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Compartment syndrome in extremities

Síndrome compartimental en extremidades

Aldo Israel Olán-De Los Santos,[*] Hilda Alejandra Manzo-Castrejón,[‡] Grecia Fernanda Hurtado-Miranda,[*] Jerath Antonio Nájera-Reyes,[§] Juan Daniel Arreola-Pérez,[¶] Iván San Pedro-Rodríguez[¶]

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Palabras clave: *síndrome, compartimental, extremidades, fasciotomía, traumatismo, quemadura.*

ABSTRACT

An analysis of the manuscripts published in the scientific literature on compartment syndrome was carried out to evaluate a patient with compartment syndrome and identify the clinical presentation and the bases necessary to establish the diagnosis and timely treatment. There is little recent literature less than five years old describing the characteristics of compartment syndrome, which can be caused by multiple etiologies, of which trauma is the most frequent, in addition to burns, fractures, contusions, coagulopathies, postischemic edema or very tight casts. A review of selected studies was carried out, among which those with the highest academic content were selected. Each selected study was analyzed, and data that could be included in each subtopic of the review article were identified. Acute compartment syndrome corresponds to a surgical emergency in that, in the case of not establishing an early intervention, the probability of irreversible sequelae increases notably. In compartment syndrome, there is an increase of interstitial pressure in the body compartments that compromises the adequate perfusion of the soft tissues, derived from an ischemic process that presents as clinical manifestations of sudden intense pain, pallor, absence of pulses, paresthesia, and paralysis, which accompanied by a directed anamnesis integrates the diagnosis with the physical examination. The decompression of the affected muscle space is a surgical emergency, requiring timely detection and early treatment to avoid sequelae and serious complications.

RESUMEN

Se llevó a cabo un análisis de los manuscritos publicados en la literatura científica sobre el síndrome compartimental para la evaluación de un paciente con síndrome compartimental e identificación de la presentación clínica y las bases necesarias para instaurar el diagnóstico y tratamiento oportuno. Es escasa la bibliografía reciente menor de cinco años que describa las características del síndrome compartimental, el cual puede ser provocado por múltiples etiologías, de las cuales los traumatismos son la más frecuente, además de las quemaduras, fracturas, contusiones, coagulopatías, edema postisquémico o yesos muy apretados. Se realizó una revisión de estudios elegidos, entre los cuales fueron seleccionados aquellos con mayor contenido académico. Se analizaron cada uno de los estudios seleccionados y se identificaron los datos que podrían incluirse en cada uno de los subtemas del artículo de revisión. El síndrome compartimental agudo corresponde a una emergencia quirúrgica que, en caso de no establecer una intervención precoz, la probabilidad de secuelas irreversibles se incrementa notablemente. En el síndrome compartimental existe aumento de la presión intersticial en los compartimentos del cuerpo que compromete la adecuada perfusión de los tejidos blandos, derivado de un proceso isquémico que presenta como manifestaciones clínicas dolor súbito intenso, palidez, ausencia de pulsos, parestesia y parálisis, lo cual acompañado de una anamnesis dirigida integra el diagnóstico junto al examen físico. Corresponde a una urgencia quirúrgica la descompresión del espacio muscular afectado, por lo que requiere detección oportuna y tratamiento precoz para evitar secuelas y complicaciones graves.

[*] Second-year resident in general surgery.
[‡] Physician assigned to plastic and reconstructive surgery.
[§] Third-year resident in general surgery.
[¶] First-year resident.

Hospital General Tacuba, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado. Mexico.

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INTRODUCTION

The compartment syndrome (CS) is defined as a surgical emergency characterized by an elevation of interstitial pressure within a closed osteomyofascial compartment above capillary perfusion pressure with compromised blood flow in muscle and nerve that conditions tissue damage triggering an ischemic process that jeopardizes viability and reversibility.[1-3] In acute compartment syndrome (ACS), intra-compartmental pressure (ICP) > 30 mmHg is described as a valuable threshold to aid in diagnosis; however, it is considered a clinical diagnosis, so a single regular ICP reading does not exclude acute compartment syndrome.[3]

