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UPDATE

Acute compartment syndrome of the foot☆

A. Dalmau-Coll,* R. Franco-Gómez, D. Codina-Granó, J. Vega-García

Departamento de Cirugía Ortopédica y Traumatología, Unidad de Pie y Tobillo, Hospital ASEPEYO, Sant Cugat del Vallés, Barcelona, Spain

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Abstract Acute and post-traumatic compartment syndrome of the foot is typically associated with mechanisms of high-energy, multiple foot fractures, particularly those caused by crushing or trapping. Early diagnosis and treatment in view of the suspicion of an acute compartment syndrome in a traumatic foot will help prevent the effects of myoneural necrosis. Even though there are various methods of compartment pressure monitoring, the diagnosis is mainly clinical. Prophylaxis is essential and the best treatment is to prevent the development of this syndrome. To achieve this a correct clinical history and initial examination is needed, with special care in the nerve, vascular and muscle evaluation. Surgical decompression using fasciotomy must be urgent and will be mandatory to prevent the severe sequelae. Delayed treatment can have disastrous consequences, such as contracture and paralysis, infection, and sometimes amputation.

PALABRAS CLAVE
Síndrome compartimental agudo;
Contractura isquémica;
Isquemia mioneural;
Fasciotomías pie

Síndrome compartimental agudo en el pie

Resumen El síndrome compartimental agudo (SCA) o postraumático en el pie se asocia típicamente a mecanismos de alta energía, pies polifracturados, aplastamientos o atrapamientos. El diagnóstico y tratamiento precoz ante la sospecha de un SCA en un pie traumático ayudará a prevenir las secuelas de la necrosis mioneural. Aunque existen diversos métodos de monitorización de la presión compartimental, su diagnóstico es fundamentalmente clínico. La profilaxis es fundamental y el mejor tratamiento es prevenir el desarrollo de este síndrome. Para ello, es necesario de una correcta historia clínica y exploración inicial, con especial interés en dejar reflejada la exploración neurológica, vascular y muscular. La descompresión quirúrgica, mediante fasciotomías, debe ser ur-

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* Corresponding author.
E-mail: antoniodalmau@aol.es (A. Dalmau-Coll).

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gente y será clave para evitar la instauración de graves secuelas. El retraso en el tratamiento puede tener consecuencias desastrosas, como la contractura, parálisis o infección, que en ocasiones pueden requerir la amputación.

Introduction

Richard von Volkman, in 1881, was the first to describe compartment syndrome with particular interest in the systemic repercussions and functional impact on the extremity. He described the contractures caused by constrictive bandages on the forearm and hand, which he believed to be ischemic in nature due to the prolonged obstruction of arterial blood flow. Later, in 1911, Bardenheuer described the aetiology of acute compartment syndrome (ACS), similar to the current one. It was not until 1940, however, in treatment of battlefield injuries during WWII, that fasciotomy came to have widespread clinical application.

In 1975, Matsen defined compartment syndrome as an increase of pressure in a closed osteofibrous space resulting in reduced blood flow and tissue perfusion, which causes ischemic pain and may damage tissues inside the compartment.[1,2]

Compartment syndrome may be acute or chronic.

Typically, the chronic or post-exercise syndrome is recurrent and associated with some repetitive physical activity or sports activity. It is unusual for this syndrome to occur in the foot.[3] Clinically, it is characterized by localized pain in the affected muscle compartment, which may be associated with neuritic pain or neurological deficit radiating across the territory of nerves passing through the compartment. These pathognomonic symptoms are triggered by exercise and subside shortly after the exercise is discontinued. Various theories may explain the pathophysiological process. One theory states that muscle hypertrophy in the compartment coupled with the transitory volume increase due to increased blood flow caused by the exercise would impede venous return, which would result in increased blood volume in the compartment and increased intracompartmental pressure. This intracompartmental pressure could continue rising to the point of exceeding the arterial perfusion pressure, which would reduce oxygen supply to the tissues and make them hypoxic. When the sports activity is discontinued, the blood volume in the muscle and, in turn, the intracompartmental pressure would drop, thus reversing the aetiological process.

ACS or post-traumatic syndrome in the foot is usually associated with high-energy, multiple foot fracture mechanisms, in particular when the foot has been crushed or trapped.[4]

The pathogenesis and treatment of acute compartment syndromes in the extremities are well known and, as a result of the duration and magnitude of the interstitial pressure increase, tissue necrosis will occur, with impairment of myoneural function.

