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Compartment syndrome

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Introduction

Compartment syndrome is one of the most frequent and severe complications after limb trauma. It is characterized by increased intra-compartmental pressure in a rigid space, which compresses muscles, small vessels and nerves, in a sequence of pathological processes which rapidly develops extensive thrombosis in microcirculation, irreversible ischaemia, nervous damage and muscular necrosis of one or more compartments, which can be followed by fatal general complications (MSOF). Decompressive fasciotomy is indicated to be done as soon as compartment syndrome is suspected, in order to avoid these situations which threaten the viability of the affected limb and the life of the patient. (Revista de Medicină de Urgență, Vol. 3, Nr. 1: 27-39)

Key words
Compartment syndrome (CS), intra-compartmental pressure (ICP), microcirculation, decompressive fasciotomy.

Definition

The Compartment Syndrome is defined as the ensemble of anatomic, pathological and clinical manifestations following the increase in pressure in a space of the body, surrounded by an inextensible structure, generally called «compartment» [1, 2].

So defined as «the increase of intra-compartimental presure» (ICP), the compartment syndrome is the expression of an imbalance, in a certain part of the body, between the capacity of an inextensible container and its contents, with a tendency towards a relative increase in volume; this imbalance can be explained:

– either by the decrease in container capacity
– either by the increase in content volume, in an inextensible space [3].

History

The first description of the consequences of uncontrolled increase in intra-compartmental pressure is attributed to Richard von Volkmann, who in 1872 published the notes regarding nerve damage and muscle contracture at forearm level complicating the compartment syndrome after a closed supra-condilian fracture, ortopedically reduced, constrictively imobilised. This lesion has since been known as «the Volkmann contracture» [4]. The treatment for compartment syndrome was first described by Petersen in 1888 and the first experimental therapeutic demonstration belongs to Jepson (1926) [5]. In 1941, Bywaters and Beall published research regarding the onset of CS after crushing during the Second World War. [6]

Localisation

What must be emphasized is that the concept of «compartment syndrome» (CS) is not specific to orthopedics, as the same pathological phenomenon occurs in different inextensible spaces in the body (e.g. the hepatic capsule, the pancreatic capsule etc.)

Moreover, phenomena that fit within the definition of CS have been described at the level of the locomotory system, especially in muscular compartments, when the container consists of the inextensible fascia, but also in bones, when the container consists of inextensible bony structures and the pathological phenomenon is the increase in intrabone pressure. This sequence, assimilated to a CS is now recognized as one of the pathogenic theories of aseptic idiopathic necrosis (most frequent in the femoral head) and called «intra-osseous compartment syndrome».

In muscular compartments, CS occurs most frequently in segments with a skeleton consisting of two bones (the shank and the forearm) and, of these, the most common localization is the CS of the shank. Though less common, localizations at thigh and arm level are not exceptional and require the same treatments as a CS located in the shank, otherwise the evolution of the traumatism complicated by CS will also worsen through the necrotic muscular component. In addition, CS has been described at hand and foot level, with pathology and treatment similar to CS in other locations.

The localisation in segments with a two bone skeleton can be explained by the rigid character of one of the elements of the compartment: thus, the shank and the forearm have compartments bordered on one side by fascias, on the other by the two bony segments and the practically inextensible inter-osseous membrane. [7]

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Therefore, the following muscular compartments are described:

1. The anterior compartment, which contains the muscles that peform the dorsal flexion of the leg (m. tibialis anterior, m.extensor digitorum longus, m.extensor hallucis longus, m.peroneus tertius), as well as the anterior tibial artery (which is often affected in fractures of the tibial plateau) and deep peronier nerve, which ensures the sensitivity of the first dorsal inter-digital space.
2. The lateral compartment, which contains part of the muscles that peform the plantar flexion and the eversion of the foot (m.peroneus brevis, m.peroneus longus), as well as the superficial peronier nerve, which sensitively innervates the dorsum of the foot.
3. The posterior compartment has two components:

– The deep posterior compartment, which contains muscles that peform the plantar flexion of the foot and the flexion of the phalanx (m. tibialis posterior, m. flexor digitorum longus, m. flexor hallucis longus), the posterior tibial artery, the peronier artery and the tibial posterior nerve, which ensures plantar sensitivity, and
– The superficial posterior compartment, which contains m. gastrocnemius, m.plantaris and m. soleus, all of which participate in the plantar flexion of the foot , and the sural nerve, with the role of sensitive innervation of the lateral part of the foot and the distal shank. [8]

Clinical Forms

From the point of view of the moment and the cause of the onset of CS, there are two clinical forms:

1. The acute form of SC, which develops rapidly after the action of the factor considered an etiologic agent, requires emergency treatment adapted to the evolutive phase and, untreated, evolves towards muscular necrosis and ischaemia by compromising arteries, beginning at microcirculation level. The acute form itself has 3 phases: the initial phase, the state phase and the phase of local and general complications.
2. The chronic form of CS, which appears repetitively after an effort, has the same localization every time and stops after the effort stops. It has typical localizations, like at shank level in athletes and high jumpers, in the anterior part of the foot in those who march fast and long, at hand level in those who execute difficult manual labors [9, 10, 11, 12, 13] .

In what follows regarding the clinical symptoms and treatment, we will be referring only to **the acute form of post-traumatic compartment syndrom of the muscular compartments in the shank**.

Phisiopathology

Regardless of the clinical form, etiology and localization, physiopathology of CS in the acute form is the same, with 2 essential components:

1. pathological phenomena start at microcirculation level and are initiated by the increase in intra-compartmental pressure (ICP) over the value of the hydrostatic pressure in the capillaries (which is usually 30mmHg), and
2. through a pathological chain of events that follow and sustain each other rapidly, in the absence of an efficient therapy, the vitality of the limb segment is jeopardized and, even worse, the patient's life is jeopardized due to the onset of MSOF by «intoxicating» the body with products resulting from ischaemic muscular necrosis [14].