Historically, one of the first descriptions of CS and its complications in the literature was attributed to skeletal muscle ischemia, made by Richard von Volkmann, a 19th-century surgeon and considered one of the fathers of orthopedic surgery in 1881, of ischemic contracture of muscle tissue due to constriction, often by bandages.[4] Subsequently, surgical management by fasciotomy for battlefield injuries was put into practice as early as World War II; however, it is Norman Rich who is credited with emphasizing the liberal and timely use of fasciotomy as a treatment for post-trauma CS.[5]

When excessively increased pressure within a muscle compartment limits adequate capillary perfusion to soft tissues, nerves, and muscles, it compromises viability and leads to potential damage 8 hours after the onset of ischemia; such tissue damage secondary to prolonged ischemia will be irreversible.[6]

The human body has approximately 46 spaces containing muscle, nerves, and blood vessels. Of these, nine are located in the trunk and the remaining 37 in the extremities in confined spaces with nerves and vessels that innervate and irrigate the muscle tissue covered by a membrane of aponeurotic connective tissue called fascia, with the function of insulation, support, and protection.[7,8]

Pathophysiologically, several theories have been proposed where the common feature of all CS, regardless of etiology or anatomical location, is an increase in intra-compartmental pressure (ICP) within a rigid fascial envelope that alters tissue perfusion,[1] since at the capillary level, the external pressure at all points of the compartment decreases blood flow with an elevation of postcapillary venular pressure and increases hydrostatic pressure with fluid extravasation, edema, nervous and muscular ischemia, leading to necrosis of the structures involved.[9]

One way of classifying CS includes acute compartment syndrome: symptoms compatible with increased compartment pressure that does not resolve independently. Subacute compartment syndrome: does not present with the characteristic symptoms of acute compartment syndrome but still leads to the typical sequelae (ischemic Volkmann's contracture). Recurrent or chronic compartment syndrome: occurs in athletes during physical activity, with muscle weakness and pain where the patient is intermittently asymptomatic, without long-term alterations, and the intra-compartmental pressure at rest is usually elevated.[10,11] During exercise, there is deficient tissue oxygenation secondary to decreased venous return and insufficient muscle tissue perfusion, with increased compartment pressure, accompanied by pain and temporary neurological deficit.[12,13]

The fourth type, crush syndrome, is described as an extreme entity of the acute syndrome, with systemic manifestations of muscle injury.[10] Crush syndrome is defined as the systemic alteration resulting from post-traumatic rhabdomyolysis due to crushing forces; it is an alteration that is frequently reported after catastrophic earthquakes worldwide, and it is due to trauma in extremities by direct force or by the highly stressful position maintained for the circulation of an extremity. Its importance lies in the fact that there is a rupture of muscle cells, which releases myoglobin and potassium into the circulation, causing hyperkalemia, severe inflammatory

response, fluid loss to the third space, metabolic acidosis, acute renal failure, and shock.[14]

EPIDEMIOLOGY

Acute compartment syndrome (ACS) most commonly develops soon after significant trauma, mainly when long bone fractures occur; however, it can be seen from non-traumatic causes.[6,15] It may be due to intrinsic factors (e.g., swelling, hemorrhage) or extrinsic or post-injury factors that restrict the ability of the fascial envelope to expand. It is more common in the lower extremity than in the upper extremity. The calf is the most common site affected in the lower extremity, and the forearm is the most common site in the upper extremity. Fasciotomies are less frequently needed in the upper extremities, accounting for approximately 20% of all extremity fasciotomies.[16,17] ACS is most commonly seen in young men < 35 years with the highest incidence, particularly after tibial diaphysis and distal radius fractures.[6,18]

ETIOLOGY

Several studies showed that fractures are the most common cause of ACS, accounting for about 69-75% of cases.[19] Multiple other etiologies can cause ACS; among all, trauma is the most frequent, particularly long bone fractures, where the tibia is most frequently affected, and approximately 1-10% of fractures develop ACS. Fractures of the forearm are the second most affected bones in the upper extremities. Other traumatic causes of CS include penetrating extremity trauma with vascular (arterial, venous) injury, intra-compartmental hemorrhage, contusions, and crush or burn injuries. Non-traumatic causes include prolonged external compression, animal bites and stings, coagulopathies, tight casts, and postischemic edema. All these causes are presented below *(Tables 1 to 3)*.[2,20,21]

Other important traumatic etiologies to describe are crushed and burn injuries. Crush injuries occur when the limb is trapped for a prolonged duration with

Table 1: Etiology of compartment syndrome due to upper or lower extremity involvement.