Anatomy of the foot compartments

Classically, 4 compartments containing muscle mass have been differentiated: interosseous (intrinsic muscles between the 1st and 5th metatarsals and digital nerves); medial (abductor hallucis and flexor hallucis brevis); central (flexor digitorum brevis, quadratus plantae, and adductor hallucis); and lateral compartment (little toe flexor and little toe abductor). In an anatomical study with special reference to decompression of the calcaneal compartment, Manoli and Weber[4,5] identify 9 compartments in the foot. The medial and lateral are the same as described above; in the central, they differentiate a superficial compartment containing the flexor digitorum brevis and another deep compartment in the hindfoot—the calcaneal compartment—containing the quadratus plantae muscle, the lateral plantar nerve, the posterior tibial neurovascular bundle, and the tendon for the flexor hallucis longus. Finally, in the forefoot, there are 4 interosseous compartments and the adductor hallucis compartment.

Aetiology

There are various causes of an ACS, the most common being fractures (open or closed), crushing or trapping injuries, casts or bandages that are too tight, circumferential scars due to burns, and the like.

Mubarak et al[6] group the causes of ACS into two large categories:

1. Those that result in the compartment being diminished:
   a. Very constrictive bandages or casts
   b. Burns and frostbite resulting in inelastic circumferential scars
   c. Surgical closures under tension
   d. Crushing
2. Those that result in increased compartment content:
   a. Post-ischemia oedema: arterial lesion, arterial thromboembolism, limb re-implantation, excessive duration of ischemia, arterial catheterisation, ergotamine.
   b. Primary hematoma: haemophilia, anticoagulant therapy
   c. Intracompartmental bleeding: fractures, osteotomies
   d. Snake bite, venoms.

Compartment syndrome in the foot is common following metatarsal or Lisfranc fracture-dislocations and in calcaneus fractures,[7] being described in up to 10% of cases. Non-traumatic causes such as haemangioma have also been described.[8]

Pathophysiology of compartment syndrome

ACS is characterized by increased intracompartmental pressure that may stem from multiple causes, and this triggers various lesions (fig. 1).

The increased intracompartmental pressure causes a reduction in capillary pressure and perfusion with the resulting muscle and nerve ischemia. If the injury mechanism continues to act, the result will be muscle and nerve necrosis.

Nerve necrosis causes paraesthesias that will lead to a total anaesthesia and/or paresis that will progress to a paralysis.

Muscle necrosis causes a degeneration of muscle fibres; these are then replaced by inelastic fibrous tissue, and this will result in a contracture.

Besides the local effects on the limb, ACS may have a generalized impact. As a result of muscle ischemia, the damaged muscle cells may release myoglobin. During reperfusion, myoglobin will pass into the blood stream together with other inflammatory and toxic metabolites. The release of toxins secondary to cell destruction may trigger a multi-organ failure that is life-threatening to the patient. Whether or not systemic effects develop and their extent will depend on the severity and duration of compromised tissue perfusion and on the size and number of muscle compartments involved.

Symptoms

The primary symptom is pain appearing within a few hours of the injury. It is a severe pain, excessive or more than would be expected, that increases with passive extension of the affected muscles.[1,2,15]

The pain is accompanied by tautness in the area, swelling, and impairment of distal sensation.

The clinical manifestations are usually described as the 6 P's[9,10]:

Paraesthesias

This is the first symptom to appear; it is difficult to interpret, however, and may be due to muscle ischemia, nerve ischemia, an antalgic phenomenon, or a combination of these 3 factors. Without treatment, they will progress toward hypoesthesia or anaesthesia.

Pain

The pain is out of proportion to the type of injury, exacerbated by passive movement or direct compression of the affected compartment, and described as sharp or deep, localized or diffuse; it increases with elevation of the extremity and does not subside with analgesics. It can be an unreliable indicator in patients with established compartment syndrome or with concomitant central or peripheral neurological deficit, however, for pain may be absent in these cases.

Pressure

The compartment is indurated, being tense and warm, and the skin is taut and shiny. It is the earliest sign.

Pallor

This is a late sign; the skin is cool and stiff, with prolonged capillary filling (>3 seconds). It is rarely seen, occurring when arterial flow is severely reduced.

Paralysis

A late sign: movement of the distal joints is weak or absent, and there is no response to direct neurological stimulation (irreversible myoneural damage).

Pulselessness

Peripheral pulses are palpable unless there is an arterial lesion. Their absence is a late sign, verified clinically by palpation and by no audible pulse on Doppler.

It is extremely important to bear in mind that, when present, these 6 P's are signs and symptoms of an established picture, so there is no need to await their appearance to make the diagnosis.

