

Current Perspectives on Compartment Syndrome: A Comprehensive Review

Perspectivas actuales sobre el síndrome compartimental: una revisión integral

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Summary

Introduction: Compartment syndrome is a pathology secondary to increased tissue pressure within an osteofascial compartment. **Objective:** To review the recent literature on the diagnosis and treatment of compartment syndrome to provide an updated view of this condition. **Methods:** A search of the Web of Science and PubMed databases was performed, using literature from 2019 to 2024. The search terms used were: compartment syndrome, diagnosis, treatment, extremities. A total of 179 articles were found. The selection criteria were: original articles, with available references, from refereed publications, less than five years old, updated articles on prevention, diagnosis and treatment, that had a relevant contribution to this research. After submitting these texts to the selection criteria, 28 articles were obtained. **Results:** Within its physiopathology, arteriolar vasodilation and collapse of the venule system play an essential role, which conditions an increase in vascular permeability, ending in the extravasation of liquid to the interstitial space, causing edema and a decrease in the rate of tissue perfusion. Its diagnosis is mainly clinical, by means of a clinical history and an adequate physical examination. The definitive treatment, once this pathology is established, is fasciotomy. **Conclusions:** Mortality in a patient with compartment syndrome can reach 47%, so a timely and efficient diagnosis is essential.

Keywords: Compartment Syndrome; Diagnosis; Treatment; Extremities.

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Resumen

Introducción: el síndrome compartimental es una patología secundaria al aumento de la presión dentro de un compartimento osteofascial. **Objetivo:** revisar la literatura reciente sobre el diagnóstico y tratamiento del síndrome compartimental para ofrecer una visión actualizada de esta condición. **Método:** se realizó una búsqueda en las bases de datos de *Web of Science* y PubMed, se consultó bibliografía del año 2019 al 2024. Se utilizaron los términos de búsqueda: síndrome compartimental, diagnóstico, tratamiento y extremidades. En un primer análisis se identificó un total de 179 artículos. Los criterios de selección fueron: artículos originales, con referencias disponibles, de publicaciones arbitradas, con menos de cinco años de antigüedad, artículos actualizados en la prevención, diagnóstico y tratamiento, que tuvieran un aporte relevante a esta investigación. Tras someter estos textos a los criterios de selección, se obtuvieron veintiocho artículos. **Resultados:** dentro de su fisiopatología juega un papel imprescindible la vasodilatación arteriolar y el colapso del sistema de vénulas, lo que condiciona un aumento en la permeabilidad vascular, finalizando en la extravasación de líquido al espacio intersticial, propiciando edema y una disminución de tasa de perfusión tisular. Su diagnóstico es principalmente clínico, mediante historia clínica y exploración física. El tratamiento definitivo, una vez instaurada esta patología, es la fasciotomía. **Conclusiones:** la mortalidad en un paciente con síndrome compartimental puede alcanzar 47%, por lo que es imprescindible un diagnóstico oportuno y eficiente.

Palabras clave: síndrome compartimental, diagnóstico, tratamiento, extremidades.

Introduction

Compartment syndrome is a clinical entity secondary to increased tissue pressure within an osteofascial continent, where there is limited room for expansion, affecting local circulation and potentially leading to ischemia and necrosis if not addressed early.¹ Although it is usually diagnosed clinically, intracompartmental pressure (ICP) greater than 30 mmHg is used as the cutoff point for being considered an abnormal value.²

It was first described in 1881 by Richard von Volkmann, and reproduced in animals in 1924 by surgeon Paul Jepson. In addition, compartment decompression was integrated as a potential management option and fasciotomy was finally incorporated in World War II.¹

The economic cost of compartment syndrome is generated by increased hospital days, averaging three to six days, with costs in the United States averaging \$23,800.00 to \$34,000.00 USD and increasing if fasciotomy wound management is required or secondary to an exposed bill.³

The critical time in which nerve and muscle tissue suffers irreversible damage is between six and twelve hours. During this period, effective management by the surgical team is critical to prevent complications resulting from failure to intervene.^{1,2}

Methods

During data collection, 179 articles available in the PubMed and Web of Science databases, obtained between January and March 2024, were identified. The search terms used were compartment syndrome, diagnosis, treatment, extremities with the Boolean operators “and” and “or”. The selection criteria were original articles, published in indexed journals,

with available references, from refereed publications, published during the period from 2019 to 2024, addressing etiology, pathophysiology, diagnosis and treatment. The decision to exclude papers that did not meet these requirements was reached by consensus of the authors. At the end of this process, 28 articles were obtained (Figure 1).

