Current Approach to the Evaluation and Management of Acute Compartment Syndrome in Pediatric Patients

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Abstract: Acute compartment syndrome is an emergent condition caused by increased pressure within a closed compartment. The most common etiology is fractures, but there a number of atraumatic causes cited in the literature. Acute compartment syndrome occurs most frequently in the anterior compartment of the lower leg, followed by the volar forearm. Patients may present with severe pain, pain with passive stretch, swelling, paresthesias, numbness, weakness, decreased pulses, and delayed capillary refill. No finding in isolation can exclude the diagnosis. Direct measurement of the intracompartmental pressure is the most important diagnostic test. Treatment involves removal of compressive dressings and surgical consultation for emergent fasciotomy.

Key Words: compartment syndrome, fasciotomy, rhabdomyolysis

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TARGET AUDIENCE

This CME activity is intended for all practitioners who care for pediatric patients presenting with possible compartment syndrome, which may include general pediatricians, pediatric emergency physicians, general emergency physicians, pediatric intensive care physicians, and orthopedic surgeons.

LEARNING OBJECTIVES

After completion of this article, the reader should be better able to:

1. Describe the signs and symptoms of pediatric compartment syndrome.
2. Explain the use of intracompartmental pressure for diagnosing compartment syndrome.
3. Identify management strategies for pediatric compartment syndrome.

Acute compartment syndrome (ACS) is an emergent condition that requires prompt recognition and management to avoid serious complications.[1] Richard von Volkmann[2] was the first to describe the complications of ACS in the 19th century, when he reported the case of a hand contracture occurring as a complication of the disorder. Since then, this disorder has been increasingly recognized and studied in the literature. The overall incidence has been suggested to be 0.7 to 7.3 cases per 100,000.[3] Unfortunately, the pediatric-specific literature is more limited, with 1 study of the National Pediatric Trauma Registry identifying 130 documented cases of pediatric ACS in a 3-year period.[4] However, as with many databases, the incidence is often underreported, and it is likely that the true incidence in pediatric patients is much higher than this.

The pediatric population is at particular risk of developing ACS because of their relatively high muscle bulk–to–compartment size ratio and stronger fascial structures, reducing the compartment's ability to accommodate significant increases in swelling.[3,5] The highest incidence of ACS occurs in the 10- to 14-year-old age range, with males having a 4-times greater risk of developing ACS than females.[4,6] While most cases of ACS occur within 24 hours of the initial injury, ACS may present up to several days after the inciting injury.[7] The most common cause of ACS are fractures, which comprise 85% of all cases among children.[4] Importantly, the presence of an open fracture does not exclude the presence of ACS.[8–11] It is hypothesized that ACS occurs in open fractures because the severe mechanism of the injury and significant energy transfer required to cause an open fracture lead to more tissue damage and local swelling, whereas the relatively small fascial tears do not allow adequate decompression of the compartment.[8–11] While fractures and other types of trauma are the most common causes, there are a number of other potential causes outlined in Table 1.

Acute compartment syndrome can be challenging to diagnose, but it is important to identify and treat these patients early in their presentation in order to improve their outcomes. The long-term sequelae of ACS can be severe, including permanent limb disability, amputation, and death.[42–44] Therefore, it is essential for providers to understand the historical features, physical examination findings, diagnostic testing, and management options in order to provide optimal care to this patient population.

Anatomy and Pathophysiology

Acute compartment syndrome occurs when there is increased pressure within an enclosed fascial space, leading to decreased perfusion and subsequent tissue damage. The causes for an increase in intracompartmental pressure (ICP) can be divided into external causes (ie, pressure applied externally to the compartment) and internal causes (ie, an increase in the contents within a compartment).[45] Following the initial injury, local inflammation and tissue damage lead to an increase in ICP.[46] As cells die, intracellular contents are released into the interstitial space, drawing additional fluid into the compartment.[47,48] As ICP increases, the venous flow and eventually arterial flow decrease, leading to further tissue ischemia and necrosis, as well as increased permeability of the damaged blood vessels.[42,45,47] Damage can occur rapidly, with 1 study finding that tissue necrosis occurred in as little as 3 hours following the initial injury.[49]

