diagnosis, adjunctive tools such as imaging and pressure monitoring may assist in difficult cases. Prompt surgical intervention with fasciotomy offers the best chance of preserving limb function.

Keywords: Acute compartment syndrome, Intracompartmental pressure, Fasciotomy, Tibial fracture, Diagnosis, Near-infrared spectroscopy.

INTRODUCTION

Compartment syndrome is one of the orthopedic and traumatology emergencies that requires rapid recognition and intervention. The condition was first introduced by Volkmann in the 19th century and is defined as an increase in intracompartmental pressure within an osteofascial compartment, which subsequently disrupts normal circulation and tissue perfusion.
The risk factors for compartment syndrome include both increased compartment content and decreased compartment capacity.

Contributing factors may involve the application of excessively tight dressings or splints, eschar formation following burns, and muscular edema. Among these, fractures represent the most common etiology, with approximately 69% of compartment syndrome cases associated with fractures, particularly involving the tibia.

If elevated compartment pressure persists, it can lead to tissue damage that may be reversible or irreversible depending on the duration and severity of ischemia. The principle of management is to reduce the intracompartmental pressure. This can be achieved either through conservative measures or surgical intervention, with fasciotomy remaining the definitive treatment. Prompt diagnosis and immediate intervention are essential to achieve favorable outcomes, as delays in management can result in permanent functional impairment.

METHOD

Literature Search Strategy

A systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines. A comprehensive search was performed across multiple electronic databases, including PubMed/MEDLINE, Google Scholar, Web of Science, and Cochrane Library.

The following Boolean operators and Medical Subject Headings (MeSH) terms were applied. ("compartment syndrome" OR "acute compartment syndrome" OR "Volkmann ischemic contracture") AND ("orthopaedics" OR "trauma" OR "fracture" OR "tibia fracture" OR "long bone fracture") AND ("fasciotomy" OR "surgical decompression" OR "management" OR "treatment")

Study Selection and Data Extraction

Two independent reviewers screened titles and abstracts for eligibility. Full texts were retrieved for potentially relevant studies. Data were extracted using a standardized form, capturing study characteristics, patient demographics, interventions, and outcomes. Any disagreements were resolved through consensus or third-party adjudication.

Risk of Bias Assessment

The risk of bias was assessed independently by two reviewers. For randomized controlled trials, the Cochrane Risk of Bias 2.0 tool was used. For observational studies, the Newcastle-Ottawa Scale (NOS) was applied, evaluating selection, comparability, and outcome domains. Discrepancies were resolved by discussion. Studies were classified as having low, moderate, or high risk of bias.

RESULT

Definition

Compartment syndrome is defined as a condition resulting from increased pressure within an osteofascial compartment.[1] This elevated pressure disrupts microcirculation, leading to tissue ischemia and potentially irreversible necrosis.[2] The condition was first described by Volkmann in the 19th century, in association with long bone fractures, severe edema, and ischemic contracture of muscles.[3]

Epidemiology and Etiology

Although compartment syndrome is most frequently associated with acute injuries of the leg, it can occur in other anatomical regions, including the hand, foot, forearm, and even paraspinal muscles. Hope and McQueen classified patients into two groups: fracture-related compartment syndrome and non-fracture-related compartment syndrome. In a study of 164 cases, McQueen et al. reported that 69% were fracture-related, with 30% involving tibial fractures.[2]

According to Tollens et al., the etiological factors can be divided into two categories: (1) reduced compartment size, and (2) increased compartment contents.[4] Examples include tight dressings, circumferential eschar from burns, hematomas, or muscle swelling due to trauma.