Two types of manifestations can thus be described, separated only for scholastic reasons, as they evolve together and influence each-other:

I. Local phenomena start with an increase in ICP over the value of capillary pressure, which reduces tissue irrigation to the point where the transfer of oxygen to the cells is no longer possible. Through a mechanism which tends to compensate for the increase in ICP, the tisular perfusion pressure increases. As the ICP increases, the process becomes self-sustained, the local compensating mechanisms are overwhelmed and a chain of unstoppable lesions develops [15].

It is known that tisular perfusion is calculated as the difference between capillary perfusion pressure (CPP) and interstitial pressure (IP). Normally, the cellular metabolism requires an oxygen pressure of 5-7 mmHg, which is easily maintained when CPP reaches 25-30 mmHg and ICP reaches 4-6 mmHg. If, however, ICP increases, the compensating mechanisms tend to increase venous pressure, as Matsen demonstrated. [16, 17]

When venous pressure exceeds CPP, capillary collapse occurs. What has not yet been established with certainty is the value at which it occurs, however it is beyond doubt that at an ICP of 30 mmHg a decompressive surgical procedure is absolutely necessary, as at this point capillary blood flow disappears, which leads to stopping the supply of oxygen to the cells, causing all the phenomena consequent to cellular anaerobiosis. [18]

An increase in ICP therefore produces:

– obstruction (through extrinsic compression) of the capillaries, as well as most of the post-capillary venules, thereby creating the pre-requisites of the **stasis** in the territory of microcirculation; as a result of stasis and hypoxia, capillary permeability increases, which leads to the extravasation of the liquid in the vascular lumen and, thus, to an increase in blood vis-

cosity. The hypoxia worsens tisular lesions, which leads to the release of tisular tromboplastine, a strong pro-coagulant. In this way, the elements of the Virchow triad appear: stasis (decrease in blood circulation speed), increase in viscosity and production of factors that initiate coagulation, elements which lead to the onset of capillary thrombosis, which quickly becomes extensive, not only in the capillary territory, but also towards the other small vessels, arterioles and venules, from which the thrombosis extends itself towards the meta-arterioles and post-capillary venules [19]. From meta-arteriole level, the thrombotic process advances towards the arterioles and the medium arteries, from where it extends itself centripetally, towards the main arterial axes. Thus develops irreversible, therapeutically surpassed ischaemia, as its centripetal evolution renders useless all maneuvers to clear large arteries, because the periphery is compromised and the muscles are necrosed. Moreover, muscular necrosis may appear, in certain areas, before the onset of ischaemia, especially if that particular area of the muscle has been subjected to the traumatism, which leads to an apparently «paradoxal» situation of muscle necrosis without major damage to the arterial axes. It is considered that, if left untreated, these phenomena lead to muscular necrosis and irreversible nerve damage within 6-10 hours. This situation is not at all abnormal or inexplicable, but knowing the physiopathology of compartment syndrome creates the pre-requirement for understanding it and, more importantly, preventing it.

– at the same time, the increase in ICP leads to the obstruction of the *vasa vasorum*, which affects the structure of the small and medium artery wall, worsening the suffering initially caused by their compression, which leads firstly to the alteration of the subendotelial layers, with two consequences: firstly, the injury of the endothelium leads to the loss of its anti-thrombotic properties, secondly, uncovering the endothelial collagen augments the pro-thrombotic status created by the endothelial disruption. Thrombosis in medium and large arteries is also caused by their initial compression and decreasing the blood circulation speed; all of these are stimulating factors for thrombosis.[20]
– an increase in ICP leads firstly to nerve compression, which causes premature onset of sensitive neurological suffering, in addition to obstruction of the vasa nervorum, which affects medium and large caliber nerves, causing motor phenomena and aggravating the sensitive ones, neurological aggravation being rapid in the absence of efficient therapeutic measures. The particularity of neurological suffering in CS, which also represents an important element of differential diagnosis, is the onset of neurological suffering **before** the onset of API, the explanation being the suffering of the nerve fibres through direct compression.

In conclusion, local phenomena in CS have the following characteristics:

1. vascular and neurological injuries begin in the structures of the smallest caliber and evolves towards those of larger caliber.
2. initial vascular injury is at microcirculation level, where, **in the absence of efficient treatment**, thrombosis develops **quickly**.
3. once extensive capillary thrombosis sets in, ischaemia becomes irreversible (as the muscles are compromised), although **the pulse may still be present**.
4. thrombosis spreads centripetally towards medium and large arteries. Until these are obstructed, we can detect arterial pulsations. The obstruction of medium and large arteries through the spread of thrombosis only increases the speed at which muscular rigidity sets in and causes the apparition of the «macroscopic» clinical sign of API- lack of pulse in the large arteries.
5. sensitive injury sets in immediately after the start of the increase in ICP (through direct compression of the vasa nervorum), being **prior** to the appearance of the clinical symptoms typical of API.

II. General phenomena The apparition of muscular necrosis areas in the territory affected by increased ICP leads to specific general modifications in metabolism, objectified by the increase in the values of muscular necrosis enzymes (CK and LDH). As a result, in the state phase of CS, mioglobine, the main product of muscle degradation, appears in the systemic circulation, which first of all affects the hepato-renal function to various extents, depending on the severity of the process of muscle necrosis.

If therapeutic intervention does not stop the local pathological phenomena before the onset of extensive capillary thrombosis, then the muscles of the affected muscular compartment die completely, the systemic discharge of mioglobine is extensive, as it even appears in urine, and the general phenomena amplify, especially the alteration of the hepatic and renal functions.

If there is no therapeutic intervention before the onset of API, the general clinical symptoms amplify, presenting the elements characteristic of the systemic echo of irreversible API. The onset of API causes general phenomena that affect the functionality of the vital systems and organs.

If this "cascade" is not stopped, MSOF sets in, which may lead to the lethal evolution of the condition. This is why we can adhere to the conclusion stipulated by Richard Paula (Director of Research, Emergency Medicine Residency Program, Tampa General Hospital, University of South Florida at Tampa), who, using a pun, described very precisely the severity of CS, underestimated before its exact physiology was known:

"Compartment syndrome (CS) is a **limb-threatening and life-threatening** condition" [21]. Therefore, CS should not be seen as a strictly localized condition. It is a pathological entity which affects the entire body, systemic

complications of overdue CS being those of irreversible API, ranging as far as exitus.