Causes in upper and lower extremities
Long bone fracture
Acute extremity ischemia with reperfusion
Burn injury
Crush injury
Animal bites and stings
Spontaneous hemorrhage/hematoma
Soft tissue infection
Non-traumatic myositis/myonecrosis/rhabdomyolysis
Systemic inflammatory response syndrome/mass fluid resuscitation
Leading causes in upper extremities
Animal or insect bites and stings
Iatrogenic high-pressure injection (liquid or gas)
Intravenous extravasation injury
Accidental/intentional intra-arterial injection
Birth injury (e.g., neonatal compartment syndrome)
Leading causes in lower extremities
Prolonged immobilization
Snakebite (due to the location of most snakebites)

Table 2: Etiology of compartment syndrome due to extrinsic and intrinsic causes.

Intrinsic causes	Extrinsic causes
They increase the volume inside the compartment	Restrict dilatation of the compartmental envelope
Bleeding:	Tight bandages, splints, or circumferential casts
Trauma (fracture, vascular injury)	Burn eschar
Coagulopathies (hemophilia, post-resuscitation coagulopathy)	Surgical closure of facial defects
Anticoagulant therapy (heparin, coumarins, thrombolytic agents)	
Edema:	Prolonged use of a tourniquet
Ischemia/reperfusion (arterial lesions, embolism, and thrombosis)	Lithotomy position during surgery
Electrical injuries	Military anti-shock pants (MAST)
Venous thrombosis (phlegmasia cerulea *dolens*)	
Exercise or rhabdomyolysis	

Table 3: Traumatic and non-traumatic causes of acute compartment syndrome.

Traumatic causes	Non-traumatic causes
Fractures of long bones	Intravenous fluids: fluid extravasation; massive fluid resuscitation (e.g., severe thermal burns, sepsis, etc.)
Blunt trauma (crush injury/crush syndrome)	Hematologic: ischemia-reperfusion injury, thrombosis, bleeding disorders, vascular disease, spontaneous hemorrhage
Burn injuries	Anticoagulation
Constrictive bandages, splints, or casts	Toxics: animal poisonings and bites; recreational drug injections
Penetrating trauma	Prolonged limb compression (following severe drug or alcohol intoxication; malposition during surgery)
High-pressure injection	Revascularization procedures (limb bypass surgery, embolectomy, thrombolysis)
Injury to vascular structures	Nephrotic syndrome (decreased serum osmolarity)
	Group A streptococcus infections of the muscle; systemic inflammatory response syndrome

increased compartment pressure resulting from external compression that induces CS determined by the state and duration of the crush or, after blunting, direct trauma. Burns can cause ACS by several mechanisms, mainly circumferential full-thickness burns, as the burn eschar may restrict tissue swelling. Other mechanisms include tissue edema (especially thermal and electrical burns) and secretion of chemical mediators that cause systemic inflammatory response syndrome (SIRS).

Any condition that decreases the capacity of a compartment or increases the volume of fluid within a compartment increases intra-compartmental pressure and puts the patient at risk for developing compartment syndrome.[21] Common sites include the leg and forearm;[15,21] however, it can also occur in the foot, thigh, and gluteal region.

ANATOMY OF THE COMPARTMENTS

The vital thing to know about the compartments in compartment syndrome is their relationship to the clinic.

At the thigh level, there are three compartments:

- **Anterior:** the sartorius and quadriceps muscle is found, passing the femoral nerve and the superficial femoral artery, which provide irrigation to this area.
- **Medial:** composed of the pectineus muscle, obturator externus, gracilis, and abductor muscles. Through here runs the obturator nerve that innervates this compartment.
- **Posterior:** in the posterior compartment are the biceps femoris, semimembranosus, and semitendinosus muscles; the nerve structure found here is the sciatic nerve, and the deep femoral artery is also found; all of them provide both innervation and irrigation to the compartment.