Pathophysiology

Compartment syndrome arises from disturbances in fluid shifts between blood, extracellular, and intracellular compartments, resulting in increased intramuscular pressure. The survival of muscle cells depends on adequate oxygenation and nutrition, which are directly related to tissue perfusion. Microcirculatory flow, governed by the balance between tissue pressure and venous outflow, determines oxygen and nutrient delivery. In compartment syndrome, elevated intracompartmental pressure compromises capillary perfusion, reducing tissue oxygen tension (pO_2) and causing metabolic deficits, ischemia, and ultimately necrosis.[4]

Two major theories explain the pathophysiological process: the arterio-venous gradient theory and the ischemia-reperfusion syndrome. Both suggest that increased tissue pressure decreases capillary blood flow and oxygen delivery, leading to metabolic failure. Compensatory mechanisms such as vasodilation, collateral vessel formation, and anaerobic metabolism may temporarily mitigate oxygen demand, but if insufficient, irreversible ischemia and necrosis ensue.

In cases where compartment syndrome is caused by external compression (e.g., tight casts, traumatic hematomas), the arterio-venous gradient mechanism predominates, with rising intravenous pressure and reduced capillary flow. In contrast, when associated with systemic hypovolemia or shock, decreased arterial pressure exacerbates impaired capillary perfusion.[4]

Reperfusion injury following fasciotomy or revascularization after embolic events contributes to a vicious cycle. This process involves oxygen free radical release, calcium influx into ischemic muscle, and neutrophil infiltration into reperfused vasculature. These mechanisms trigger vasodilation, capillary leakage, edema formation, and further compartmental pressure elevation, reactivating the arterio-venous gradient cycle.[4]

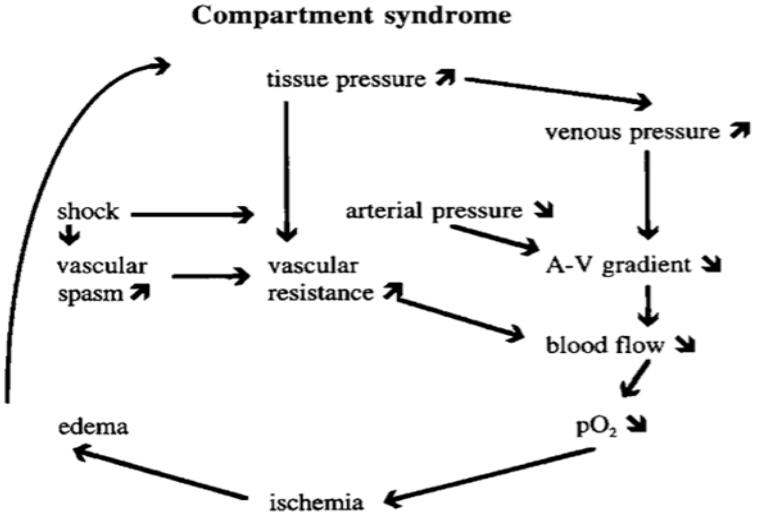

Figure 1. Pathophysiology of compartment syndrome (Janzing HMJ, 2007)

Diagnosis

Acute compartment syndrome typically develops within a few hours after trauma, although in some cases it may present up to 48 hours later. The earliest objective physical finding is a tense, "board-like" feeling of the involved compartment. Pain is usually severe and disproportionate to the degree of injury. Initially, pain is elicited primarily during passive stretching of the affected muscles; however, this sign may be absent in advanced cases of acute compartment syndrome. In the early phase, patients may describe a burning sensation or deep, poorly localized pain within the compartment. Paresthesia, hypoesthesia, or deep muscular pain may

also serve as indicators of evolving compartment syndrome.

Classically, the clinical presentation of acute compartment syndrome is summarized by the "6 Ps": pain on passive stretch, pressure (tense compartment), pulselessness, paresthesia, paralysis, and pallor (or pink skin color changes). These signs are essential in differentiating compartment syndrome from arterial or isolated nerve injuries. In contrast to arterial lesions—where peripheral pulses are often absent—peripheral pulses and capillary refill usually remain preserved in patients with compartment syndrome.[4,5]

Physical examination generally follows the "6 Ps" as described above. However, focused assessment of the neurovascular status of the affected limb is essential. A systematic approach can be summarized as look (inspect the skin for lesions, swelling, or discoloration), feel (palpate the involved compartment, noting temperature, tenseness, and tenderness and also assess peripheral pulses, perform two-point discrimination, and evaluate sensory deficits) and Move (test motor function of the involved muscle groups)