The anterior compartment of the lower leg is the most common location for ACS.[8,47,50–52] Tibial shaft fractures are a significant risk for this, with 1 study finding an 11.6% incidence of ACS among 212 patients with tibial shaft fractures.[52] In the upper extremity, ACS occurs most frequently in the volar forearm compartment.[53–57] The most common cause of upper extremity ACS are forearm fractures, followed by supracondylar humerus and wrist fractures.[4,58]

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TABLE 1. Potential Causes of ACS[12–41]

Burns
Casts
Crush injury
Deep venous thromboses
Electromyography
Excessive exercise
Extravasation of contrast media or intravenous infusions
Fractures
Incorrect patient positioning during surgery
Infections
Insect bites
Intramuscular hematomas
Intraosseous infusions
Prolonged immobilization
Sickle cell disease
Skeletal traction
Snake bites

Studies have reported an ACS incidence of 0.2% to 1% among isolated forearm fractures,[4,59] 0.2% to 0.3% among supracondylar humerus fractures,[60] and 7% when concurrent supracondylar humerus and forearm fractures are present.[59]

History and Physical Examination

It can be challenging to diagnose ACS in children given the relatively subtle presentations and overlap with pain from the inciting injury.[42] Moreover, younger children may not be able to communicate their symptoms clearly, whereas older children may have concomitant injuries or be under the influence of alcohol or illicit drugs, which can inhibit their ability to provide a reliable history.[3,5,42,43]

Severe pain out of proportion to the examination is usually the first symptom to occur.[61–63] The pain is often described as severe, deep, burning, and exacerbated by passive stretch of the compartment.[43] Unfortunately, many of the symptoms (ie, severe pain, pain with passive stretch, paresthesia, and paresis) have poor sensitivity for the diagnosis, and therefore, their absence cannot be used to exclude the diagnosis.[61] Paresthesias, sensory deficits, and focal motor weakness may be found later in the diagnosis and can suggest a worse prognosis.[47,61,64]

On examination, the patient may have swelling or tenseness of the affected compartment, pain with palpation, pain with passive stretch, focal motor or sensory deficits, decreased pulse, or delayed capillary refill time.[47,61,64] Importantly, digital palpation of the compartment has been found to be unreliable, with 24% sensitivity and 55% specificity for ACS of the leg and 49% sensitivity and 79% specificity for the hand.[65,66] When possible, examinations should be performed serially by the same provider each time.[9,64] An alternate approach is for both providers to see the patient together to establish a baseline examination before care is transitioned between providers.

Given the difficulty with diagnosing this condition in children, some physicians and researchers have proposed replacing the 6 P's (ie, pain, paresthesia, paresis, pallor, poikilothermia, and pulselessness) that are commonly described in adults with the 3 A's (ie, increasing anxiety, agitation, and analgesic requirement).[42]

Diagnostic Testing

Laboratory testing can include a complete blood count, basic metabolic panel, creatinine kinase (CK), urinalysis, and urine myoglobin level. An elevated CK greater than 1000 U/mL or the presence of myoglobinuria is suggestive of ACS, although they may also be seen in rhabdomyolysis.[5,6,11,61] Of note, concurrent rhabdomyolysis can be present in up to 40% of patients with trauma-induced ACS.[6,67,68] Importantly, the absence of an elevated CK does not exclude the diagnosis of ACS. Imaging studies are not typically helpful for diagnosing ACS. However, radiographs are frequently obtained to evaluate for fractures or other potential underlying etiologies.