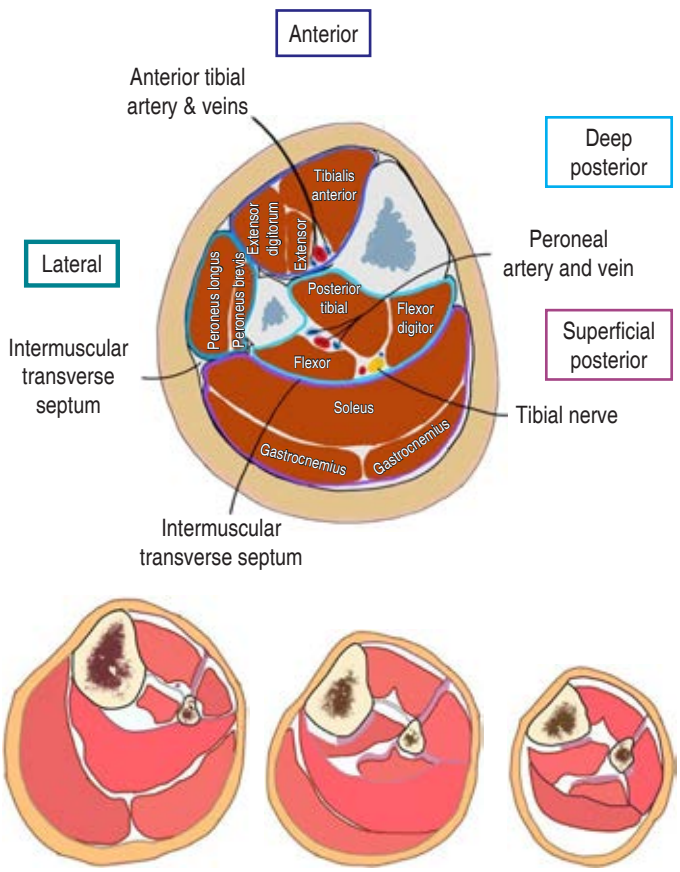

Figure 1: *Leg compartment.*

At the level of the leg *(Figure 1)*, a compartment is added and divided into anterior, lateral, superficial posterior, and deep posterior, which are formed as follows:

- **Anterior:** tibialis anterior muscle, extensor muscles of the foot, peroneal muscle. The anterior tibial artery and the deep perineal nerve are part of its irrigation and innervation.
- **Lateral:** the peroneus longus and peroneus lateralis brevis muscles are found here, the superficial perineal nerve runs through here, and the branches of the anterior tibial artery are found, which provide irrigation and innervation to these muscles.
- **Posterior superficial:** the gastrocnemius, soleus, and plantaris muscles are found, the tibial nerve provides innervation to these muscles, the sural arteries (medial and lateral) are also found, which provide irrigation to the gastrocnemius, while the popliteal, posterior tibial and peroneal arteries irrigate the soleus muscle.
- **Deep posterior:** the posterior tibial muscle, the foot flexor muscles, and the popliteal muscles are found. The tibial nerve and the posterior tibial and peroneal artery innervate it.

The foot *(Figure 2)* has medial plantar, central plantar, lateral plantar, interosseous, and dorsal compartments.

- **Medial plantar:** the abductor hallucis and flexor hallucis brevis are found, as well as the medial plantar nerves and vessels.
- **Central to the sole:** the short and long flexors of the toes are found, as well as Stan, the nerve, and plantar vessels.
- **Lateral to the sole:** the abductors and flexors of the little toe.
- **Dorsal:** which is limited by the dorsal fascia; it is the one that is more on the surface of the compartments.[21]

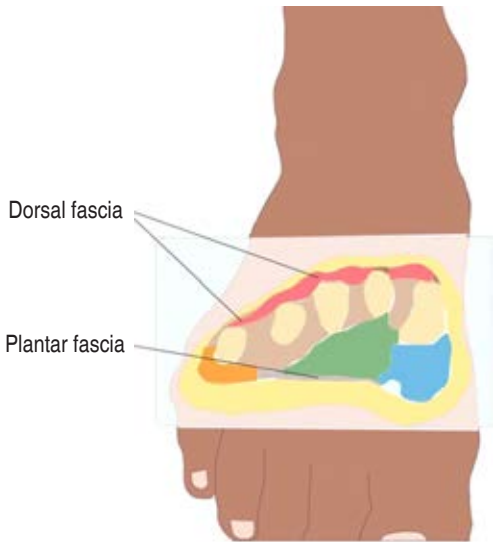

Figure 2: *Plantar and dorsal fasciae of the foot.*

At the level of the upper extremity *(Figures 3 to 5)*, specifically in the arm, there are anterior and posterior compartments.

