

Acute and chronic compartment syndromes: Know when to act fast

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ABSTRACT

Compartment syndrome is a fairly common condition noted in patients with fractures or crush injuries to the extremities (acute compartment syndrome) and in athletes (chronic compartment syndrome). Compartments bound by fascia are found in the extremities, buttocks, and abdomen; conditions that cause intracompartmental swelling and hypertension can lead to ischemia and limb loss. This article reviews diagnosis of the problem, monitoring of compartment pressure, and appropriate treatment.

Keywords: compartment syndrome, acute, chronic, crush syndrome, fasciotomy, Volkmann ischemic contracture

Learning objectives

- Identify the clinical presentation of acute and chronic compartment syndromes.
- Differentiate the causes of acute from chronic compartment syndrome.
- Describe the treatment and prognosis for acute and chronic compartment syndromes.

Acute and chronic compartment syndromes are caused by increased intracompartmental pressures, but present with very different clinical scenarios. In both syndromes, high intracompartmental pressure impairs blood flow and causes excessive pain; without treatment, muscle and nerve cells can die.¹ Acute compartment syndrome (ACS) is a medical emergency that must be treated promptly to prevent irreversible muscle damage (Figure 1); chronic compartment syndrome (CCS) is rarely a medical emergency. The literature can be

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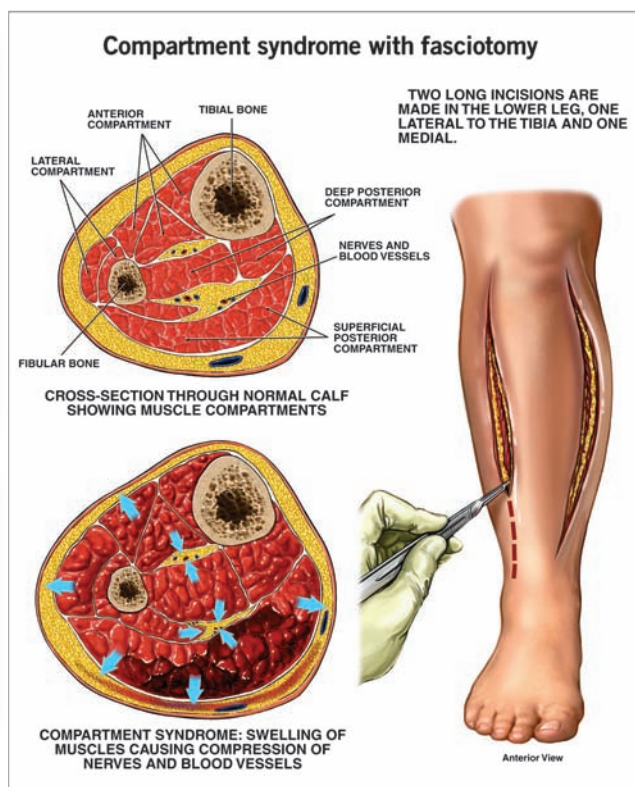


FIGURE 1. A closer look at compartment syndrome

confusing because many terms for these conditions are used interchangeably: acute, subacute, chronic, and recurrent compartment syndrome; crush syndrome; and Volkmann ischemic contracture.²

REVIEWING ANATOMY

Compartments are groupings of muscles, nerves, and blood vessels in the arms and legs.¹ Inelastic fascia covers the compartments, protects the tissue, and maintains the tissue shape.³ Under the proper set of conditions, compartment syndrome can develop in any tissue that is encased in fascia. ACS can affect any compartment; CCS typically affects the compartments of the lower extremities due to repetitive stress during exercise. The anterior compartment of the lower extremity, below the knee, is the most commonly affected site, and tibial fractures are the most common precipitating event.²

Key points

- Compartment syndrome can lead to ischemia and limb loss; acute compartment syndrome is a medical emergency and may require surgical decompression.
- Because of anatomic differences between adolescents and adults, younger patients are at an increased risk of developing compartment syndrome.
- Chronic compartment syndrome can be treated nonemergently as long as compartment pressures decrease when the patient stops the inciting activity.

Because of anatomic differences between adolescents and adults, younger patients are at an increased risk of developing compartment syndrome. Shadgan and colleagues noted that “physicians often believe that younger individuals are more likely than older individuals to have tight, stronger fascia, that there is more muscle filling,” which increases their risk of compartment syndrome.⁴ Also, younger patients may participate in higher-risk activities that can cause traumatic injuries that may lead to ACS. CCS frequently is diagnosed in young adult athletes who participate in repetitive activities such as distance or cross-country running.