Etiology

Compartment syndrome can appear under two types of situations which will be analyzed in the following:

1. Increase in content volume can be produced by oedema (interstitial fluid) or blood (localised haematoma or diffuse bleeding which leads to a diffuse intra-muscular haematoma)

Intra compartmental haemorhage can appear after:

- Major vascular injuries without fractures [22]
- Fractures complicated or not with vascular injuries
- Post-operative complications – after reaming, osteotomies,
- Coagulopathy

Most of the times, the mechanism which increases the content volume is **oedema**, which can be caused by:

- Trauma
- Burns
- Ischaemia, including haemostatic compression devices
- Reperfusion syndrome following revascularisation
- Venous or lymphatic obstruction,
- Nephrotic syndrome,
- Intra-arterial medication,
- Exagerated physical effort

There are certain situations when both haemorrhage and oedema increase content volume, such as:

- Open or closed **fractures** [23, 24],
- Post-operative – after reaming, [25, 26], osteotomies, lengthenings, artroscopies. [27]

2. **Decrease in container capacity** appears after:

- Fascial sutures
- External compresions by cast or bandages
- Inextensibile scars
- Scars after burns
- Compressions due to other mechanisms, including positions during surgery [28, 29]

The main mechanism implicated in post-traumatic CS is increase volume content by **blood and oedema,** while decrease in container capacity can be implicated when after trauma, a cast or a bandage is necessary, and this becomes constrictive. Even in this case it is very difficult to establish whether external compression by constrictive bandage is the only damaging condition, or if it adds its effect to that produced by post-traumatic oedema, thus contributing in almost equal proportions to CS generation. [30]

Symptomes and signs

The evolution of CS consists of the following phases:

1. the alarm (initial) phase

2. **the state phase (constituted CS)**, after which two posibilities exist:

- the treatment is efficient and the resources of the organism successfully lead to the **healing phase**, the local evolution is favourable and the fasciotomy will be covered after approximately 1 week, by secondary suture or skin grafting, or,
- healing resources are overwhelmed, irreversible ischaemia appears starting within the microcirculation- a phase of **local complications** (overwhelmed acute peripheral ischaemia – API- without MSOF), or **general complications** (overwhelmed API with MSOF).

1. The alarm (initial) phase

The clinical elements characterizing the alarm phase of the post-traumatic CS of the calf, acute form, are the following:

- pain – unusually severe, compared to the nature of the trauma, which is enhanced by passive elongation of the affected muscular compartment (for example, if CS affects the anterior compartment, pain is augmented by passive plantar flexion)
- enlargement of the circumference of the shank, compared with the healthy one,
- minor increase in consistency of the muscular compartment
- no neurological signs
- minimum skin oedema on all the shank
- **Arterial pulse** (posterior tibial artery and pedious artery) is present **unless** arterial damage coexists with CS

Differential diagnosis is to be done with:

- deep venous thrombosis, when pain is situated in the calf and is enhanced by passive dorsal flexion of the foot, but also by the compression of the calf
- post-traumatic oedema
- other disorders which produce increase in the size of the shank: infection (the infectious syndrome also appears), tumors (the deformation is prior to the trauma), chronic venous insufficiency (elevated position decrease the oedema and Doppler examination shows the pathological venous condition).

After differential diagnosis is carried out, we can establish the diagnosis of «Compartment syndrome, alarm phase», which is to be added to the diagnosis of the primary injury.

Positive diagnosis of CS is established by measuring the intra-compartmental pressure (ICP), which is higher than normal, but still **under** 30 mm Hg, but the most important thing is the clinical assumption.

2. The state phase

Clinical characteristics of this phase are:

- Pain within the muscular compartment, which spreads rapidly distally (from the site of the traumatic injury to the toes), where it becomes significant. The pain is enhanced, as described when presenting the initial phase, by passive movements of the ankle which produce elongation of the affected muscles.
- Enlargement of the shank, compared with the healthy one, medium oedema of the ankle and foot, which is not typical,
- Increase in consistency of the affected muscular compartment, which is called, in severe cases, to be "wooden".
- The skin is shiny, tensioned, with venous circulation visible.
- Decrease of the force of the muscles of the affected area.
- Paraesthesias localised distally, which are then followed by hipoaesthesia, localised distally (at the toes) at first, and then extending proximally.
- Partial functional impairment of the active movements, starting distally, at the toes.
- **Arterial pulse is present** (at the arteries of the ankle and foot) until irreversible API onset. Most of the times, when the ankle and the foot are swollen, arterial pulse is diminished, but they are detectable palpatory and using Doppler ultrasound. Anyway, when inequality of the pulse appears, it must be understood as an alarm sign suggesting that there is a factor which affects local blood flow, which requires permanent monitoring, in order to avoid irreversible API. Depending on the affected compartment, the symptoms are:
- CS of the anterior compartment is characterized by: anterior pain which is enhanced by passive plantar flexion of the foot, diminished force of dorsi-flexion, and, due to early peroneal nerve injury, paresthaesia and decreased sensibility of the first interdigital space of the foot [31]
- CS of the posterior compartment is characterized by posterior pain, augmented by walking on the heels (when it is possible) , by passive dorsiflexion of the foot, with diminished force of plantar flexion. After differential diagnosis as described before, positive diagnosis of "Compartment syndrome of the shank- the state phase" is established by measuring the intracompartmental pressure (ICP), which is considered to be the speciffic element. Different systems were used in order to measure ICP, all these manometers using a needle introduced in the muscle, connected to a mechanical or electronic device.

Most of the authors agree that CS diagnosis requires a value of ICP higher than 30 mmHg, but debates concerning this problem still exist., since others consider that 45mmHg must be the ICP considered speciffic for CS. [32, 33, 34, 35]. Another opinion is that CS should be diagnosed when the difference between diastolic arterial pressure and ICP is less than 30 mmHg. [36]

Recent researches underlined the importance of **differential pressure** (DP) in understanding the phenomena which characterise CS. The differential pressure is the difference between the diastolic arterial pressure (DAP) and intracompartimental pressure (ICP).