- **Anterior:** contains the elbow flexor muscles (biceps brachii, brachialis) and the ulnar and median nerves.
- **Posterior:** contains the elbow extensor muscles (triceps) and the radial nerve.

PATHOPHYSIOLOGY

Multiple theories have been elaborated on the pathophysiology of compartment syndrome; however, they all conclude in the scenario of tissue anoxia.[20]

Ischemia begins once the local blood flow is restricted by increased compartment pressure, causing the metabolic demands of the local tissue to be unsatisfied.[21] Any etiology that causes increased fluid or pressure within a muscle compartment will elevate the internal pressure of the compartment due to the null distensibility capacity of the muscle fascia.[20]

The arteriovenous pressure gradient theory is the most widely accepted theory of acute compartment syndrome in extremities. Increased compartment pressure restricts local tissue perfusion by decreasing the arteriovenous pressure gradient, which consists of a decrease in arterial pressure and an increase in venous pressure. In turn, it will lead to a stage of cellular anoxia that will affect nervous and muscular tissue. A cycle is generated in which the effects of tissue hypoperfusion increase vascular permeability and, in turn, internal pressure.[20]

When intra-compartmental pressure rises within 10-30 mmHg of diastolic pressure, muscle oxygenation decreases as tissue pressure approaches mean arterial pressure, meaning that acute compartment syndrome of the extremities develops as a function of both compartmental and systemic arterial pressures.[20]

Nerve conduction is impaired when there is a difference between compartment pressure and a diastolic pressure of less than 30 mmHg or when compartment pressure is greater than 30 mmHg. If the pressure rises, conduction is wholly interrupted, and motor paralysis occurs-progression of ischemia results in cell death and myocytolysis. The severity of muscle damage is relatively proportional to the duration of ischemia of the affected limb;