Diagnosis

Diagnosing compartment syndrome is essentially a matter of bringing it to mind and doing a series of examinations. In the foot, there is clinical suspicion of the diagnosis when severe swelling follows an injury (fig. 2A). It is difficult to distinguish which compartment is affected, however, and each compartment must be meticulously evaluated for sensitivity.

It is very important to highlight that, in these patients, there is a history of fracture, trauma, recent orthopaedic surgery, or some other triggering cause of the clinical picture.

The diagnosis is based primarily on the symptoms and the physical examination. On occasion, however, the clinical picture may be ambiguous or the examination equivocal or difficult to perform. In these cases, the intracompartmental pressure must be measured.

There are different methods for measuring compartment pressure,[11,12] the most widely used being a pressure monitor connected to a catheter or the easy-to-handle and precise portable devices.

An intracompartmental pressure of less than 10 mm Hg is considered normal. Paraesthesias begin to appear at 20-30 mm Hg. If the pressure is less than 30 mm Hg, the patient should be under continuous monitoring or serial examinations over the next 24 hours (the period of greatest risk). Although there is no exact consensus among authors as to when surgical decompression should be performed, pressures above 30-40 mm Hg or a difference of less than 30 mm Hg between compartment pressure and diastolic pressure are considered an indication for fasciotomy. If pressures remain above 40 mm Hg for 8 hours, irreversible permanent lesions occur.[13-16]

There are accepted indications for measuring compartmental pressure[1,10]:

– At least 1 symptom of compartment syndrome together with confusion factors.

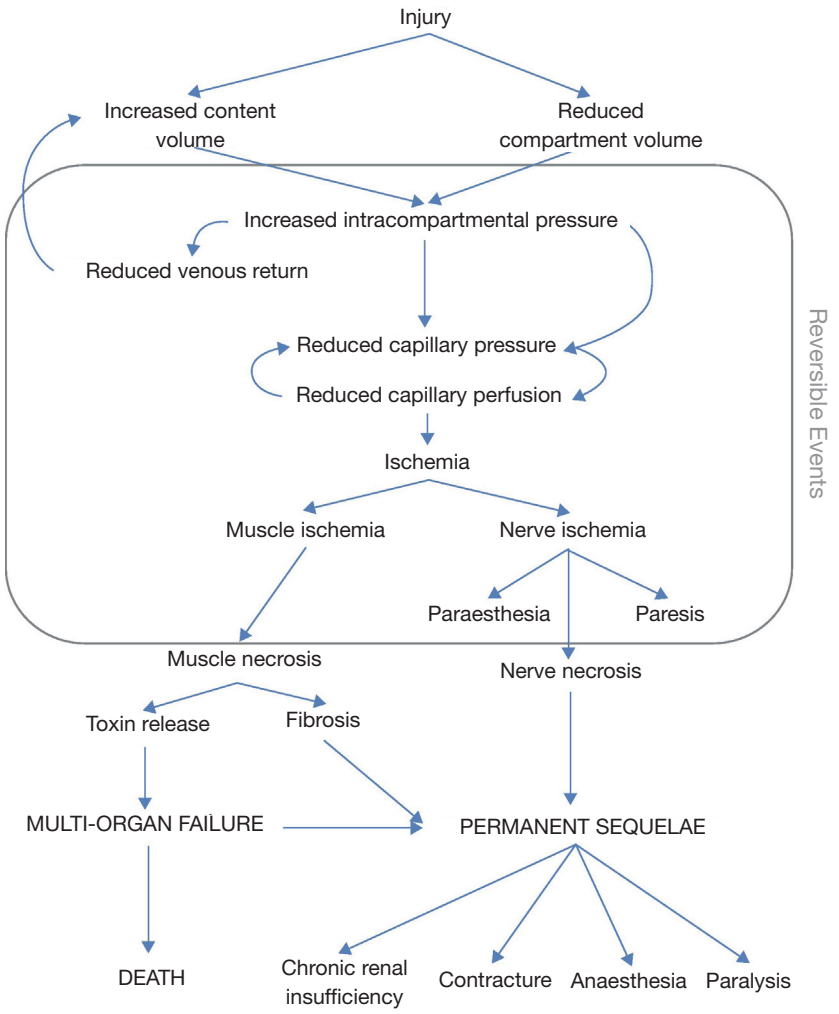

Figure 1 Pathophysiology of compartment syndrome.

- Post-operative induration or inflammation of an extremity in a patient who has undergone regional anaesthesia.
– Unreliable examination when there is induration or inflammation of an extremity.
- Prolonged hypotension in an inflamed extremity of questionable induration.
– Spontaneous increase of pain in an extremity following appropriate treatment for pain.

