KEY POINTS

- Severe pain out of proportion to the clinical situation is the main feature of acute compartment syndrome and often the only early finding.
- Early diagnosis, consultation, and treatment are the keys to a good outcome.

EPIDEMIOLOGY

The true incidence of acute compartment syndrome (CS) varies with the inciting event. Almost half of all cases of CS are related to tibia fractures. The incidence of CS with tibia fractures is 1.2% with closed fractures, 6% with open fractures, and as high as 19% with concomitant vascular injury. The forearm is the second leading site. CS can occur in any contained compartment.

PATHOPHYSIOLOGY

CS is a condition of impaired microcirculatory perfusion related to increased interstitial pressure within a closed compartment. CS begins when increased pressure as a result of increased volume within or external pressure compromises microcirculatory perfusion within that space. Once autoregulatory reductions in the arteriovenous gradient are overwhelmed, interstitial pressure will rise above capillary perfusion pressure (normally between 20 and 30 mm Hg in a normotensive patient for most compartments), and tissue ischemia will occur. CS is characterized by a self-propagating cycle of impaired perfusion resulting in ischemia, release of osmotically active particles, and edema, which further increase interstitial pressure and diminished perfusion (Fig. 91.1).

PRESENTING SIGNS AND SYMPTOMS

CS should be on the working list of “worst-case” diagnoses for every patient with musculoskeletal pain. CS may result from either externally applied compressive force or internally expanding force, and a suggestive history should be elicited (Box 91.1). Myofascial causes include long-bone fracture, vascular injury, reperfusion after ischemia, burns, prolonged positioning from a drug overdose or operating procedures, compression from tight casts and dressings (including military antishock trousers), overexertion, hemorrhage, injection of fluid into the compartment, massive intravenous fluid infusions, envenomations, hypothyroidism, and rhabdomyolysis. CS has also been reported with deep vein thrombosis and ruptured Baker cysts (see Box 91.1). High-risk patients with an altered sensorium may be unable to provide an appropriate history. CS usually develops hours after the inciting event and rarely more than 48 hours after the inciting event.

Clinicians must remain vigilant and retain a high index of suspicion to avoid missing the diagnosis. Patients with a high mechanism of trauma and an altered sensorium should be examined carefully. The physical examination should focus on evidence of trauma and gross deformity, as well as assessment of neurovascular abnormalities. Comparing one extremity with the unaffected side is often very useful.

Serial examinations are often required.

Acute CS is a clinical diagnosis. The essential clinical feature in a conscious patient is severe pain out of proportion to the injury that is aggravated by active or passive stretching of the muscles of the affected compartment or by palpation of the affected compartment. In early cases, pain is the only abnormality. Increasing pain or pain refractory to analgesics suggests the diagnosis. Severe pain while at rest or without any movement should raise suspicion for acute CS. Increasing need for analgesia or increased analgesic dosing is common.

The diagnosis is far more challenging in patients who cannot communicate (i.e., altered mental status).

The natural progression of untreated acute CS is severe pain, decreased sensation, decreased strength, and eventual paralysis of the affected limb. With the exception of pain and paresthesia, the traditional five P’s (pain, paresthesia, pallor, pulselessness, poikilothermia) are misleading and more relevant for arterial injury or occlusion. Patients with severe CS, even those with extensive myonecrosis, may have palpable pulses and preserved capillary refill until late in the course. Pulse deficits should raise suspicion for a vascular injury alone or coexisting with acute CS. Decreased two-point discrimination is consistently the earliest physical abnormality and can help differentiate which compartments are affected. Correlation has also been reported between decreasing vibratory sense (256 cycles per second) and increasing compartment pressure.
The differential diagnosis includes any disorder that can cause musculoskeletal pain, including fracture, contusions, and hematomas; however, pain from other causes generally diminishes after the inciting event, whereas in acute CS, the pain generally increases even while the patient is at rest. Arterial injury will be manifested as abnormal pulses at the onset. Crush injury shares some overlapping features with CS, but the primary pathophysiology is thought to be direct tissue injury, and fasciotomy is rarely indicated.

The diagnosis cannot be made with radiographic imaging. However, because the differential diagnosis for acute CS includes fracture or dislocation and because the risk for CS increases with fracture, radiographic imaging may be useful. Computed tomography or ultrasound is rarely indicated but may be helpful in establishing the cause of the CS (i.e., fracture, hematoma).

**TREATMENT**

**PREHOSPITAL MANAGEMENT**

The affected limb should be placed at the level of the heart. Elevation is contraindicated because it decreases arterial flow, which narrows the arteriovenous pressure gradient and thus worsens ischemia. Circumferential bandages should be removed, and casts should be removed or split. Because hypotension potentiates CS, it should be corrected with crystalloid or blood products. Supplemental oxygen should be administered routinely to improve tissue oxygenation.