DP = DAP - ICP

Whiteside suggested that values of DP arround 10-12 mmHg are enough for inducing pathological changes in microcirculation [37], while other researches on animals showed that these changes appear even after ICP raises at values which are 30 mmHg less than DAP. [38].

Besides the invasive technique, PIC can be evaluated using non-invasive methods, such as methoxy isobutyl-isonitrile and phosphate-MRI, technetium-99m (^{99m}TC) and xenon (Xe)-scintigraphy, thalium-chloride scintigraphy combined with SPECT (single photon emission computed tomography) which are useful for diagnosing the compartment syndrome. [39]

Establishing 30 mmHg as the limit value of the ICP beyond which CS develops is due to the researches which demonstrated that elevated ICP to 20-30mmHg produces pain and paresthaesia (normal ICP in rest is 8-10 mmHg), while other authors sustained the idea of DP being the initiating factor of the muscular injuries. [40, 41].

Also useful in the diagnosis of CS, the values of CK and LDH are considered to reflect the amount of the muscular necrosis. Their values in patients with CS are higher than normal, and their evolution is suggestive for the prognosis of the patient.

Treatment

The treatment of the CS is differentiated, depending on the phase, as following :

- The alarm phase: the treatment of the basic trauma, medical treatment and non-speciffic treatment of the oedema
- The state phase: **decompressive fasciotomy**, the treatment of the basic trauma, medical treatment and non-specific treatment of the oedema
- The phase of CS complicated with irreversible API without MSOF (muscular necrosis of the shank): Thigh amputation (do not suture!) and systemic sustained treatment for MSOF prophylaxis.
- The phase of CS complicated with irreversible API with MSOF: Thigh amputation and systemic sustained treatment, including dyalisis, if necessary. The term "treatment of the basic trauma" refers to the orthopaedic or surgical treatment of the injury which then developed the CS as a local complication.

It must be underlined that the CS of the shank may appear after various traumatic injuries, affecting not only

the shank, but also the knee and the ankle **[42], injuries which produce bleeding or intra-muscular oedema:**

I. Fractures of the shank: fractures of the tibial plateau (Figure 1–3), of the dyaphisis, with or without fracture of the peroneum, of the distal tibia [43],

II. Contusions of the shank, followed by post-traumatic oedema or by muscular injuries with intra-compartmental bleeding,

But also :

III. Trauma of the knee which affect the popliteal vessels and produces elongation of the triceps suralis, which comes from above the knee :

– Knee dislocations,
– Fractures of the distal femur,
– Capsular and ligamentous injuries of the knee, producing articular instability

IV. Trauma of the ankle, which produce the elongation of the muscless, followed by bleeding or oedema :

– fractures – dislocations of the ankle,
– Ankle sprains

The onset of the CS after one of these trauma is favourised by:

1. considerable muscular mass,
2. maltreatment of the traumatised limb- continuing the effort after trauma, keeping the leg downwards, massage on the muscles with revulsive ingredients, constrictive bandages,
3. combination of the traumatic mechanisms, for example: a ski injury, produced by falling with rotation of the shank on the ankle fixed on the ground, may produce a sprain of the ankle (by inversion) with spiroid fracture of the tibial dyaphisis and of the proximal peroneum (so the inter-osseous membrane is intact) in a patient with hypertrophic muscles, can get complicated with CS of the shank, especially without the proper treatment.

The treatment of the compartment syndrome **in the initial phase** is:

Local treatment

I. Orthopaedic or surgical treatment of the primary injury (for example, osteosynthesis of the fracture, stabilisation of the dislocation following reduction), including the proper imobilisation of the affected limb [44, 45]

II. After cleaning the skin, local anti-inflamatory (non-steroidal- Diclofenac, Ibuprofen) and anti-thrombotic (heparin-derived substances, like those contained in Lioton, or Hepathrombine) ingredients are to be applied, without massage on the muscles and taking care of skin injuries, such as wounds, which must be covered with sterile bandages.

III. A large bandage with alcohol and aethacridinum lacticum (Rivanol) , which is a very strong anti-inflammatory and anti-thrombotic combination.

IV. Elevated position of the affected limb. [46]

General treatment

Consists of the following classes of substances:

I. Anti-thrombotics, preferably **low molecular weight heparines (LMWH): Enoxaparine, Nadroparine** etc. with doses adapted to each patient

II. Non-steroidal anti-inflammatory drugs

III. Dextran 40 with secondary anti-thrombotic role (!!! NOT Dextran 70, which has undesired effects in the pathology we are discussing))

IV. Medication that protects against adverse reactions of those mentioned above (especially gastric-protection, which is also necessary for the profilaxy of stress ulcer)

V. Treatment to hydrate and sustain diuresis

VI. If necessary, antibiotic treatment, adapted to the lesion for which it is indicated

VII. Although there is no agreement as to whether they should be used, it is considered that the following have beneficial effects in CS:

– Anti-oxidants, especially vitamin C500, in doses of 1 g daily
– steroidal anti-inflammatory, especially Dexametazone, which stabilizes the cellular membrane and protects (to a certain extent) against hypoxia-induced cellular necrosis

It is **unadvisable** to administer anti-coagulants (except when these are indicated for a lesion which is potentially vital-thrombotic or embolic) as, by modifying the bleeding-coagulant balance, they enhance/produce bleeding in the affected compartment.

The treatment of CS in the **state phase** consists, firstly, of **decompressive fasciotomy** of the affected muscular compartments, in addition to the specific treatment of the primary injury and the general treatment described above (to be noted that, once the fasciotomy is done, antibio-therapy becomes mandatory). Therefore, if CS is the complication of a shank fracture, then osteosynthesis of the fracture **must** be accompanied by **decompressive fasciotomy** and if CS appears as the complication of an injury that does **not** have surgical indication, only fasciotomy will be carried out. [47]

As for establishing the indication of fasciotomy, the discussions are the same as those described in establishing a diagnosis; there are two theories. One says that fasciotomy must practiced at a ICP of over 30 mmHg (Mubarak) [48], the other considers the difference between the diastolic and the ICP an indicator for carrying out decompression.