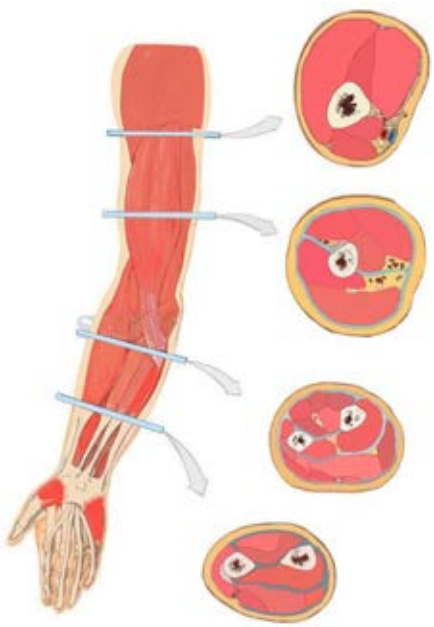

Figure 3: *Compartments of the upper extremity.*

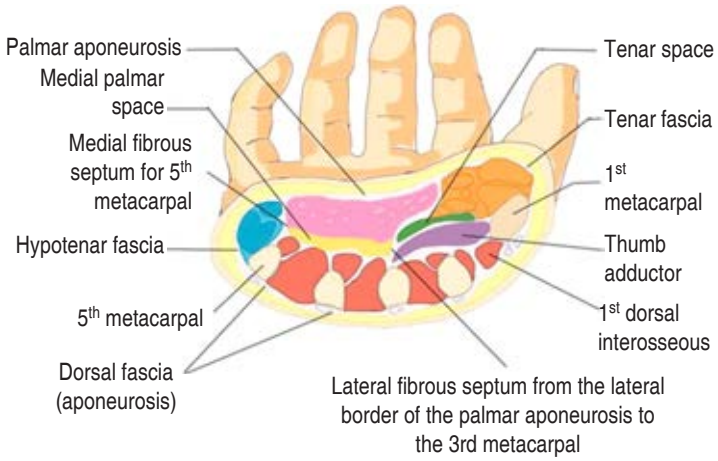

Figure 4: *Compartments at hand level.*

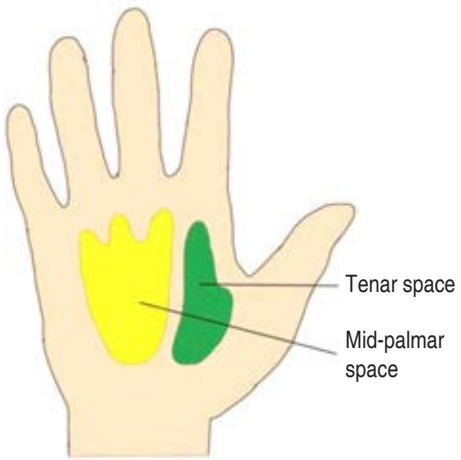

Figure 5: *Tenar and mid-palmar space.*

these effects will be irreversible after four to eight hours.[20]

CLINICAL PICTURE

Signs and symptoms of compartment syndrome (CS) include the "6 P's", of which pain out of proportion to the injury and increased analgesic requirements should raise suspicion of developing compartment syndrome,[22] other signs appear late and indicate delayed diagnosis, including pallor, paralysis, and absence of pulses.

1. **P**aresthesias: corresponds to the first indication of nervous ischemia and is the first symptom manifesting as a tingling sensation, burning, numbness, and loss of discrimination between two points.
2. **P**ain: intense and disproportionate to the lesion of stabbing or deep type, may be localized or diffuse, exacerbated by passive movement or direct compression of the affected compartment or elevation of the limb, and does not subside with narcotics.
3. **P**ressure: greater than 30-40 mmHg inside the compartment, measured by continuous infusion method or Stryker® (normal pressure 0-10 mmHg). The extremity is tense and warm on palpation, the skin taut and shiny.
4. **P**allor: late sign associated with progressive compromise of tissue perfusion, accompanied by cold and stiff skin with prolonged capillary filling (> 3 seconds).
5. **P**aralysis: late sign, weak or absent movement of distal joints, absence of response to direct neurological stimulation (damage of the myoneural junction).
6. The absence of pulses (Pulselessness) is a late sign. Clinically verified by palpation and absence of audible Doppler.[2]

Eliminating pain proportional to a muscle group's injury or passive stretching is considered the most important clinical finding; however, an assessment may be impaired due to altered consciousness, sedation, and mechanical ventilation.

Some general symptoms that can be found are abdominal pain or distension, dyspnea, dizziness, weakness, and malaise. Despite these clinical data, physical examination of the abdomen is a poor predictor of ACS.[23] Paralysis and paresthesias are less valuable data acutely, as they may result from neural trauma. Pulselessness is a late sign of CS but may be seen more frequently in combat wounds, firearm injury, arterial injury, or an expanding hematoma.

Other clinical data are tachycardia, hypotension, the elevation of jugular venous pressure, peripheral edema, and hypoperfusion data such as cold skin, obnubilation, restlessness, or lactic acidosis.[23]

DIAGNOSIS

The diagnosis of CS can be made clinically using the "6 P's"; however, there are more specific methods to determine the mmHg in a compartment to identify whether compartment syndrome is present. Signs and symptoms generally appear stepwise, although specific findings' appearance time varies.[15] When the diagnosis of ACS is suspected on clinical grounds, it is often confirmed by measuring compartment pressures. A careful and thorough neurological examination of the extremity should be performed and documented as part of the initial evaluation. A table describing signs and symptoms is provided *(Table 4)*.

MEASUREMENT OF COMPARTMENT PRESSURES

In remote areas and hospitals with limited surgical coverage, this approach is only sometimes possible but is preferable.

Direct measurement techniques

A hand-held manometer (e.g., Stryker device®), a simple needle manometer system, and the wick or slit catheter technique are the main approaches for direct measurements. The hand-held manometer method is most often used because it is portable, simple, and relatively accurate.[24] The slit catheter technique involves inserting a catheter into the compartment and monitoring the pressure through a transducer connected to a pressure amplifier and recorder.[25] The tip of the catheter or device should be within approximately 5 cm of the fracture level to obtain the maximum intra-compartmental pressure measurement.