Laboratory tests

Certain laboratory tests may contribute to the diagnosis of compartment syndrome. Creatine phosphokinase (CPK) elevation reflects muscle necrosis and will show a declining trend following compartment decompression. The persistence of elevated levels is an indication that muscle necrosis is continuing and suggests that decompression was insufficient. Myoglobinuria also confirms lysis of muscle cells. Myoglobin is toxic to the renal glomerulus, so if the compartment syndrome is not properly treated, renal insufficiency will develop.[2,10]

Treatment

Fasciotomy is the treatment of choice. The only reliable means of treating an established ACS is surgical decompression via fasciotomy. The majority of authors[1,2,4,10,16,17] agree that intracompartmental pressures above 30 mm Hg or a difference of less than 30 mm Hg between the compartment pressure and the diastolic pressure are indications for fasciotomy. When there is uncertainty but reason to think that the symptoms may progress to a compartment syndrome, we support decompression fasciotomy. It is also advisable to perform prophylactic fasciotomy as a matter of course in cases of complex fractures of the extremities, crushing injuries, limb re-implantation, or neurovascular lesions. In addition to treatment by local decompression, it is absolutely necessary that the patient be hyperhydrated, which will help to prevent damage to the renal glomeruli from myoglobin and toxic metabolite deposits.

Generally speaking, for decompression in the foot, the approach will be two longitudinal dorsal incisions located between the 1st and 2nd and between the 4th and 5th