The sensitivity and specificity of clinical manifestations alone are limited; therefore, adjunctive methods such as compartment pressure measurements may be required to support diagnosis. Given the rapidly progressive nature of compartment syndrome, serial examinations are critical to avoid delayed recognition.[3]

Although compartment syndrome remains primarily a clinical diagnosis, supporting investigations can provide valuable information to guide decision-making. Radiographic examination of the injured limb plays an important role in identifying the underlying pathology, with fractures being the most common cause of compartment syndrome. Plain X-rays are therefore routinely performed in the acute setting, as they help establish the primary diagnosis and clarify the mechanism leading to increased compartmental pressure.

In addition to imaging, direct measurement of intracompartmental pressure may be performed in selected cases, particularly when the clinical presentation is equivocal. Although not routinely indicated, this invasive technique provides objective data that can strengthen diagnostic certainty. One commonly described method involves the use of a manometer, in which saline is injected into the compartment and the resistance is recorded to calculate pressure levels. An alternative approach is the slit catheter technique, whereby a catheter is introduced into the compartment and connected to an arterial line transducer. Compared with the manometer, this technique provides greater accuracy and has the advantage of allowing continuous monitoring of compartment pressures over time. Such measurements can be particularly useful in patients who are unconscious, sedated, or unable to report symptoms reliably.

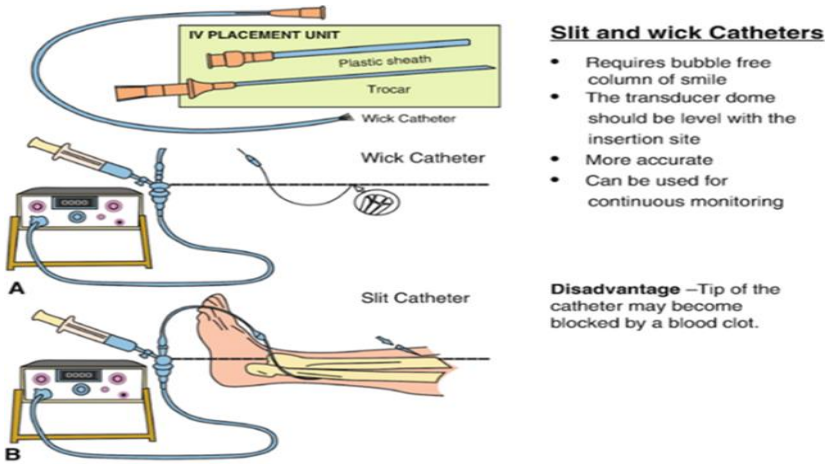

Figure 2. Created by authors; adapted from Elliott KGB, 2003

Normal intracompartmental pressure ranges between 0–8 mmHg. A pressure exceeding 30 mmHg is generally considered the threshold for fasciotomy, with values between 30–50 mmHg indicating compromised circulation. At pressures around 30 mmHg, capillary perfusion becomes inadequate to sustain tissue oxygenation, and fascia compliance markedly decreases. Once an absolute intracompartmental pressure of 33 mmHg is reached, the fascia is unable to stretch further, accelerating ischemic injury.[6]

Delta pressure, defined as the difference between diastolic blood pressure and intracompartmental pressure, is a widely accepted parameter for diagnosing compartment syndrome. A delta pressure of 10–30 mmHg suggests inadequate perfusion and relative ischemia of the affected limb.[7] Some authors advocate using the mean arterial pressure (MAP) minus intracompartmental pressure as a more accurate diagnostic reference.