Upper extremity The upper arm has two compartments. The anterior compartment contains the biceps and brachialis muscles, and the ulnar, median, and radial nerves; the posterior compartment contains the triceps muscle.³ The forearm has two compartments as well. The volar compartment contains the wrist and finger extensors, and the dorsal compartment contains the flexors of the hand and fingers. The hand has 10 compartments: dorsal interossei (4 compartments), palmar interossei (3 compartments), adductor pollicis, thenar, and hypothenar.²

The original description of ACS is attributed to Richard von Volkmann, who in an 1872 publication, documented nerve injury and upper extremity contracture after a supracondylar fracture.^{2,5} Volkmann ischemic contracture is a serious complication of ACS in the forearm, leading to a permanent flexion contracture of the hand at the wrist, which results in a claw-like deformity of the hand and fingers.

Lower extremity The thigh has three compartments. The anterior compartment contains the vastus lateralis, vastus intermedius, vastus medialis, sartorius, and rectus femoris muscles; the femoral artery; and the femoral nerve.³ The medial compartment contains the adductor longus, adductor brevis, adductor magnus, and gracilis muscles.³ The posterior compartment contains the semimembranosus, semitendinosus, and biceps femoris muscles and the sciatic nerve.³

The lower leg has four compartments. The anterior compartment is the most likely to be affected and most recognizable clinically. It contains the tibialis anterior

muscle, extensor muscles of the toes, anterior tibial artery, and deep peroneal nerve.^{3,4} The lateral compartment contains the peroneus longus and peroneus brevis muscles and the superficial peroneal nerve.³ The deep posterior compartment contains the tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles; the posterior tibial artery; and the tibial nerve.³ The superficial posterior compartment contains the gastrocnemius and soleus muscles and the sural nerve.³

Trunk Three compartments are found in the buttocks. The first contains the fascia lata muscle, the second contains the gluteus medius and gluteus minimus muscles, and the third contains the gluteus maximus muscle and the sciatic nerve.³

PATHOPHYSIOLOGY

ACS and CCS result from increased intracompartmental pressure, which can be caused by a number of precipitating events. The amount of pressure found in a muscular compartment depends on the local blood flow, which is determined by local arterial pressure, venous pressure, and local vascular resistance.² Tissue perfusion is proportional to the difference between the capillary perfusion pressure (CPP) and the interstitial fluid pressure, which is calculated using the formula:

$$\text{local blood flow} = (\text{local arterial pressure} - \text{venous pressure}) / \text{local vascular resistance}^2$$

When the interstitial pressure exceeds the CPP, capillaries begin to collapse, causing ischemia. Ischemic skeletal muscle releases a histamine-like substance that increases vascular permeability, causing blood sludging and worsening ischemic conditions. Myocytes with lyse-releasing proteins cause water to permeate out of arterial blood into the compartment.² Intracompartmental pressure is established by taking a pressure reading of the suspected compartment using a pressure monitor.

CAUSES OF ACS

Any event, external or internal, that increases the pressure within a compartment can cause ACS. Bleeding, either from vascular trauma or from cancellous bone after fractures or osteotomies, is the most common cause of ACS.⁵ Any imbalance between fluid content and/or decrease in compartment size can lead to compartment syndrome.⁵ Traumatic events typically associated with ACS include fractures, gunshot wounds, crush injuries, constricting bandages, and orthopedic surgery.^{2,3,6} Less-common causes related to increased fluid retention include envenomation, burns, rhabdomyolysis, autoimmune vasculitis, androgen abuse/muscle hypertrophy, and deep vein thrombosis.² Patients on anticoagulation therapy also may be at increased risk if excessive bleeding occurs in a closed compartment. Because anticoagulants increase the risk of hemorrhage,

relatively minor traumas can produce conditions favorable for the development of compartment syndrome.

Iatrogenic causes of ACS include tight casts, splints, and dressings; intraosseous infusions; malfunctioning sequential compression devices; and surgical positions such as lithotomy position.^{1-3,7} For reasons that are unclear, compartment syndrome related to surgical positioning typically presents 15 to 24 hours postoperatively.² Likewise, casts should never be placed on an acutely injured extremity because they can constrict a swelling extremity, increasing the risk of ACS.

CAUSES OF CCS

CCS is common in athletes who perform repetitive motions such as running. As the repetitive activity continues, the pressure in the compartment builds, causing pain and paresthesias in the lower extremity and causing the athlete to stop the activity. Once the activity has been discontinued, the pressure in the affected compartment falls to baseline levels and pain or discomfort resolves.