Results

Epidemiology

Compartment syndrome has an incidence of 3.1 cases per 100,000 population, 7.3 per 100,000 men and 0.7 per 100,000 women. The group of patients under 35 years of age was the most frequent. This incidence is due to the higher level of muscle mass in men than in women, and to the risk of presenting high impact injuries in this group.⁴

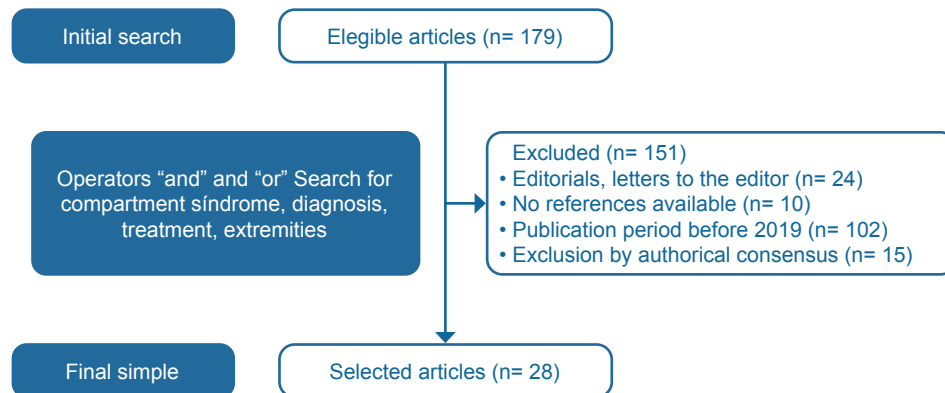
In Mexico there is no epidemiological information available on this condition, so estimates based on the approximately 6.8 million Americans treated annually for fractures are used.⁵

Pathophysiology

This pathology is caused by an elevation of tissue pressure within the affected muscle compartment.⁵ These conditions the decrease in capillary flow and the potential progression to localized necrosis due to hypoxia.⁶

Normal intracompartmental pressure is known to average around 10 mmHg. At the time of high-energy trauma, arteriolar vasodilatation and collapse of the venule system occur, which increase vascular permeability and fluid extravasation to the interstitial space. The resulting alteration of the arteriovenous gradient leads to anaerobic metabolism due to lack of perfusion.⁷

Figure 1. Algorithm for selection of articles by inclusion criteria



This inability to meet the requirements of cellular metabolism results in a significant decrease in ATP, dysfunction of the Na⁺/K⁺ ATPase pump (intracellular osmotic imbalance), elevation of sodium and calcium in the cell cytoplasm, activation of calcium-activated protein kinases, oxidative stress and myocyte necrosis. Loss of regulation increases the content of intermediary products of metabolism (potassium, myoglobin, nucleosides) that will be released into the circulation. These molecules may favor systemic complications such as acute renal failure or cardiac arrhythmias.^{5,8}

It has been described that after eight hours of ischemia irreversible damage

occurs at the muscle tissue level. At the neurological level, neuropraxia occurs at four hours and axonotmesis at eight hours. These changes have been demonstrated with intracompartmental pressure of 40-80 mmHg.⁶

Etiology

Three out of four cases of compartment syndrome are secondary to a long bone fracture, with this value increasing with the presence of other coexisting fractures.⁹ The tibial fracture is the cause of the syndrome in 10 to 36% of all cases, followed by the radius and ulna, in almost 9% of cases. In pediatric age, supracondylar fractures are a major cause.⁶

After fractures, soft tissue damage is the second leading cause of this syndrome, followed by burns, vascular damage, crush injuries, improperly placed bandaging equipment, penetrating trauma, animal bites and bites or strenuous physical activity.¹⁰ Some non-traumatic causes of the pathology include hemophilia and thrombosis, poisoning, infections, extravasation of intravenous solutions, anticoagulation and nephrotic syndrome, in addition to other pathologies that decrease serum osmolality.¹¹ The classification into acute and chronic compartment syndrome is broken down in the following table.

Risk factors

The predisposing factors for this entity are those that favor or perpetuate the proinflammatory state, and are subdivided according to Table 2.¹²⁻¹⁴

Clinical Presentation

Symptomatology of the condition is of rapid onset and progressive escalation of symptoms.¹ Due to the possibility of omission of pathology in an initial physical examination and attention to the underlying cause, any tense and painful muscle compartment should be considered a possible compartment syndrome and should be confirmed with intracompartmental pressure measurement.¹⁵

An early and classic symptom is burning pain disproportionate to the traumatic injury or apparent pathology, progressing from thirty minutes after the injury to the presence of paresthesias.¹⁶ Other symptoms such as muscle weakness, compartment tension described as “wooden firm” and pain on passive muscle stretching, require constant monitoring.⁵

There is uncommon symptomatology, such as the presence of the five “P’s” of arterial insufficiency (pain, pallor, pulselessness, paresthesias, poikilothermia). The complications of the syndrome give rise to specific symptoms for each one, such as the presence of choluria in the case of rhabdomyolysis.¹⁷

Diagnosis

The diagnosis is clinical, based on questioning and physical examination of the affected region.¹⁸ Intense pain, which does not subside with anesthetic management and is exacerbated by passive stretching of the fingers, is a cardinal point of suspicion.¹⁹

Paresthesia is the second presenting

Table 1. Differences between acute and chronic compartment syndrome^{1,4-7}

Criteria	Acute	Chronic
Onset	Sudden, usually after a serious injury.	It develops gradually, commonly after intense physical activities.
Causes	Acute traumatic events such as fractures, injuries, orthopedic surgery, or bleeding.	Repetitive movements, common in sportsmen and high-performance athletes.
Management urgency	Medical emergency, immediate attention is required.	It is not usually an emergency, but it can be extremely painful.
Reversibility	Without surgical treatment, adequate management cannot be performed.	It can be managed by resting the affected limb.