The severity of ischemic damage depends not only on the magnitude of pressure elevation but also on the duration of exposure. Higher pressures lead to more rapid injury, but prolonged moderate elevations can cause comparable damage. For example, an absolute intracompartmental pressure of 40 mmHg sustained for six hours in patients with calf trauma is associated with poor outcomes. Biochemical and histological changes may appear at a delta pressure of 20 mmHg within 4–6 hours, and when delta pressure approaches zero, changes can occur within 2–3 hours. Delays in fasciotomy of more than 12 hours are consistently linked to irreversible muscle necrosis.[6]

NIRS oximetry is a non-invasive, real-time, and continuous monitoring technique that measures tissue oxygenation within suspected compartments. Modern NIRS devices can assess multiple locations simultaneously and measure oxygenation at depths up to 3 cm. In acute compartment syndrome, NIRS values decrease significantly in the affected limb compared to the uninjured side, supporting early diagnosis.[8]

Ultrasonography can demonstrate increased blood flow and compartmental enlargement, particularly in exertional compartment syndrome, though its role in acute cases remains limited.[9] Advanced imaging such as CT and MRI provide detailed anatomical information but are not routinely recommended in acute settings due to the urgency of surgical intervention.[4]

Several biochemical parameters have been proposed to support the diagnosis of compartment syndrome. These include serum creatine kinase >4000 U/L, chloride >104 mg/dL, and BUN <10 mg/dL. While these markers show high specificity, their low sensitivity precludes their use as gold-standard diagnostic tests in routine practice.[10]

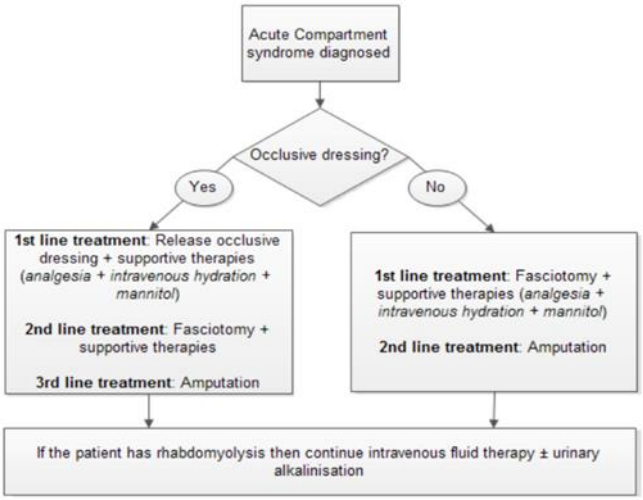

Figure 2. Acute compartment syndrome management algorithm (Mabvuure NT, 2012)

Management

For non-surgical management, initial measures focus on relieving external compression and optimizing systemic conditions. These include removing constrictive dressings or casts, providing adequate analgesia, intravenous hydration, and administering mannitol when indicated. In cases complicated by rhabdomyolysis, aggressive fluid resuscitation and urinary alkalinization with sodium bicarbonate are recommended.[11]

While surgical management consist of surgical fasciotomy and indicated when conservative measures fail or when clinical signs strongly suggest acute compartment syndrome. First described by Bardenheuer in 1906, fasciotomy remains the definitive treatment. In vascular injury, fasciotomy should precede vascular repair. While traditionally performed under general or regional anesthesia in the operating theatre, bedside fasciotomy under local anesthesia has also been reported.

Intraoperatively, muscle viability must be assessed. Viable muscle is pink, bleeds upon incision, and contracts when stimulated. Necrotic muscle should be debrided until healthy tissue is reached. Fasciotomy wounds are generally left open due to swelling and tissue retraction. Delayed closure is attempted within 7–10 days; if primary closure is not possible, split-thickness skin grafts (STSG) may be required.