CLINICAL PRESENTATION

Patients suffering from ACS may complain of increased pain when the compartment is stretched actively or passively. Other telltale signs of ACS include pain that is more intense than what would be anticipated with the original injury, paresthesias to the skin, increased palpable tenseness in affected muscle groups, and the late-stage findings of altered sensation and paralysis.¹ The patient may have decreased two-point discrimination due to peripheral nerve compression, but clinical symptoms may be delayed because prolonged pressures greater than 30 mm Hg are needed to produce these findings. Relying on the classic five P's (pain, pallor, pulselessness, paresthesias, and paralysis) may lead to a delay in diagnosis.⁵ Furthermore, arterial pulses may persist indefinitely because compartment pressure is almost always less than systolic BP.^{4,7} In addition, muscle strength is not a good clinical marker as it is difficult to judge in an injured person.⁴

Conversely, patients suffering from CCS will complain of pain and cramping with exercise that might include numbness or difficulty moving the foot, but these symptoms are relieved with rest.¹ In addition, a fascial defect may appear during activity caused by increasing pressure, which might be described as a bulge over the affected muscle. The bulge will typically decrease when activity is halted.

Patients with ACS that has not been promptly treated may develop crush syndrome. This term was first used in 1941, and later reported by Mubarak and colleagues, after physicians observed civilians injured during aerial bombings in World War II who presented with grossly swollen limbs, myoglobinuria, shock, and renal failure.⁸ Crush syndrome was subsequently defined as a systemic manifestation of prolonged extremity compression and compartmental swelling causing muscle necrosis.⁸ The result

is acute renal failure with myoglobinuria, shock, and cardiac complications such as acidosis and hyperkalemia.

DIAGNOSIS

The key to rapid diagnosis and treatment is to recognize clinical symptoms and clues in the patient's history. Another important factor is to have a high suspicion in the face of trauma.

Determining intracompartmental pressure is mandatory in patients with suspected ACS, and requires skin preparation, local anesthetic, and anatomic knowledge. Pressure readings should be taken from each compartment in the injured extremity. Normal compartmental pressures at rest are between 0 and 4 mm Hg. Compartmental pressures higher than 30 mm Hg require surgical decompression (fasciotomy).^{2,6} If high compartmental pressures are left untreated, within 6 to 10 hours the patient can develop muscle infarction, tissue necrosis, nerve injury, loss of limb, and could die.^{2,5,7}

Compartmental pressures between 15 and 20 mm Hg are concerning, and the patient should have follow-up in 12 to 24 hours with an orthopedic surgeon.⁹ Any patient with a compartment pressure above 20 mm Hg should be admitted for observation.⁹ Documented serial physical examinations are imperative if patients are not taken to surgery immediately.⁴ The literature lacks recommendations of intervals between serial examinations, but one author suggests performing these examinations hourly because irreversible damage can occur in less than 3 hours.⁵ Diagnosing ACS can be particularly challenging if the patient has taken large doses of analgesics, is under the influence of alcohol, has an altered level of consciousness, or is a child.^{4,5} Under these clinical situations, a high degree of suspicion and more aggressive diagnostic testing with pressure monitoring may be required.

Diagnosis of CCS also can be challenging because the pressures in the affected compartment only increase with the inciting activity. Therefore, patients with suspected CCS must undergo the activity that produces the symptoms, and diagnosis depends on how quickly the pressures are measured after completion of the activity. As time passes from completion of the activity, pressures will gradually return to baseline. Often, clinicians will forgo the measurement of compartment pressures and treat based on history and physical examination findings.

Falsely elevated pressures may be a result of pressure monitoring needles placed into tendons or fascia, plugged catheters, or faulty monitoring systems.⁶ Falsely low readings may result from bubbles in the lines or transducer, plugged catheters, or faulty monitoring systems.⁵ Being proficient in pressure monitor use is imperative to prevent false positives and negatives.

TREATMENT

ACS must be surgically corrected to prevent muscle and nerve cell death. As mentioned earlier, the definitive

surgical therapy for ACS is fasciotomy, and the goal is to restore muscle perfusion as quickly as possible to prevent irreversible nerve damage and muscle necrosis.¹⁻⁷ Following fasciotomy, fracture reduction or stabilization and vascular repair can be performed, if needed. Fasciotomies of the suspected compartments are performed in an OR. Tissue debridement is performed at that time, and the surgical wound is left open and packed with sterile gauze. The patient may return to the OR for further debridement as needed. This sequence of events may be repeated until all necrotic tissue has been removed.

Lower-limb four-compartment fasciotomy can be accomplished using one or two incisions. The most commonly used two-incision technique involves an anterolateral and posteromedial incision.⁴ The single-incision method requires an incision over the fibular axis extending 5 cm below the fibular head to 5 cm above the lateral malleolus.⁴ Both methods can cause a poor cosmetic outcome. With either method, the length of the skin incision should extend the length of the muscle bulk of the affected compartments, as skin can continue to cause intracompartment pressure despite an adequate fascial release.⁴

The skin closure will loosely approximate the skin edges but leave the fascia open. The final closure can be accomplished with many different methods, including delayed primary closure, split-thickness skin grafts, dermatotraction, and negative-pressure wound therapy.⁴ Although expensive, negative-pressure wound therapy not only helps reduce tissue swelling, but also prevents retraction of wound edges, increasing the chances of delayed primary closure.^{5,10} Some patients may need skin grafting for large dermal defects. If fasciotomies are delayed, limb amputation may be the only surgical option if significant muscle ischemia has occurred.