Table 2. Risk factors for the development of compartment syndrome⁵

Group	Examples
Orthopedics	Diaphyseal fractures (tibia, radius or femur).
Vascular	Revascularizations, deep vein disease, hemophilia (ability to cause intramuscular hematomas).
Soft Tissue	Crushing, burns, viral myositis, hemotoxin (rattlesnake bite).
Iatrogenic	Use of tourniquet, compressive bandage or tight casts.
Others	Obesity, arterial hypertension, hyperlipidemia, diabetes mellitus and sustained Trendelenburg position.

symptom in compartment syndrome, mainly referred to as numbness and loss of sensation (neuronal ischemia).¹¹ Symptoms such as paralysis, pallor or absence of pulses are late findings that imply established neurovascular damage.¹⁷

The diagnostic gold standard is the measurement of intracompartmental pressure (Figure 2).^{1,20} Measurements of 35 mmHg are considered as part of the diagnosis of this syndrome.¹⁷ In patients with hypotension a result of 20 mmHg is symptomatic.

To perform this procedure, a transducer connected to a catheter should be

introduced at 5 cm from the lesion.⁵ In addition, radiographs in anteroposterior and lateral projections of the affected area are indicated to rule out the presence of fractures.²⁰

Treatment

Early management of the pathology avoids the presence of complications, so compartment pressure reduction should be performed immediately. Removal of constrictive material (and maintaining elevation of the affected limb to the level of the heart helps to improve arterial flow.²¹ Perfusion of muscle tissue should

be ensured with the use of supplemental oxygen and saline boluses should be adjusted to the patient's needs.²²

The definitive treatment for this pathology is fasciotomy, supplemented with prophylactic antibiotics adjusted according to the presence or absence of an exposed fracture. Incisions, 12 to 20 cm in the lower limbs, should be wide, anatomically guided and made as small as possible. Closure should be carried out progressively, according to the patient's evolution.^{23,24}

It is important to emphasize the refusal to perform preventive or late fasciotomies (in the presence of necrotic muscle), given the infectious complications that tissue exposure can generate.²⁴

Other therapies such as the use of hyperbaric oxygen are under evaluation for the management of this pathology.^{21,22}

Prognosis and Prevention

This clinical entity has a mortality of 47% if not treated in a timely manner, being the time of evolution and the timely performance of surgical treatment the most relevant prognostic factors for the development of complications.^{5,25}

Mortality has been reported to be 37% in late fasciotomies (more than six hours) and 25% in early fasciotomies (less than six hours).²⁶ The need for amputation is reported in 5 to 21% of cases, with the subsequent increase in morbidity and mortality that these procedures entail.²¹

To prevent this pathology, compressive casts/bandages should be identified and removed, as well as the use of ice and elevation of the affected extremity.²⁶ Similarly, in patients with risk factors for developing this entity, adequate oxygen saturation and normotension (improvement of perfusion pressure) should be

avored.²³ Studies have shown the benefit of mannitol in reducing free radicals and the subsequent formation of edema in patients with risk factors for developing compartment syndrome.²¹

Complications

In case of delayed management, muscle necrosis, chronic sensory pathologies, paralysis, infections, osteosynthesis failure, limb amputation, rhabdomyolysis, renal failure and death are possible.^{27,28}

Discussion and Conclusions

Compartment syndrome occurs in 3.1 per 100,000 people, commonly as a sequelae of tight cast/bandage appliances, muscle contusions, vascular injuries, coagulopathies, burns, tourniquets or, in most cases, trauma (whether or not associated with fracture).^{4,5}

Interstitial pressure (myofascial space) is the key point that leads to sustained ischemia, manifested by pain, pallor, decreased pulses, paresthesias, paralysis, etc.; which will end in necrosis of the cellular components of the affected compartment.⁶

It is emphasized that although tools are available to confirm clinical suspicion of this pathology (analysis of intracompartmental pressure), it should not be forgotten that the diagnosis is mainly clinical.^{1,20}

Compartment syndrome is considered a surgical emergency (due to the narrow time interval for an effective treatment), so once the diagnosis is made, it should be approached with fasciotomies, emphasizing that the ideal moment for this decompressive treatment is from the moment the patient presents the first symptoms (remembering that paralysis and paresthesias are late manifestations).^{21,24,28}

Patients with risk factors and a high index of suspicion for developing compartment syndrome should be under continuous monitoring, with the removal of plaster/compressive bandages and elevation of the affected limb in a preventive way.^{6,14}

Authors' contribution

J P-P: conceptualization, development and writing.

J L-R: development, writing and revision.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

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