Begun in 1975, Whiteside's research lead him to indicate fasciotomy when ICP rises to values which come within 10-12 mmHg of the diastolic pressure, this difference being known as "delta-P"

In 1996, McQueen, studying CS in dogs, concluded that the difference between diastolic pressure and the ICP at which fasciotomy is indicated is under 30 mmHg and that the measurement of ICP relative to the diastolic is closer

to reality, therefore, if the diastolic is 70 mmHg, the ICP must not be more than **70-30 mmHg = 40 mmHg**

If ICP is greater than 40mmHg, therefore approaching diastolic, that is, the difference between them is 30 mmHg or less, fasciotomy is indicated [40].

Decompressive fasciotomy is carried out in the operating room by the team that treats the trauma of the limb segment. It consists of the incision of the skin and especially of the FASCIA which surrounds the tensed compartment; it is indicated to execute a fasciotomy on one or more compartments. It can therefore be done with one or more cutaneous incisions, depending on the number of affected compartments and the surgeon's preferences. Techniques have been described for subcutaneous fasciotomy (cutaneous incision of 8-10 cm, then subcutaneous fasciotomy proximal and distal on the entire length of the segment), which have been criticized because they do not permit a correct exploration of the vitality of the muscles and carrying out hemostasis (figure 4, 5).

Regardless of the number of incisions and of the chosen technique, it is **essential** that the fasciotomy attain to the purpose for which it was carried out, which is **to completly decompress the muscular compartment** (figure 6).

In order to do this, the following are recommended:

1. a large cutaneous incision from the proximal to the distal end of the compartment
2. **an incision of the fascia on the entire lenght** of the muscular compartment, otherwise the muscles will get hernia because of the too short incisions, which will increase muscular compression and necrosis (figure 7, 8).
3. detailed hemostasis
4. exploring the muscular compartments, excision of the devitalized areas, evacuating the hematomas, hemostasis
5. re-evaluating the tension in the muscular compartments after opening each of them; there are cases where decompressing one compartment automatically leads to a decrease in ILP in the others, especially when the inter-bone membrane is ruptured. [50, 51, 52]

After fasciotomy, the operated limb segment must be elevated in order to facilitate the remission of the post-traumatic circulatory disorders and, depending on the degree of retraction of the borders of the wound , covering the cutaneous defect will be done through secondary suture after the remission of the edema or through cutaneous skin graft, if suture is no longer possible.

If CS evolution passes the phase where the limb can be saved, it is necessary to carry out an emergency thigh amputation without sutures, in the inferior or medium third, depending on the extent of the thrombotic process proximal to the popliteal region, combined with specific measures of intensive therapy addressed to systemic imbalances.

Therefore, in the state phase of CS:

- **decompressive fasciotomy** will be anyway carried out regardless of whether the primary lesion is surgically indicated or not.
- **decompressive fasciotomy** has a **curative** function (it can also have a **profilactic** function, when it is carried out after revascularization to prevent the onset of CS as a clinical manifestation as a part of tisular reperfusion syndrome) [45]
- **decompressive fasciotomy** must be done widely, on the entire length of the affected muscular compartment or it will become harmful itself (figure 9, 10).

As we have specified that the certain diagnosis of CS is made on the basis of ICP measurements, which need a manometer, one can ask:

What is the terapeutic atitude in case the traumatology unit does not posses a manometer?

The answer is based on the elements described above, which emphasize the necessity of a **premature**, **quick** therapeutic intervention before the onset of ischaemic phenomena:

Once the **presumptive diagnosis** for CS in the state phase has been established, **emergency decompressive fasciotomy is indicated and carried out.**

Moreover, a CS in the initial phase will be intensely monitored: if, under local and general treatment circulatory phenomena do not get better OR, worse, aggravate, then the situation is assimilated to the state phase and **fasciotomy is done as an emergency**.

Whenever there is a suspicion regarding the onset of a CS, especially if the patient and the type of trauma fit within the profiles described above, **emergency fasciotomy is carried out**. In these circumstances, with a quasi-profilactic function, a fasciotomy is much less harmful to the patient than its delay (under the pretext of avoiding an extra incision) until the onset of irreversible phenomena.

Conclusions

1. CS of the muscular compartments in the shank can appear after a GREAT VARIETY of traumatisms, not necessarily accompanied by fractures
2. The moment of onset and the extent of the lesions depend on many factors (the patient's muscle mass, the severity of the traumatism, local circulation conditions, post-traumatic treatment)
3. CS physiopathology is dominated by microcirculation phenomena, which, in the absence of adequate treatment (depending on the moment of onset and the measures taken), evolve in a cascade until irreversible API, which jeopardizes the vitality of the affected limb segment and even the patient's life once multiple system and organ failure (MSOF) sets in
4. CS treatment must be initiated when clinical suspicion appears, even in the absence of ICP measurements (which need manometers, not always

available). In other words, once the clinical presumptive diagnosis is established as CS, it is best that we act as if CS were confirmed, even if there is no material possibility to measure ICP

5. Decompressive fasciotomy must be carried out as soon as there is a suspicion of a CS evolving towards state phase.
6. The promptitude and correctness of the fasciotomy influence both the affected limb segment and the patient's life, threatened at the moment of the systemic response to the irreversible lesions of therapeutically overdue CS (figure 11-14).

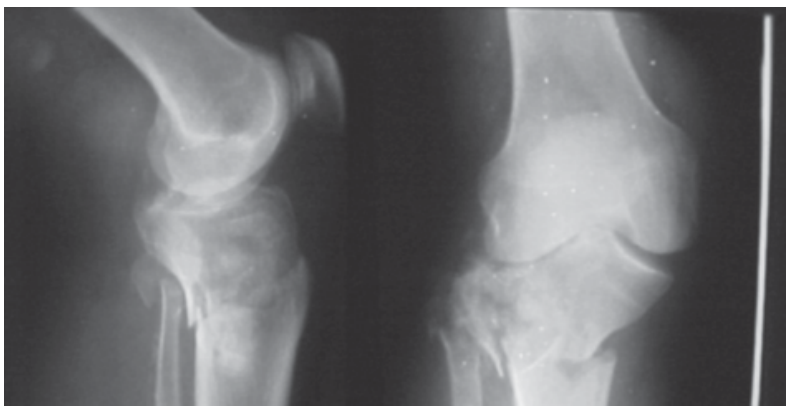

Figure 1 Case 1.
Comminuted fracture of the proximal tibia

Figure 2 Case 1.
Clinical aspect – enhanced diameter of the affected limb

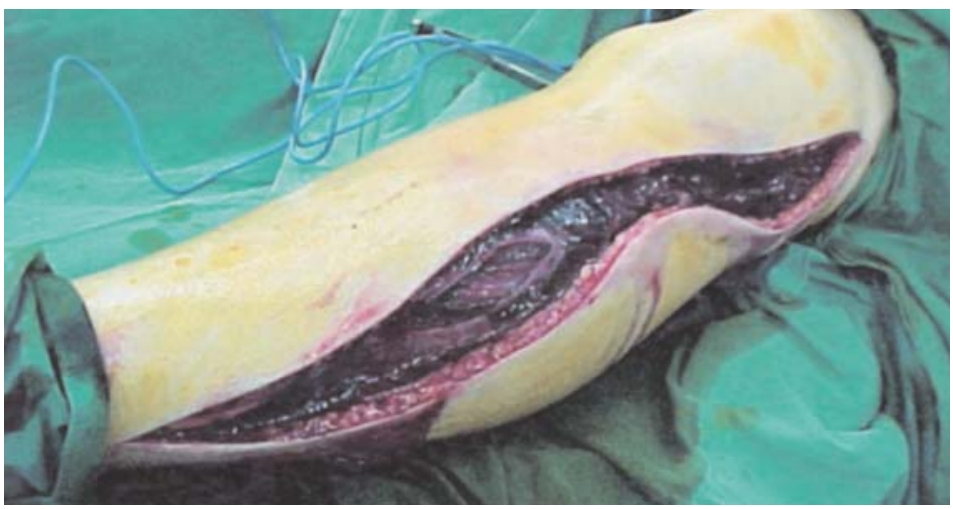

Figure 3 Case 1.
Internal fasciotomy

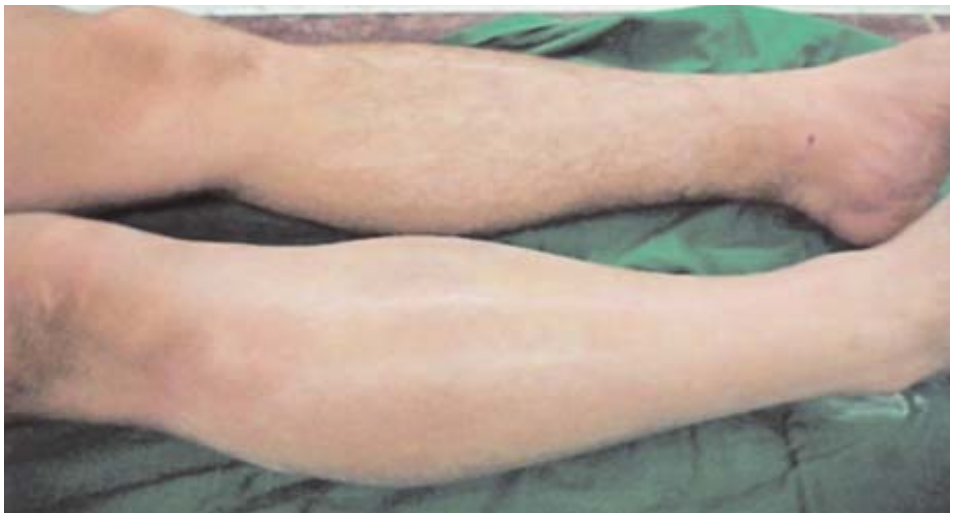

Figure 4 Case 2
Clinical aspect – enhanced diameter of the affected limb

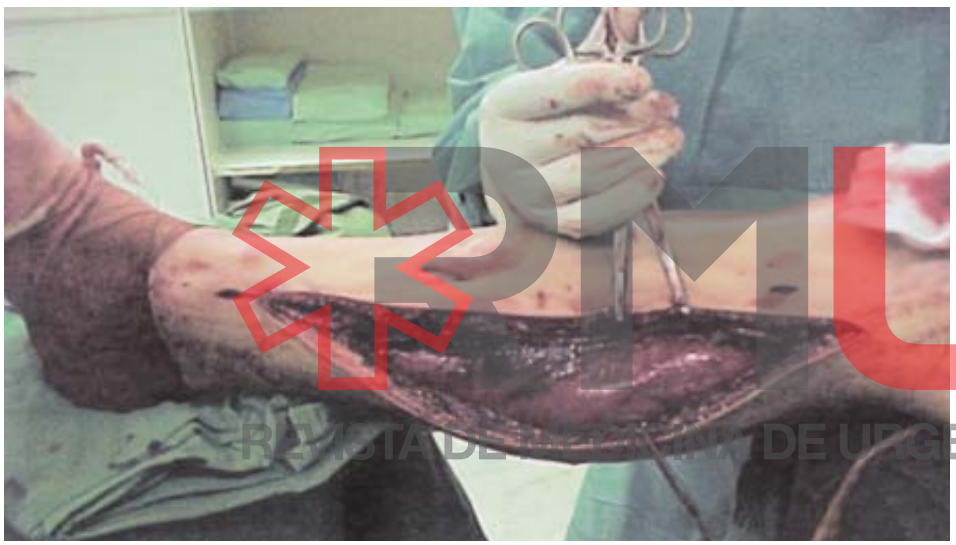

Figure 5 Case 2
Internal fasciotomy

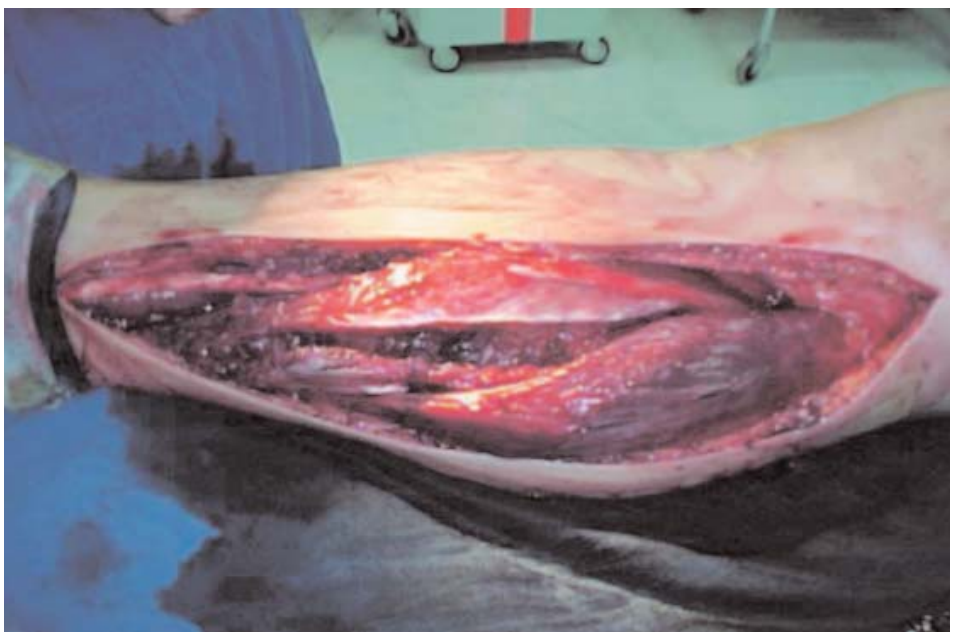

Figure 6 Case 3
Effective fasciotomy

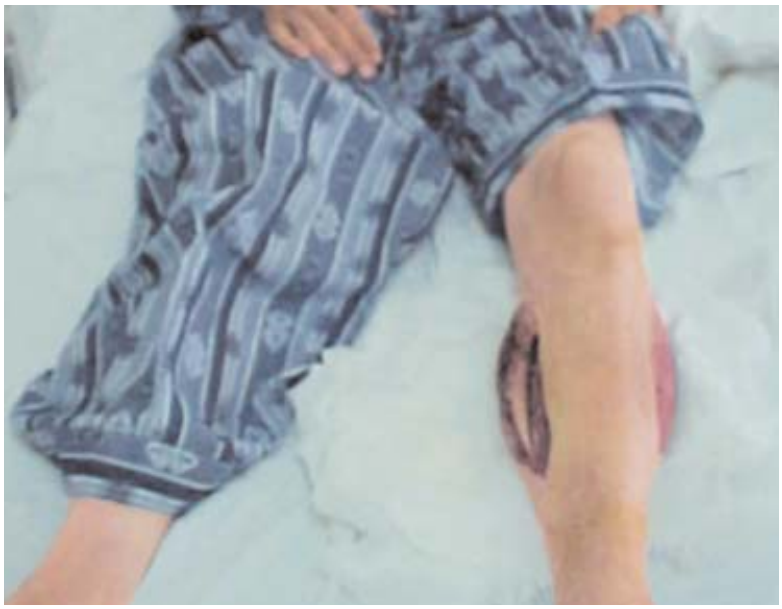

Figure 7 Case 4
inefficient fasciotomy
– too short

Figure 8 Case 4
Detail – muscular necrosis

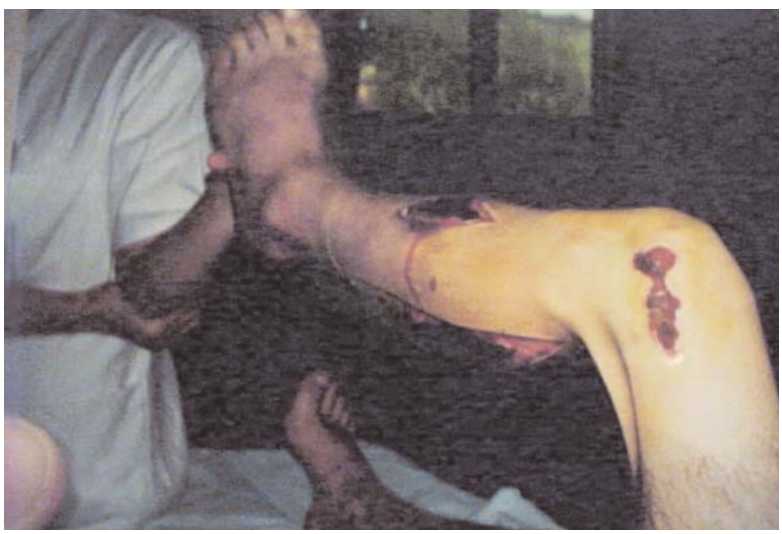

Figure 9 Case 5
Irreversible acute peripheral ischaemia after CS with inefficient fasciotomy – anterior aspect

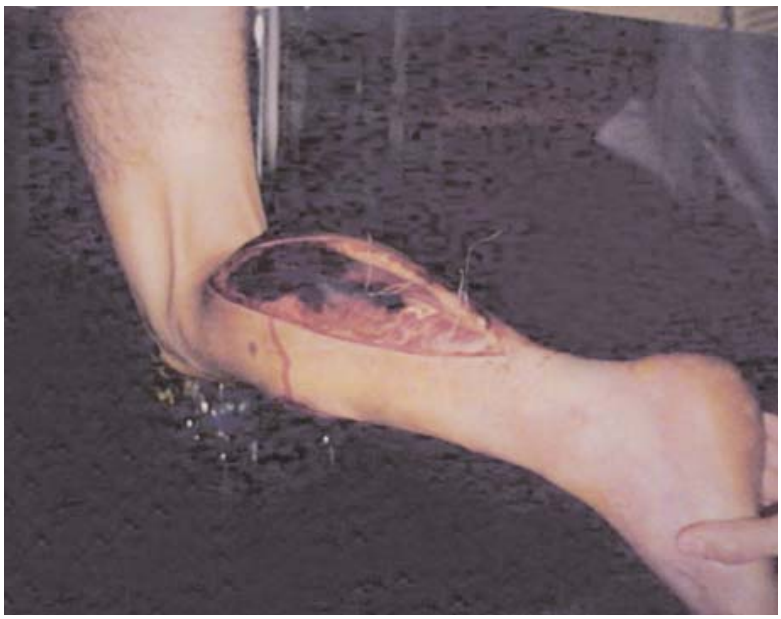

Figure 10 Case 5
Posterior aspect

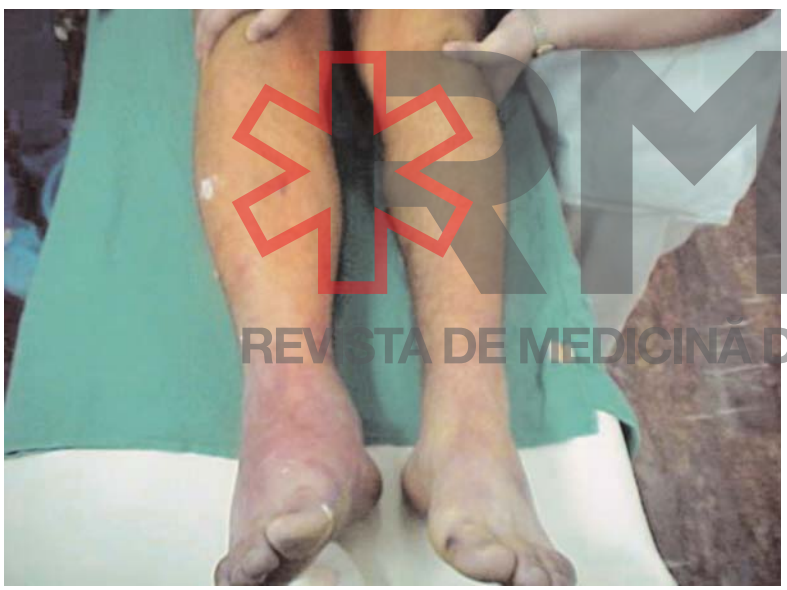

Figure 11 Case 6
Irreversible API with MSOF after CS (overwhelmed)

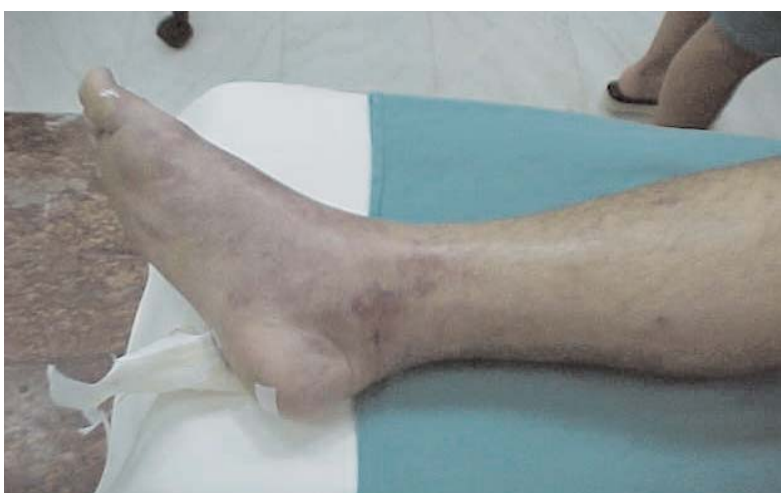

Figure 12 Case 6
Ischaemic gangrene after CS

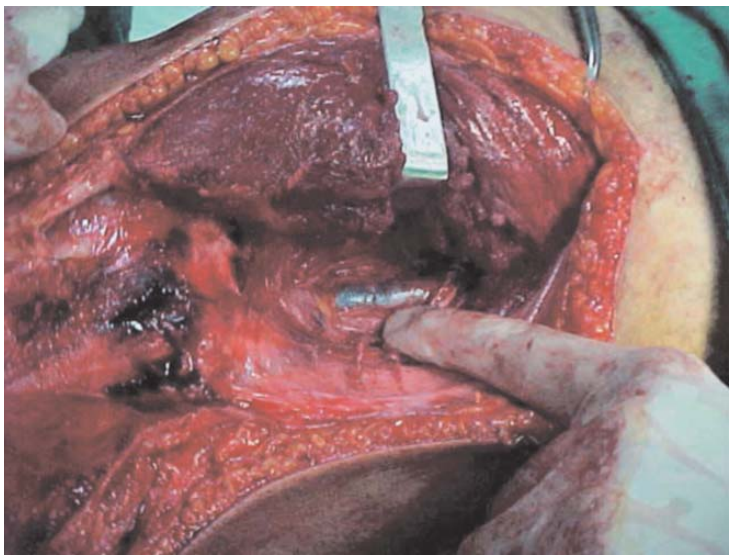

Figure 13
Thrombosis extended into bigger vessels

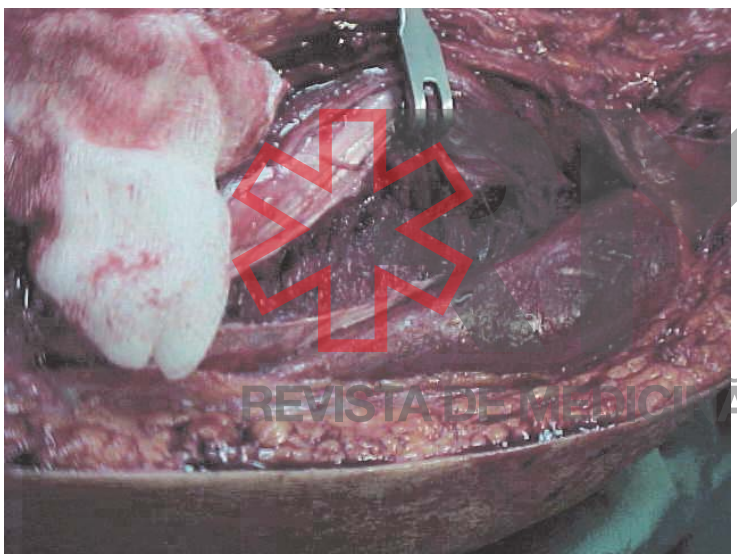

Figure 14 Case 6
Generalized muscular necrosis and capillary thrombosis – MSOF – lethal evolution

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