Adjunctive wound management techniques include negative pressure wound therapy (VAC™) and gradual approximation methods such as shoelace or loop-vessel techniques. Both have demonstrated safety and effectiveness, although cost-effectiveness and superiority remain debated.[12]

Complications

Acute compartment syndrome is associated with several serious complications, many of which can result in long-term morbidity if not managed promptly. One of the most recognized sequelae is Volkmann's contracture, which occurs due to ischemic muscle necrosis followed by fibrotic scar formation. This process leads to muscle shortening, decreased elasticity, and adhesions with surrounding tissues, ultimately restricting the range of motion. Patients may require extensive rehabilitation or surgical interventions such as tendon transfer, contracture release, or neurolysis to restore function.[3]

Another major complication is rhabdomyolysis, which arises from widespread muscle necrosis. The breakdown of ischemic muscle releases myoglobin into the circulation, placing patients at risk of acute kidney injury and, in severe cases, renal failure. Rhabdomyolysis represents not only a local but also a systemic consequence of untreated compartment syndrome.[13]

Limb loss represents the most devastating outcome, with amputation rates reported between 5.7% and 12.9%. Even in patients who avoid amputation, permanent disabilities such as chronic pain or foot drop are common, and only about two-thirds of patients are able to return to their pre-injury occupational activities.[13]

Finally, reperfusion injury may complicate cases in which revascularization or fasciotomy is performed after a prolonged ischemic period. This phenomenon is mediated by the generation of reactive oxygen species, neutrophil adhesion to the vascular endothelium, and microvascular dysfunction. The systemic impact can be profound, manifesting as systemic inflammatory response syndrome (SIRS) or multiple organ dysfunction syndrome (MODS), highlighting the life-threatening potential of delayed intervention.[14]

Differential Diagnosis

Several conditions can mimic the clinical presentation of acute compartment syndrome, making timely and accurate diagnosis challenging. Deep vein thrombosis and cellulitis may present with limb swelling, pain, and erythema, while gas gangrene can also produce severe pain and soft tissue changes, sometimes accompanied by

crepitus. Phlegmasia cerulea dolens, a severe manifestation of venous thrombosis, may lead to marked swelling, pain, and compromised perfusion, closely resembling compartment syndrome. Similarly, rhabdomyolysis can cause muscle pain, swelling, and biochemical abnormalities that overlap with the syndrome's presentation. More unusual but relevant considerations include cnidarian envenomation, which may result in localized swelling and pain, and peripheral vascular injuries, which share overlapping signs such as pain, pallor, and pulselessness. Distinguishing these entities from compartment syndrome requires careful clinical evaluation, supported by adjunctive investigations when appropriate, to ensure that surgical fasciotomy is not unnecessarily delayed.[15]

Prognosis

The prognosis of acute compartment syndrome depends heavily on the timeliness of diagnosis and intervention. When fasciotomy is performed within six hours, full recovery of limb function is achievable in most cases. Delays beyond six hours increase the risk of residual neurological deficits, while fasciotomy after 12 hours is often associated with irreversible damage and a higher likelihood of amputation.[15]

CONCLUSION

Compartment syndrome is a surgical emergency that requires immediate recognition and intervention. The most common cause is tibial fractures following trauma to the calf, although the condition may also occur in other regions such as the forearm, hand, and thigh. While fractures represent the leading risk factor, additional causes include tight casting, muscular edema, and coagulopathies such as hemophilia.

Clinically, compartment syndrome is classically described by the "6 Ps": pain on passive stretch, pressure, pulselessness, paresthesia, paralysis, and pallor/pink discoloration. These findings guide the diagnosis; however, confirmatory investigations may be required. The most specific adjunctive test is intracompartmental pressure measurement using a slit catheter. Surgical intervention with fasciotomy is indicated when absolute compartment pressure exceeds 30 mmHg or when the differential between diastolic blood pressure (or mean arterial pressure) and intracompartmental pressure is less than 30 mmHg.

Delay in appropriate management significantly worsens outcomes. Several reports emphasize that interventions performed beyond six hours of onset are associated with irreversible damage and poorer prognoses. Potential complications include Volkmann contracture, peripheral nerve injury, rhabdomyolysis, reperfusion injury, and in severe cases, limb amputation. Timely management is critical: fasciotomy performed within six hours provides the best chance for complete functional recovery. Delays beyond this window significantly increase the risk of irreversible complications, including Volkmann contracture, nerve injury, rhabdomyolysis, reperfusion injury, and even limb amputation.

Declaration by Authors

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