Interpretation of measurements: normal tissue compartment pressure is between 0 and 8 mmHg.[26] Clinical findings associated with ACS generally correlate with the degree to which tissue pressure within the affected compartment approaches systemic arterial pressure: capillary blood flow is compromised when tissue pressure increases between 25 and 30 mmHg from mean arterial pressure. Pain may develop when tissue pressures reach 20 to 30 mmHg. Ischemia occurs when tissue pressures approach diastolic pressure.[27,28]

Indirect measurement techniques

There is a difference between diastolic blood pressure and compartment pressure (delta pressure) of 30 mmHg or less as a threshold for diagnosing ACS.[29,30] The delta pressure is found by subtracting the compartment pressure from the diastolic pressure, i.e.:

1. ACS delta pressure = diastolic blood pressure – measured compartmental pressure.

Table 4: Signs and symptoms.

Pain disproportionate to the apparent lesion (early and common finding)
Persistent deep aching or burning pain
Paresthesias (onset within approximately 30 minutes to 2 hours in acute compartment syndrome; suggests ischemic nerve dysfunction)
Pain with passive stretching of the muscles in the affected compartment (early finding)
Tight compartment with a firm "wooden" feel
Pallor due to vascular insufficiency (rare)
Decreased sensitivity
Muscle weakness (onset within approximately 2 to 4 hours in acute compartment syndrome)
Paralysis (late finding)

Modified from: Hammerberg EM.[21]

2. ACS delta pressure < 20 to 30 mmHg indicates the need for fasciotomy.[29,30]

If left untreated, ACS can lead to muscle necrosis, sensory deficits, paralysis, infection, fracture nonunion, and limb amputation.[6] Rhabdomyolysis may occur with muscle ischemia, resulting in myoglobinuria and possible renal failure requiring dialysis.

Treatment

In most cases, decompressive fasciotomy is the definitive treatment for acute compartment syndrome, where immediate open fascial decompression of all affected compartments is an emergency because delaying fasciotomy increases morbidity, including the need for amputation. Patients with late presentation or diagnosis (more than 12 hours) are at high risk for complications with surgery. Decision-making is complex and should involve two consultants. Upon clinical suspicion of compartment syndrome, immediate action should be taken by relieving external pressure on the compartment, which includes removing any bandages, splints, casts, or other restrictive coverings to examine the limb.[31,32]

Following fasciotomy, patients should undergo re-exploration at approximately 48 hours, or sooner if indicated, with the early involvement of a plastic surgeon to achieve adequate soft tissue coverage.[32]

Placement of the limb at the level of the heart is recommended; this helps to avoid reductions in arterial flow and elevations in compartment pressures due to swelling.

Indications and contraindications for fasciotomies are described below.[33]

Indications

Fasciotomy is indicated in clinical evidence of acute compartment syndrome (impending or established), which in some cases may be supported by objective evidence of elevated compartment pressure.

In impending compartment syndrome, symptoms may not be classic, and compartment pressure may not meet the criteria for acute compartment syndrome. However, there is a high likelihood of progression to compartment syndrome. Fasciotomy is performed earlier in this population based on the clinical judgment that the compartment is highly likely to develop. Fasciotomy should be performed immediately after worsening symptoms among those for whom careful management has been chosen.

Contraindications

Sometimes fasciotomy is not indicated or may not be necessary; for example, it should be avoided when the muscle is already dead. Fasciotomy, in such cases, provides no benefit and may increase the risk of infection. The definitive treatment for this type of injury often involves amputation.

A fasciotomy may not be necessary for specific low-risk circumstances, but the treating surgeon should make this decision.

CONCLUSION

In compartment syndrome, there is an increase of interstitial pressure in the body compartments that compromises the adequate perfusion of soft tissues, derived from an ischemic process that presents as clinical manifestations of sudden intense pain, pallor, absence of pulses, paresthesia, and paralysis. Multiple etiologies can cause an ACS, such as trauma, fractures, contusions, coagulopathies, burns, postischemic edema, or very tight casts, integrating the diagnosis with the physical examination.

The decompression of the affected muscle space is a surgical emergency, requiring timely detection and early treatment to avoid sequelae and serious complications.

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Correspondence:
Aldo Israel Olán-De Los Santos
E-mail: aldoi27@hotmail.com