**HOSPITAL MANAGEMENT**

Surgical specialty evaluation is mandatory in suspected cases because the therapy for CS is usually surgical decompression. Full trauma evaluation and correction of hypotension, anemia, and coagulopathy, as well as preoperative assessment, are expected. CS can cause rhabdomyolysis, acute renal failure, and death, and these complications should be anticipated and treated accordingly.

Measurement of intracompartmental pressure is not necessary if the diagnosis of acute CS is clinically apparent. Equivocal cases may require further evaluation. Multiple methods and devices for measuring compartmental pressure are available. Devices that use a side-ported needle or slit catheter are recommended rather than those using a simple needle.

Normal intracompartmental pressure is in the range of 0 to 10 mm Hg. Pain and paresthesias are common at 20 to 30 mm Hg, and ischemia generally ensues at pressures greater than 30 mm Hg. Many surgeons traditionally consider a measured compartment pressure of 30 mm Hg or higher as an indication for fasciotomy, although higher values have been used, especially for the thigh. More recently, some authors have recommended the use of a ΔP value (diastolic blood pressure minus the measured intracompartmental pressure) of less than 30 to 50 mm Hg as an indication for fasciotomy and have found it to be more reliable than an absolute compartment pressure (Box 91.2).

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The differential diagnosis includes any disorder that can cause musculoskeletal pain, including fracture, contusions, and hematomas; however, pain from other causes generally diminishes after the inciting event, whereas in acute CS, the pain generally increases even while the patient is at rest. Arterial injury will be manifested as abnormal pulses at the onset. Crush injury shares some overlapping features with CS, but the primary pathophysiology is thought to be direct tissue injury, and fasciotomy is rarely indicated.

The diagnosis cannot be made with radiographic imaging. However, because the differential diagnosis for acute CS includes fracture or dislocation and because the risk for CS increases with fracture, radiographic imaging may be useful. Computed tomography or ultrasound is rarely indicated but may be helpful in establishing the cause of the CS (i.e., fracture, hematoma).

**PRIORITY ACTIONS**

Suspect acute compartment syndrome when a patient has severe pain, especially with passive movement, but few other objective physical findings.

Measure compartment pressures when indicated.

Consult the orthopedic or general surgery department.

Treat hypotension and anemia, provide oxygen, and reverse coagulopathy if present.

The affected compartment should be positioned at the level of the heart.

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

All patients with acute CS must be admitted to the hospital. Most will be transferred to the operating room for emergency fasciotomy or surgical decompression. Patients with abnormal
**CHAPTER 91  ACUTE COMPARTMENT SYNDROMES**

**BOX 91.2 Management of Acute Compartment Syndrome**

- Consult surgery.
- Maintain the affected area at the level of the heart.
- Reverse hypoperfusion/hypotension.
- Maximize oxygenation/administer supplemental oxygen.
- Correct coagulopathy if present.
- Correct anemia if present.
- Modify the cast, splint, or dressing if this is the precipitant of the syndrome.
- Order antivenom if envenomation is the cause of the syndrome.

compartment measurements that do not reach the threshold for emergency surgical decompression should be admitted for observation. Patients with suspected impending acute CS should be admitted for serial examinations and observation.

The prognosis depends on early diagnosis and treatment. Severe disability, amputation, and even death may occur when the diagnosis is delayed or missed. Pitfalls include failure to make the diagnosis or consult surgery when indicated.

**DOCUMENTATION**

**History:** A careful history will include the mechanism, timing, history of paresthesia or weakness, medical conditions that may impair oxygenation or perfusion, coagulopathy or anticoagulation therapy, intravenous drug use, increasing pain, and pain with active or passive movement.

**Physical examination:** Including neurovascular signs, compartment palpation, discoloration, masses, range of motion.

**Medical decision making:** Reasons to pursue or not pursue work-up or consultation.

**Patient teaching:** Patients with significant extremity pain but who do not have clinical evidence of acute compartment syndrome should be counseled to avoid activity, maintain the extremity at the level of the heart, and return if any increase in pain occurs.

**SPECIAL CIRCUMSTANCES**

**ACUTE ORBITAL COMPARTMENT SYNDROME**

Acute orbital CS is considered a rare complication of facial trauma (usually blunt) or surgery. The globe and retrobulbar contents are encased in a continuous cone-shaped fascial envelope that is bound on all sides by seven rigid bony walls, except anteriorly, where the orbital septum and eyelids form another, fairly inflexible boundary. The medial and lateral canthal tendons attach the eyelids to the orbital rim and limit forward movement of the globe.