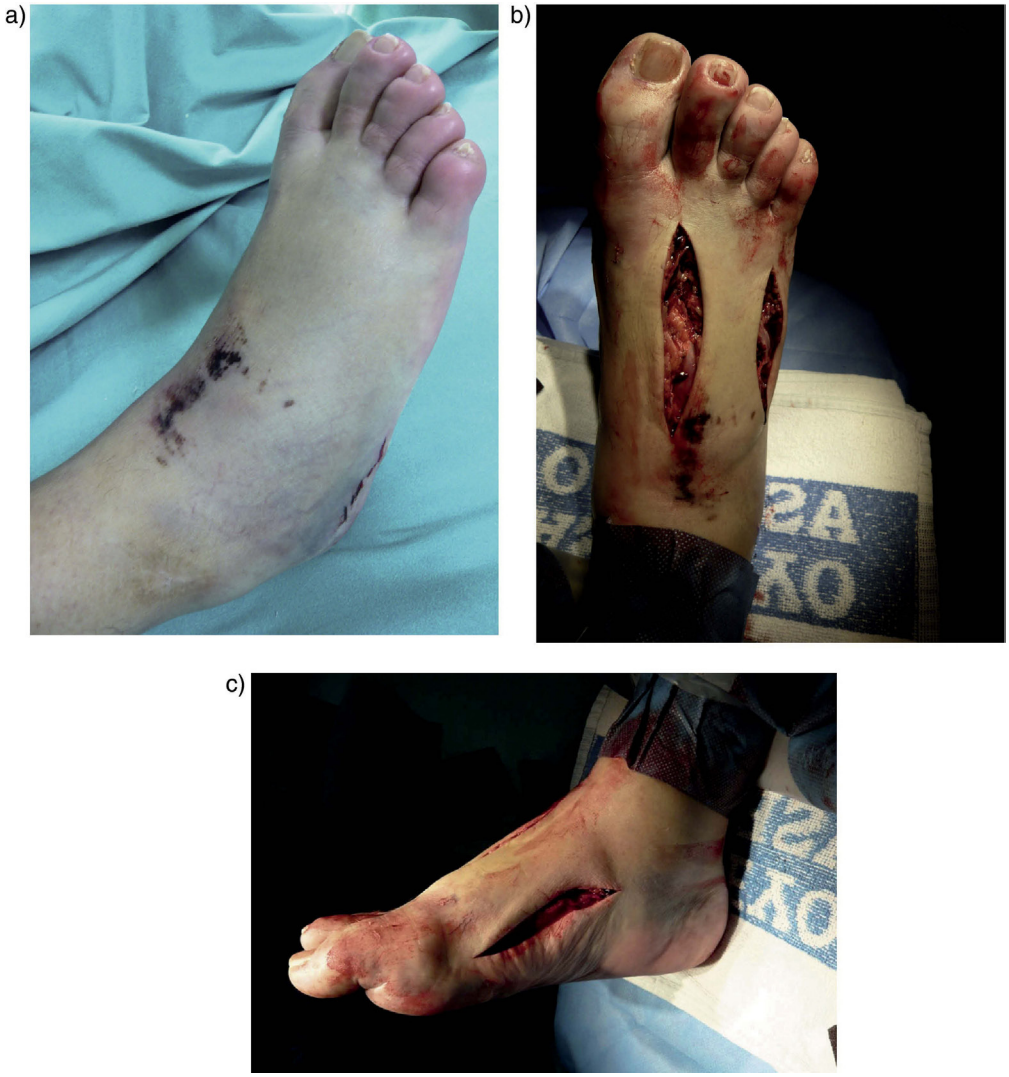

Figure 2 Compartment syndrome in a crushing injury of the foot. a) Clinical appearance of the foot. b) and c) Dorsal and medial compartmental fasciotomy incisions for surgical debridement.

metatarsals to access the forefoot compartments (fig. 2B) and a medial incision (fig. 2C) for decompressing the calcaneal, medial, superficial, and lateral compartments; however, the lateral and superficial compartments can also be accessed via the dorsal approaches.[10,15-17] Fasciotomy includes incision of the compartment's aponeurotic layer, which allows the tissues to expand without restriction and the pressure in the tissues to drop. In performing the fasciotomy, not only must the compartment's aponeurotic layer be opened quickly but the overlying skin and subcutaneous tissue must also be opened for proper decompression of the compartments. Decompression in an ACS should not be performed percutaneously or via mini-incisions. After the incision is made, careful dissection of all compartments should be undertaken, evacuating the retained haematomas and ensuring that no noble structures are damaged. It is important to check that there is no bleeding, which could cause structures to be compressed again, and distal perfusion as well as muscle condition must be assessed. Fractures will be stabilized and, finally, wounds will be left open, without closure of either fascia or skin. Edge approximation sutures crisscrossing the wounds may cause decubiti and ischemias over the muscle tissue and are not recommended. It is preferable that they be avoided or that some loose suture be placed to join each edge of the wound with the underlying fascia or muscle. We will dress the wounds with gauze impregnated with silver sulfadiazine or nitrofurazone ointment, which debrides, promotes granulation, and provides anti-bacterial protection.

Strict monitoring is recommended following fasciotomy. Wound treatments and dressing changes should be performed daily. As far as subsequent treatment, the principles of the primary treatment are followed: assess the muscle for viability, extirpate necrotic muscle, and leave the wounds open. Fractures should be stabilized via internal or external fixation. Stabilization facilitates wound care and permits patient and joint mobility which, in turn, reduce the development of fibrosis and joint rigidity. At 24-48 hours,

patient should be checked to see whether further necrosis has occurred and whether further surgical cleansing is required.[18-20]

The wound should not be closed until all necrotic tissue has been debrided. The type of closure will depend on the nature of the open wound. If subsequent progress is such that the wound edges can be approximated without tension, patient will undergo deferred primary closure of the skin and subcutaneous tissue. If the edges cannot be approximated but there is good granulation tissue with no exposed vessels, nerves, or osseous tissue, a free skin graft covering may be applied. When bone or noble tissues are exposed, coverage with a microvascular fasciocutaneous free flap or graft will be required. Vacuum-assisted closure has been shown to be effective for resolving tissue inflammation and oedema quickly following fasciotomy by increasing local blood supply, promoting tissue granulation, and inhibiting bacterial colonization.[21]

Finally, some authors[22] use hyperbaric oxygen therapy in patients with crushing injuries or early-onset compartment syndrome, for it appears that this therapy reduces the risk of late necrosis and ischemia by increasing oxygen release in the tissues, reducing oedema, and improving leukocyte function. However, the optimal time for implementation and the appropriate dose of this therapy are not clear.

It is extremely important that ACS be diagnosed early, which is why it must be brought to mind. Prophylaxis is essential, and the best treatment for ACS is to prevent its development. This requires a proper clinical history and initial examination, with special attention to recording information on the nerve, vascular, and muscle examination.

Correct reduction and fixation of fractures, with little manipulation, and proper placement of bandages and casts are also essential so as not to aggravate the lesion and to avoid compromising the compartment.

With injuries that may progress to an ACS, we must be alert and institute strict routines for clinical monitoring (pain, mobility, sensation, pulses, oedema) along with monitoring the intracompartmental pressure.

A delay in treatment may have disastrous consequences, such as contracture and paralysis, infection, and sometimes amputation.

We must avoid closure under tension, in cases of open surgery, and we advocate prophylactic opening of the compartment in the case of surgery of the foot with multiple injuries.

Conflict of interest

The authors declare that they have no conflict of interest.

Evidence level

Evidence Level V.

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