The diagnosis of ACS requires evaluation of the ICP, which is usually assessed with direct, invasive monitoring.[5,69] One of the most common methods to obtain ICP is the solid-state transducer intracompartmental catheter (STIC) device (eg, Stryker Intra-Compartmental Pressure Monitor System, Kalamazoo, MI).[47,69] An STIC device uses a pressure transducer to directly measure the pressure within the catheter lumen.[70–72] This device has been demonstrated to be highly accurate with a sensitivity of 94% and a specificity of 98%.[62,73,74] If an STIC catheter is not available, a needle attached to an arterial line transducer or pressure manometer may be used as a surrogate.[5,47,61,69,75,76]

When evaluating ICP, providers should target the compartments that correspond to the symptoms first. This should include the muscle compartment that has pain with passive stretch or through which the affected nerves traverse. If a fracture is present, the provider should place the catheter within 5 cm of the fracture level but should not place the transducer within the fracture site, as the latter may falsely elevate the ICP readings.[5,77–81] It is also important to ensure that the transducer is at the same level as the catheter tip, as it may read falsely high or low if placed at the incorrect level.[78–80] If pressures are normal, but the clinical concern remains, providers should consider repeat ICP assessments or measuring the ICP in surrounding compartments, as well.[5,47,61,69]

There is some controversy regarding the critical pressure for diagnosing ACS.[5,47,61,69,82] The normal resting ICP in children is 13 to 16 mm Hg.[83] Consequently, prior literature had recommended using an absolute ICP of 30 to 40 mm Hg as an indication for fasciotomy.[75,82,84,85] However, individuals can vary widely with respect to their ICPs based on the specific extremity and preexisting conditions (eg, hypertension, vascular disease).[5,11,61,75,85–88] As a result, some experts have now suggested that a differential pressure (ΔP) of 30 mm Hg or less should be utilized as an indication for fasciotomy.[5,8,79,89,90] Differential pressure is defined as the difference between the diastolic blood pressure and the ICP.[5]

Isolated single compartment measurements may lead to overdiagnosis and overtreatment if used in isolation, whereas a single measurement cannot identify subsequent changes in ICP.[5,43,62] As a result, several studies have evaluated the use of continuous ICP monitoring. One study found that the combination of continuous ICP monitoring with a ΔP of 30 mm Hg or less in conjunction with clinical symptoms resulted in a sensitivity of 61% and specificity of 97% for ACS.[91] Another study demonstrated that evaluating ICP over 2 hours using ΔP of 30 mm Hg or less as diagnostic criterion had a 94% sensitivity and 98% specificity.[62] Therefore, continuous ICP monitoring should be considered in patients in whom there is not an absolute indication for fasciotomy.

Management

The goal of managing ACS is to reduce the increased ICP, which threatens the limb or affected area. The first step should be to remove any casts or dressings on the affected area, which can reduce ICP by as much as 65% to 85%.[47,69,92,93] It is also important to reduce any displaced fractures, as this can reduce further tissue edema.[47,69] Providers should ensure adequate pain control, but regional blocks are not recommended as they can make it difficult to monitor the patient's symptoms.[27,94] Intravenous fluids should be considered in patients who are hypotensive in order to restore circulating volume and improve perfusion.[47,69]

The definitive treatment for ACS is fasciotomy, which should ideally be performed in the operating room under general or regional anesthesia. Prophylactic antibiotics such as cefazolin, vancomycin, or clindamycin should be administered prior to fasciotomy.[95]

There are many different techniques for fasciotomy, including variations on the number of incisions made. The most commonly affected area in ACS is the leg, for which the fasciotomy is usually performed with 2 incisions placed anterolaterally and posteromedially.[9] For ACS of the forearm, a single volar incision should be made to decompress the carpal tunnel, the superficial compartment, and the deep compartment.[1] Fasciotomy incisions should be large enough to decompress all the affected compartments.[11] All nonviable muscles should be excised.[6] However, surgeons should be cautious when operating on children, because muscle that looks to be necrotic on initial fasciotomy may become viable upon secondary debridement.[96] After the initial surgery, the patient should return to the operating room every 24 to 72 hours for reassessment of tissue viability, debridement, and dressing changes.[43,97]

Wound closure should be delayed for about 1 week to allow approximation of wound edges at closure and facilitate better healing.[98] Fasciotomy wounds can be closed with the assistance of negative-pressure wound therapy, which has been found to reduce the risk of infection.[99] Another alternative is dynamic wound closure using a vascular loop or shoelace technique, which may reduce the need for subsequent skin grafting when compared with wounds closed by negative-pressure wound therapy.[100]

Other potential treatments include indomethacin, mannitol, ultrafiltration of interstitial fluid, and hyperbaric oxygen therapy. Importantly, these should be considered only as adjunctive therapies with fasciotomy in patients diagnosed with ACS. These may also be considered as prophylactic therapies in patients at risk of ACS. Indomethacin acts by reducing the inflammatory response, which may decrease the associated cell damage and improve perfusion.[101] Indomethacin also has the benefit that it can assist with pain management in ACS. Mannitol is more controversial, as there is some evidence that suggests that mannitol lowers ICP and reduces reperfusion injury.[64,102] However, there have also been reported cases of ACS resulting from the mannitol extravasation, so caution must be exercised when using this drug.[103] It is important to ensure that patients remain adequately hydrated when using mannitol because of its diuretic effect.[64] Ultrafiltration of interstitial fluid using percutaneous intramuscular catheters has also been demonstrated to reduce ICP in select cases.[104,105] Finally, hyperbaric oxygen therapy can be considered as it can promote hyperoxic vasoconstriction, which may reduce edema and improve tissue viability.[106] In patients at risk of ACS, hyperbaric oxygen therapy in the first 48 hours has been suggested to improve symptoms and reduce the need for fasciotomy.[46] However, this should not delay fasciotomy or other interventions.[106]

Complications

It is estimated that up to 35% of patients with ACS will experience muscle necrosis within the first 2 hours of injury, and irreversible injury can occur after 6 to 8 hours.[49] Delayed fasciotomy increases the risk of complications, which can include venous insufficiency, cosmetic problems, nerve injury, muscle weakness, and amputation.[107,108] One study of adult patients found that fasciotomy performed within 12 hours of ACS resulted in a 4.5% complication rate, compared with a 54% complication rate among patients who underwent late fasciotomy.[109] The authors also found that 68% of patients who underwent early fasciotomy maintained normal function of the affected extremity, compared with 8% among patients who underwent fasciotomy after 12 hours.[109] Postfasciotomy mortality rates have been reported at 11% to 15%, and amputation rates have been reported at 11% to 21%.[97] Additionally, patients should be monitored for rhabdomyolysis, which can occur in up to half of patients, and can lead to acute renal failure requiring dialysis.[67]

Fortunately, complication rates appear to be lower in the pediatric population. Despite typically delayed time to fasciotomies in this patient population, most patients tend to have good outcomes. In a study of 24 children and adolescents with ACS, the mean time from admission to fasciotomy was 27 hours, whereas the overall complication rate was only 4.2%, with 2 patients having delayed wound healing and 2 patients having peripheral nerve damage.[110] In another study, 43 pediatric patients underwent fasciotomy an average of 20.5 hours after the injury.[111] Of these, 41 patients had no sequelae, whereas only 2 patients suffered loss of function.[111] These patients had a fasciotomy 82.5 and 86 hours after injury.[111] Among a different population of 32 pediatric patients who underwent fasciotomies for ACS, the most common complication was concerns about the scar appearance (23%), followed by neurapraxia (6.7%), stiffness (6.7%), swelling (3.3%), scar pain (3.3%), and weakness (3.3%).[112] Contrary to the adult literature, there have been very few reported cases of infection in pediatric patients who are postfasciotomy.,[111,113]

Disposition

All patients suspected of having ACS or symptoms that may progress to ACS should be admitted for monitoring. Orthopedic surgery or general surgery should be consulted emergently. It is crucial to expedite professional consultation because irreversible damage can occur quickly after symptom onset.[45] Patients diagnosed with ACS should be taken to the operating room for fasciotomy.

Many pediatric patients will experience a rapid recovery and should follow up with their pediatric surgeon until the wounds have healed.[114] Physical therapy and wound care following discharge may minimize stiffness and contractures.[115]

CONCLUSIONS

Acute compartment syndrome is an emergent condition caused by increased ICP within a closed compartment leading to tissue ischemia and necrosis. This article reviews the pathophysiology, historical and physical examination findings, diagnostic strategies, and treatment for this dangerous condition. Knowledge of these components can assist providers in effectively identifying and managing this condition.

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