The orbit may compensate for small increases in orbital volume by forward movement of the globe and prolapse of fat, followed immediately by a rapid rise in orbital tissue pressure. The orbit therefore follows pressure-volume dynamics with a pathophysiology akin to that with CS in other compartments: increased tissue pressure in an enclosed space is associated with decreased perfusion. Ischemia ensues when orbital pressure exceeds central retinal artery pressure.

Symptoms and signs include eye pain, visual loss, proptosis, reduction of ocular mobility, chemosis, and (late) afferent pupillary defect. Diagnosis requires high clinical suspicion and may require serial examinations, including visual acuity tests.

Suspected acute orbital CS with a decrease in vision, loss of vision with increasing intraocular pressure, or high suspicion in a comatose patient requires treatment to prevent permanent blindness. Computed tomography or magnetic resonance imaging is not necessary to make the diagnosis. Irreversible optic nerve pathology may occur with as little as 2 hours of ischemia.

Medical therapy and ophthalmologic consultation should proceed promptly, before the diagnosis is established. Osmotic agents and carbonic anhydrase inhibitors are part of established protocols at many centers. Most experts also recommend high-dose steroid therapy. Less agreement exists about the use of topical beta-blockers and multiple osmotic agents.

The emergency procedure of choice for loss of visual acuity associated with acute orbital CS is lateral canthotomy and cantholysis of the canthal ligaments (Box 91.3; Figs. 91.2 and 91.3; also see the Tips and Tricks box). Primary indications for lateral canthotomy and cantholysis include intraocular pressure greater than 40 mm Hg with visual loss.

**Fig. 91.2 Lateral canthotomy.**

**BOX 91.3 Primary Indications for Lateral Canthotomy/Cantholysis**

- Suspected orbital compartment syndrome with one or both of the following:
  - Decreasing vision
  - Increasing intraocular pressure
- When vision cannot be assessed and clinical suspicion is high (e.g., a comatose patient)

**Primary Indications for Lateral Canthotomy/Cantholysis**

- Suspected orbital compartment syndrome with one or both of the following:
  - Decreasing vision
  - Increasing intraocular pressure
- When vision cannot be assessed and clinical suspicion is high (e.g., a comatose patient)
and proptosis, which may be used as a criterion for an unconscious patients whose visual acuity cannot be determined. Secondary criteria include an afferent pupillary defect, ophthalmoplegia, cherry-red macula, optic nerve head pallor, and severe pain, but these signs are all considered less sensitive or very late.

A contraindication to lateral canthotomy is a suspected ruptured globe. In an experimental model, lateral canthotomy/cantholysis produced a mean decrease in intraocular pressure of 30.4 mm Hg. Emergency department personnel should be familiar with and able to perform this procedure in the event that emergency ophthalmology consultation is delayed.

**ABDOMINAL COMPARTMENT SYNDROME**

Abdominal CS is a sudden increase in intraabdominal pressure that results in dysfunction of the respiratory, cardiovascular, and renal systems. Normal intraabdominal pressure is 0 to 5 mm Hg. Acute abdominal CS was defined by the 2004 International Abdominal Compartment Syndrome Consensus Definitions Conference Committee as sustained intraabdominal pressure greater than 20 mm Hg that is associated with new organ dysfunction or failure. It is most common after abdominal surgical procedures but can also occur with peritonitis, intraabdominal abscesses, intestinal obstructions, ruptured abdominal aneurysms, acute pancreatitis, intraperitoneal or retroperitoneal hemorrhage, ascites, ovarian tumors, and massive edema following resuscitation.

The diagnosis depends on high clinical suspicion combined with the presence of clinical parameters, including intraabdominal pressure elevated to greater than 20 to 25 mm Hg (most commonly assessed with a device used to measure bladder pressure), a distended abdomen, elevated peak airway pressure, large intravenous fluid requirements, elevated central venous pressure, oliguria or anuria not responding to volume repletion, decreased cardiac output, hypoxemia, hypercapnia, acidosis, and a wide pulse pressure.

Treatment consists of rapid surgical decompression, as well as restoration of intravascular volume, maximization of oxygen delivery, and correction of acidosis and coagulopathies. Mortality associated with abdominal CS can exceed 50%.

**CHRONIC COMPARTMENT SYNDROME**

Chronic CS was first described in 1956 and initially thought to be a form of shin splints (anterior tibial enthesitis). Chronic CS (also known as exertional or recurrent CS) is not a surgical emergency. It is commonly reproducible with a certain specific exercise or exercise distance. Symptoms subside with termination of the exercise and are minimal with normal daily exercise. When suspected, patients should be advised to rest and be referred to an orthopedic or sports medicine specialist.

**SUGGESTED READINGS**


**REFERENCES**

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES