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Acute Compartment Syndrome of the Lower Leg: A Review

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Abstract

Acute compartment syndrome (ACS) of the lower leg is a time-sensitive orthopedic emergency that relies heavily on precise clinical findings. Late findings of ACS can lead to limb amputation, contractures, paralysis, multiorgan failure, and death. Hallmark symptoms of ACS include the 6 P's: pain, poikilothermia, pallor, paresthesia, pulselessness, and paralysis. Suspicion of ACS is confirmed by measurement of intracompartmental pressure of the affected compartment. The definitive treatment of ACS is timely fasciotomy. We review the pathophysiology, common causes, diagnosis, and treatment of this potentially devastating condition.

Keywords

acute compartment syndrome; delta pressure; intracompartmental pressure; orthopedic; Stryker

Introduction

Acute compartment syndrome (ACS) of the lower leg is a limb-threatening condition and a surgical emergency. Lower leg ACS is a condition in which increased pressure within a muscle compartment surrounded by a closed fascial space leads to a decline in tissue perfusion and compromises motor and sensory function.¹⁻³ In addition to muscle, key structures within the fascial compartment that are affected by increased compartment pressures include nerves and vasculature.¹ Importantly, delay in diagnosis or treatment can result in irreversible damage to these components with devastating and permanent complications such as contractures, paralysis, amputation, sepsis with multi-organ failure, and even death.^{1, 4, 5}

The average annual overall incidence of ACS is 3.1 per 100,000 people, and is found higher in males than females.^{1, 3, 6} Fracture, caused by trauma, accounts for approximately 75% of ACS cases.³ Early recognition of ACS is critical and requires a high index of clinical

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suspicion based on a thorough knowledge of risk factors, pathophysiology and clinical manifestations.^{1, 7, 8} In addition, accurate intra-compartmental pressure (ICP) measurement and serial monitoring of changes in ICP levels within the muscle compartment is necessary to confirm ACS.⁹⁻¹¹ Last, prompt intervention and treatment with fasciotomy will ultimately prevent permanent damage to the extremity. Despite this, ACS is one of the most highly litigated conditions in orthopedic surgery with awards as high as 14 million dollars for failure to diagnose and adequately manage ACS.^{2, 8}

Common causes of ACS

The most common types of injury contributing to the development of ACS are trauma related tibial shaft fracture (36%), followed by soft-tissue injury (23%) and forearm fractures (> 9%).^{2, 3, 9} However, a variety of other etiologies, including non-traumatic causes, can contribute to ACS and are listed in Table 1. There are atypical causes of ACS that nonorthopedic providers need to be cognizant of. Male patients are ten times more impacted by ACS than females, and this may be explained by males having larger muscle mass within a fixed compartment.^{3, 8, 12} Younger patients (< 35 years of age) are also at a greater risk to ACS due to having tighter fascia and larger muscle mass and as they are prone to injuries or accidents.^{3, 8, 12}

Pathophysiology

Although the pathophysiology of ACS remains unclear, a primary hypothesis behind the development of compartment syndrome is the Arteriovenous Pressure Gradient theory (APG).^{3, 6} The APG principle proposes that ACS occurs when blood flow does not meet the metabolic demands of affected tissues subsequently resulting in tissue ischemia. This leads to a series of events to ensue, including increased inflammation, arterial spasm, disrupted capillary flow, increased osmotic pressure, proteinaceous exudate, muscle fiber swelling, and edema.^{1, 3, 6, 11} If not intervened upon, this cycle self-propagates and contributes to further increased ICP. Bleeding that may be present from an injury further contributes to the build-up of pressure within the compartment. Consequently, there is continual rise in edema within the compartment enclosed in an inextensible fascia, leading to tighter compartments and higher ICP. Eventually, when tissue and venous pressure compromises capillary perfusion, muscle and nerve ischemia ensues. If the compartment is not surgically decompressed, the pressure-induced ischemia results in tissue and cellular necrosis and irreversible damage.^{1, 3, 6, 11}

Anatomy of the lower leg

There are four compartments in the lower leg and these include the anterior, lateral, superficial posterior and deep posterior compartments (Figure 1 and Table 2).^{2, 11, 13} Each compartment contains specific nerves, arteries and veins, muscles, and bony structures that with injury contribute to the unique clinical presentations in ACS. Knowledge about the most important structures within these compartments is critical to efficiently assess and diagnose physiologic changes in ACS that contribute to pathologic development.^{2, 3, 8, 9}

Diagnosis

Diagnosis of ACS is based largely on physical examination and six cardinal clinical manifestations described as the six P's.^{1-3, 6, 8} The six P's include: (1) Pain, (2) Poikilothermia, (3) Paresthesia, (4) Paralysis, (5) Pulselessness, and (6) Pallor.^{1-3, 8} The earliest indicator of developing ACS is severe pain. Pulselessness, paresthesia, and complete paralysis are found in the late stage of ACS. Additionally, serial measurement of ICP is critical in confirming and determine progression of ACS.^{1-3, 9} Accurate ICP measurement is especially important when assessing for ACS in patients who are incapacitated or unable to provide reliable answers. Vigilant monitoring of ICP is also critical in patients where epidural anesthesia is in use as motor and sensory symptoms of ACS can be easily masked.^{10, 14, 15}

Other diagnostic considerations including the use of ancillary testing such as laboratory testing or imaging is briefly discussed below. Lastly, it is important to consider differential diagnoses while assessing for ACS including the possibilities of cellulitis, deep vein thrombosis, neuropraxia, or peripheral arterial injuries.¹⁶

The six P's

All characteristics of the six P's may not be present in every individual. Furthermore, presentation of these symptoms will vary depending on time that has lapsed since the initial pressure began to rise, the rate of ICP increase, blood pressure, and damage within the compartment.²

Pain^{2, 6, 8}—As ACS progresses, the extremity becomes edematous and tense.² There is increased pressure placed on nerve fibers and injured components within the compartment.² Pain is characteristically described as being out of proportion to the injury with passive stretching.

Pulselessness and Pallor^{2, 6, 8}—A late finding is pulselessness which is a poor indicator of ACS, whereas pallor is a less common finding.^{6,8} Arterial insufficiency is atypical in the early stages of ACS thus both dorsalis pedis and posterior tibial pulses are palpable; capillary refill is brisk and the extremity is typically pink. However, as ICP rises, loss of limb pulses and pallor indicates compression of arterial perfusion.

Paresthesia and Paralysis^{2, 6, 8}—As ICP increases, neuronal tissues become ischemic and this contributes to nerve dysfunction and paresthesia, paresis, and ultimately paralysis. Paresthesia may occur within 30 minutes following injury to nerves.² Motor function may deteriorate within four hours of muscle tissue ischemia.² At eight to 24 hours of ischemia, functional losses may be irreversible.^{2,3} The loss of light touch sensation commonly emanating from increased pressure on the deep peroneal nerve often precedes limb weakness. Light touch assessment can be assessed using two-point discrimination or pin prick testing.

Poikilothermia—Poikilothermia is described as a change in temperature or the presence of coolness in the affected extremity.¹¹

Intra-Compartmental Pressure Monitoring

Pressure^{2, 6, 8}—Normal resting limb ICP is 0–4 mmHg.⁶ With exertion, typical limb ICP may increase up to 10 mmHg.⁶ With ACS, an ICP of 30 mmHg or above is considered critical and treatment with emergent surgical decompression should be considered.⁶ Time is of essence to salvage motor and sensory function of the limb. Within eight hours of an ICP at 30 mmHg, nerve conduction is disrupted. However, the higher the ICP, the quicker damage to compartment structures occurs (at an ICP of 80 mmHg, damage to limb compartments could occur within two hours).⁶

The most common and validated method to measure limb ICP is by using the handheld Stryker Intra-Compartmental Pressure (STIC) Monitor System (Figure 2; Stryker Instruments, Kalamazoo, MI).^{2, 6, 8} This device assists medical professionals with the diagnosis of compartmental syndrome. Use of the STIC monitor involves injection of saline into the compartment of interest. The system is designed to measure tissue fluid pressure. An alternative method to determine limb ICP is the use of a delta pressure (ΔP). The ΔP is defined as the difference between the diastolic blood pressure and measured compartment pressure.¹ A ΔP of ≥ 30 mmHg is diagnostic of ACS.¹

In Table 2 we present key structures within the four compartments with associated assessment findings that may be altered with injury and increased ICP.

Ancillary testing

Laboratory tests—The use of blood or urine testing to assess degree of muscle damage (the presence of rhabdomyolysis) can be helpful in the clinical assessment of ACS.

The most common of these tests include measurements of serum creatinine phosphokinase (CPK) and the presence of urine myoglobin.³ The normal level of blood CPK is <130 international units (IU).³ In ACS, levels of CPK may exceed 1000 international units (IU). Severe rhabdomyolysis may lead to acute kidney dysfunction and failure.³

Vascular and Noninvasive Testing—Angiography may help identify focal arterial or venous injury and perfusion defects contributing to increased ICP.³ Additionally, the use of magnetic resonance imaging (MRI), ultrasonography, and near-infrared spectroscopy or laser doppler flowmetry have also been evaluated in diagnosing ACS as an adjunct to clinical findings and ICP measurement but not in the primary diagnosis of ACS.^{3, 9} MRI is favorable for chronic conditions of exertional compartment syndrome; while, ultrasonography allows for noninvasive, serial monitoring of compartment pressures. Near-infrared spectroscopy or laser doppler flowmetry is commonly used to determine oxygenation in muscles and decreases as ICP increases; however, its use is problematic in hypoperfused conditions.^{3, 9}

Treatment

The standard treatment for ACS is emergent surgical fasciotomy. Fasciotomies can vary in surgical technique. Currently, both single and double incision techniques are used by surgeons.^{3, 9} The single incision technique involves a single long incision made from the

head of the fibula to the lateral malleolus.⁹ However, the most common fasciotomy method performed is the double-incision, four compartment technique incorporating two longitudinal anterolateral and posteromedial incisions.^{8, 9} Important principles of fasciotomy include: (1) an adequate length, depth and accurate landmark of incision, (2) full release of the constricted compartment, (3) avoidance in injuring major underlying structures such as the superficial peroneal nerve, (4) debridement of ischemic and necrotic tissue, (5) return to the operating room every 24 to 72 hours for dressing changes, debridement as necessary and assessment of tissue viability, and last (6) closure of skin (skin grafting may be necessary) and incision within 7 to 10 days.^{1, 2}

Nonoperative treatment

Whenever safe and possible, simple treatment measure in ACS include loosening ace wraps, compression dressings, splints and uni- or bivalving casts.⁶ Elevation of extremity no higher than the level of the heart facilitates venous drainage, reduces edema and maximize tissue perfusion.⁶ Further, avoidance of knee flexion and foot dorsiflexion will facilitate uncompromised circulation throughout a limb and limit increases in ICP in the deep posterior compartment respectively.⁶

Postoperative care

Postoperative care following ACS fasciotomy focuses on the following: (1) completion of frequent neurovascular examinations to ensure both adequate release of the affected compartment and that no new damages were incurred during the operative procedure, (2) tissues, if left open, are pink and viable, (3) use of negative pressure devices such as the wound vacuum assisted device (V.A.C.®; Kinetics Concepts Inc., San Antonio, Texas) to facilitate sealing of wound and removal of wound exudate, and last (4) control of swelling.²

Conclusion

Acute compartment syndrome is a rare complication but serious orthopedic surgical emergency. ACS creates a myriad of symptoms that signal increased pressure in an affected muscle compartment resulting to compromised tissue perfusion. Clinical diagnosis of ACS can be difficult and is based largely on a thorough neurovascular assessment (the six P's) and a high clinical suspicion. ICP monitoring, used accurately, is an objective and valid test to confirm ACS diagnosis particularly in patients in whom subjective clinical assessment is unclear. Early consultation and collaboration with an orthopedic, vascular or general surgery team is critical for limb salvage. Timely surgical intervention with a fasciotomy is the primary treatment for ACS preventing potentially devastating and permanent downstream complications such as contractures, paralysis, amputation, multi-organ failure, and even death.

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Reference

1. Donaldson J, Haddad B, Khan WS. The pathophysiology, diagnosis and current management of acute compartment syndrome. *Open Orthop J*. 2014; 8:185–193. [PubMed: 25067973]
2. Bowyer, MW. Compartment Syndrome.. In: Gahtan, V.; Costanza, MJ., editors. *Essentials of Vascular Surgery for the General Surgeon*. Springer; New York: 2014. p. 55-69.
3. Mauser N, Gissel H, Henderson C, Hao J, Hak D, Mauffrey C. Acute lower-leg compartment syndrome. *Orthopedics*. 2013; 36:619–624. [PubMed: 23937740]
4. Blanchard S, Griffin GD, Simon EL. Atraumatic painless compartment syndrome. *Am J Emerg Med*. 2013; 31:1723, e1723–1724. [PubMed: 24041642]
5. Shuler FD, Dietz MJ. Physicians' ability to manually detect isolated elevations in leg intracompartmental pressure. *J Bone Joint Surg Am*. 2010; 92:361–367. [PubMed: 20124063]
6. Konstantakos EK, Dalstrom DJ, Nelles ME, Laughlin RT, Prayson MJ. Diagnosis and management of extremity compartment syndromes: an orthopaedic perspective. *Am Surg*. 2007; 73:1199–1209. [PubMed: 18186372]
7. Wardi G, Gortz S, Snyder B. A case of delayed presentation of thigh compartment syndrome. *J Emerg Med*. 2014; 46:e145–148. [PubMed: 24560014]
8. Raza H, Mahapatra A. Acute compartment syndrome in orthopedics: causes, diagnosis, and management. *Adv Orthop*. 2015; 2015:543412. [PubMed: 25688303]
9. Shadgan B, Menon M, Sanders D, et al. Current thinking about acute compartment syndrome of the lower extremity. *Can J Surg*. 2010; 53:329–334. [PubMed: 20858378]
10. Vegari DN, Rangavajjula AV, Diiorio TM, Parvizi J. Fasciotomy following total knee arthroplasty: beware of terrible outcome. *J Arthroplasty*. 2014; 29:355–359. [PubMed: 23786987]
11. Murdock M, Murdoch MM. Compartment syndrome: a review of the literature. *Clin Podiatr Med Surg*. 2012; 29:301–310. viii. [PubMed: 22424490]
12. Dover M, Memon AR, Marafi H, Kelly G, Quinlan JF. Factors associated with persistent sequelae after fasciotomy for acute compartment syndrome. *J Orthop Surg (Hong Kong)*. 2012; 20:312–315. [PubMed: 23255636]
13. Thompson, JC. Thompson JC, eds. *Netter's concise orthopaedic anatomy*. 2nd ed.. Saunders Elsevier; Philadelphia, PA: 2010. Leg/Knee.; p. 285-336.
14. Hailer NP, Adalberth G, Nilsson OS. Compartment syndrome of the calf following total knee arthroplasty--a case report of a highly unusual complication. *Acta Orthop*. 2007; 78:293–295. [PubMed: 17464621]
15. Tang WM, Chiu KY. Silent compartment syndrome complicating total knee arthroplasty: continuous epidural anesthesia masked the pain. *J Arthroplasty*. 2000; 15:241–243. [PubMed: 10708095]
16. Iannotti, JP.; Parker, RD. Etiology of Compartment Syndrome.. In: Iannotti, JP.; Parker, RD., editors. *The Netter collection of medical illustrations: Musculoskeletal System, Volume 6, Part III - Biology and Systemic Diseases*. 2nd ed.. Elsevier; Philadelphia, PA: 2013. p. 284-90.

Highlights

- Acute Compartment Syndrome (ACS) of the lower leg is a time sensitive limb threatening surgical emergency.
- Late findings of ACS can lead to limb amputation, contractures, paralysis, multi-organ failure and death.
- Diagnosis is based on clinical suspicion, assessment of the six P's (pain, poikilothermia, pallor, paresthesia, pulselessness and paralysis) and intracompartmental pressure (ICP).
- ICP measurement above 30mmHg is considered critical and treatment with emergent surgical decompression should be considered.
- The gold standard of acute compartment treatment is full fasciotomy.

Figure 1. Cross-section of the lower leg depicting the 4 compartments and select key structures

Figure 1. Springer and the original publisher of Essentials of Vascular Surgery for the General Surgeon, eds. Gahtan V and Costanza MJ, 2014©, Compartment Syndrome, Bowyer MW, figures 4.5 and 4.7, pgs. 55-69, original copyright notice is given to the publication in which the material was originally published: With kind permission from Springer Science and Business Media.

Figure 2. The Stryker® Intra-Compartmental Pressure (STIC) Monitor System

Figure 2. The Stryker Intra-Compartmental Pressure (STIC) Monitor System: With kind permission from Stryker Instruments, Kalamazoo, MI.

Table 1Common Factors Contributing to Lower Leg ACS ^{1-3, 6, 8, 9, 14, 15}

Orthopaedic
Tibial Fracture
Blunt or Penetrating Trauma
Total knee arthroplasty
Vascular
Reperfusion therapy
Arterial puncture or injury
Hemorrhage
Deep Vein Thrombosis
Soft Tissue
Crush injury
Contusion
Burn
Envenomation (Snake Bite)
Iatrogenic
Drugs (Anticoagulants)
Bleeding Disorders (Sickle cell)
Casts or Splints
Constrictive Dressings
Extravasation of drugs and fluids
Prolonged lithotomy positioning
Viral myositis
Diabetic muscle infarction

Table 2

Key structures within the lower leg compartments and alterations seen with injury 2, 13

Compartment	Muscle	Artery/Vein	Nerve	Alterations with injury
Anterior	Extensor muscles: • Tibialis anterior • Extensor hallucis longus (EHL) • Extensor digitorum longus (EDL)	Anterior tibial	Deep peroneal	↓/(-) Sensation of great toe web space ↓(-) Foot dorsiflexion and inversion ↓(-) Lateral four toes dorsiflexion ↓(-) Great toe dorsiflexion (EHL test) (-) Dorsalis pedis pulse
Lateral	• Fibularis longus • Fibularis brevis	Peroneal	Superficial peroneal	↓(-) Sensation anterolateral shin/dorsum foot ↓(-) Foot plantar flexion ↓(-) Foot eversion
Superficial posterior	Superficial flexor: • Soleus • Gastrocnemius • Plantaris	Posterior tibial	Tibial	↓(-) Foot plantar flexion
Deep posterior	Deep flexor: • Tibialis posterior • Fibularis hallucis longus (FHL) • Fibularis digitorum longus (FDL)	Posterior tibial and Peroneal	Tibial	↓(-) Sensation plantar aspect of the foot ↓(-) Great toe plantar flexion ↓(-) Lateral four toes plantar flexion ↓(-) Foot inversion (-) Posterior tibial pulse

Alterations with injury: ↓ depicts decrease sensation or motor function in early ACS or (-) depicts absence of sensation, motor function or pulses in late ACS.