CCS can be treated nonemergently as long as compartment pressures decrease when the patient stops the inciting activity. Conservative treatment such as physical therapy, nonsteroidal anti-inflammatory or diuretic medications, and orthotics may help decrease the symptoms. If conservative treatment fails, the patient will need a fasciotomy. Unlike with ACS, fasciotomies for CCS usually require shorter incisions, serial debridements are not needed, and procedures are elective.

PROGNOSIS

The prognosis for a patient with ACS depends on the underlying cause: A patient with multiorgan system damage due to trauma will have a worse overall prognosis than a patient whose ACS was caused by an isolated fracture. In a study following 26 patients who underwent fasciotomies for ACS, about 15% complained of pain at rest and 27% reported pain with exertion 1 to 7 years after trauma.¹¹ Infections due to fasciotomy have been reported in about 38% of patients.¹²

Patients with CCS who have surgery generally have an excellent prognosis. One study showed that 96% of patients who underwent fasciotomy reported unlimited

exercise after a mean follow-up of 5 years.¹³ A separate case series of 11 athletes with CCS found that they exhibited preinjury levels of athletic conditioning at 2 years postsurgical intervention.¹⁴

CONCLUSION

Compartment syndrome is a serious condition that may present in the ED, OR, or primary care clinic. The condition may be acute or chronic; complications of ACS include crush syndrome and limb disabilities if ACS is not promptly diagnosed and managed. Clinicians should obtain a thorough patient history and have a high index of suspicion for compartment syndrome based on patient presentation and mechanism of injury. Prompt diagnosis and management are the keys to reducing morbidity in the face of a potentially serious problem. **JAAPA**

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REFERENCES

1. American Academy of Orthopedic Surgeons. <http://orthoinfo.aaos.org/topic.cfm?topic=a00204>. Accessed August 24, 2013.
2. Rasul A, Lorenzo C, Agnew S, et al. Acute compartment syndrome. <http://emedicine.medscape.com/article/307668-overview>. Accessed August 25, 2013.
3. Cline DM, Ma OJ, Tintinalli JE, et al. *Emergency Medicine, A Comprehensive Study Guide, Companion Handbook*. 4th ed. New York, NY: McGraw Hill; 1996:881-884.
4. Shadgan B, Menon M, Sanders D, et al. Current thinking about acute compartment syndrome of the lower extremity. *Can J Surg*. 2010;53(5):329-334.
5. Frink M, Hildebrand F, Krettek C, et al. Compartment syndrome of the lower leg and foot. *Clin Orthop Relat Res*. 2010;468(4):940-950.
6. Rappolo L, Davis D, Kelly S, Rosen P. *Emergency Medicine Handbook: Critical Concepts for Clinical Practice*. Philadelphia, PA: Mosby-Elsevier; 2007.
7. Jagminas L, Shraga E, Becker J, Windle M. Compartment pressure management. <http://emedicine.medscape.com/article/140002-overview>. Accessed August 25, 2013.
8. Mubarak S, Owen CA. Compartmental syndrome and its relation to the crush syndrome: a spectrum of disease. A review of 11 cases of prolonged limb compression. *Clin Orthop Relat Res*. 1975;(113):81-89.
9. Ma J, Cline DM, Tintinalli JE, et al. *Emergency Medicine: Just the Facts*. 2nd ed. New York, NY: McGraw-Hill; 2004:604-605.
10. Wall CJ, Richardson MD, Lowe AJ, et al. Survey of management of acute, traumatic compartment syndrome of the leg in Australia. *ANZ J Surg*. 2007;77(9):733-737.
11. Frink M, Klaus AK, Kuther G, et al. Long term results of compartment syndrome of the lower limb in polytraumatized patients. *Injury*. 2007;38(5):607-613.
12. Johnson SB, Weaver FA, Yellin AE, et al. Clinical results of decompressive dermatomy-fasciotomy. *Am J Surg*. 1992;164(3):286-290.
13. de Fijter WM, Scheltinga MR, Luiting MG. Minimally invasive fasciotomy in chronic exertional compartment syndrome and fascial hernias of the anterior lower leg: short- and long-term results. *Mil Med*. 2006;171(5):339-403.
14. Mouhsine E, Garofalo R, Moretti B, et al. Two minimal incision fasciotomy for chronic exertional compartment syndrome of the lower leg. *Knee Surg Sports Traumatol Arthrosc*. 2006;14(2):193-197.