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Problems concerning diagnosis and treatment of compartment syndromes after lower limb trauma

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Rezumat

Probleme privind diagnosticul și tratamentul sindromului de compartiment în traumatismele membrului inferior

Sindromul de Compartiment este caracterizat printr-un dezechilibru generat de creșterea presiunii într-un spațiu inextensibil (numit compartiment), nefiind caracteristic numai ortopediei, este din ce în ce mai frecvent în traumatologia modernă. Fiziopatologia sa are caracter auto-agravant prin perturbările din microcirculație. La nivelul membrelor inferioare, tabloul clinic este patognomonic, fiind dominat de creșterea consistenței lojelor musculare, prezența pulsului periferic ne-excluzând Sindromul de Compartiment. Deși diagnosticul de certitudine se pune prin măsurarea presiunii intra-compartimentale, esențială este suspiciunea clinică. Tratamentul corect (în faza de stare) este chirurgical, și constă în fasciotomie decompresivă precoce și largă. În absența tratamentului corect, Sindromul de Compartiment pune în pericol nu numai vitalitatea segmentului respectiv, (prin apariția ischemiei periferice acute cu debut în microcirculație și extensie centripetă), dar și viața pacientului, putând deveni o leziune cauzatoare de deces. Autorii subliniază valoarea unei evaluări clinice corecte și a unui tratament corect precoce în prevenirea complicațiilor locale și generale severe ale acestui sindrom.

Cuvinte cheie: compartiment inextensibil, presiune intra-compartimentală, strivire, microcirculație, colaps capilar, fasciotomie

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Abstract

Compartment Syndrome (CS) is characterised by an imbalance produced by increased pressure in an inextensible space (called "the Compartment"). Without being specific for orthopaedics, CS has increasing frequency in modern traumatology. Microcirculation disturbances generate the syndrome's self-augmenting physiopathological character. The pathognomonic feature of the clinical panel in CS of the lower limbs is increased consistency of the muscular groups, while peripheral pulse maintainance does not exclude CS. Although positive diagnosis is based on measuring the intra-compartmental pressure, (ICP) clinical suspicion is crucial. The correct treatment is surgical, consisting in early and large decompressive fasciotomy. Without proper treatment, CS endangers not only the vitality of the limb (due to Acute Peripheral Ischemia with onset in microcirculation and centripetal extension), but also the patient's life, thus becoming a life-threatening disorder. The authors underline the importance of correct clinical evaluation and early treatment in order to prevent the serious local and general complications of the CS.

Key words: inextensible compartment, intra-compartmental pressure, crushing, microcirculation, capillary collapse, fasciotomy

Introduction

Modern traumatology is characterized by high energy traumatic agents, affecting younger patients, producing more severe injuries, involving not only the limbs, but threatening the life of these patients. This article refer to one clinical situation with special characteristics - diagnosis and treatment of the acute compartment syndrome (CS) of

the lower limb.

The Compartment Syndrome represents the anatomo-pathological and clinical aspects caused by increased pressure within a limited, enclosed space surrounded by an inextensible structure, generically named as *"compartment"*. (1,2)

Defined as *"the increase of the intra-compartmental pressure"*, the Compartment Syndrome (CS) represents the expression of the inbalance between: the container, which is inextensible –and- its content, which trends to extend, to increase its volume. This inbalance may be produced by two mechanisms:

1. by the decrease of the capacity of the container, or
2. by the increased volume of the content, in an inextensible area. (3)

It should be underlined that CS appears not only in the limbs, but in any inextensible space within the human body, but the common feature, no matter the location of the CS is increased ICP.

The calf has the following inextensible structures: tibia, peroneum, interosseous membrane, and the fascia (with several components: anterior, external, superficial posterior and deep posterior), limiting the following compartments:

- anterior - containing: muscles responsible for the dorsal flexion of leg and toes (tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus tertius) and the anterior tibial artery and vein together with the deep peroneal nerve;
- lateral - containing muscles: peroneus longus, peroneus brevis, peroneus communis and peroneal common nerve which splits into superficial peroneal nerve and tibial anterior nerve that passes through to the previous compartment;
- superficial posterior containing gemellus and soleus muscle and
- deep posterior containing: muscles (flexor hallucis longus, popliteus, tibialis posterior and the tibio-peroneal posterior artery, posterior tibial veins, posterior tibial nerve. *(Fig. 1)*

This description is important because the functional impairment (which of the movements of the ankle, foot and toes are limited) depends on the muscles affected by increased ICP.

As shown before, there are two mechanisms responsible for increased ICP:

1. decrease of the capacity of the container can be produced by: constrictive fascial scars, after severe trauma, with extensive crushing, external iatrogenic compressions- compressive casts or bandages, or prolonged tractions for reductional purposes or for post-operative immobilization that induce tension in the compartmental fascias. (4)
2. increase of the contents volume is caused by the excess of:
   A. blood, due to a located haematoma or to a diffuse bleeding;
   B. interstitial liquid (edema) or,
   C. both of them, like after fractures

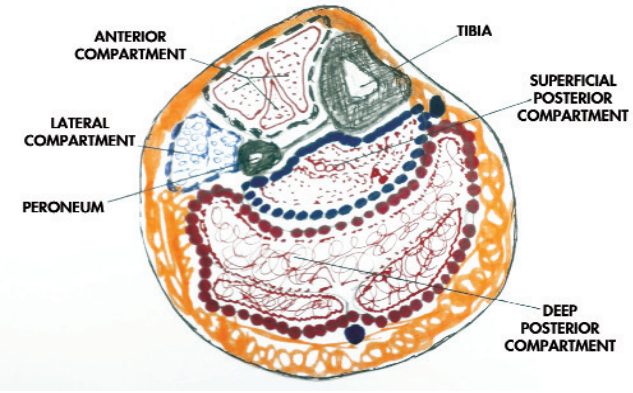


Figure 1. *Cross section through the leg*

A. Intra-compartmental bleeding can occur due to: major vascular injuries, fractures, surgery (reaming, osteotomies) and crushes (with extensive muscular injuries). Fractures with or without vascular injuries may produce CS due to the bleeding in the fracture site; the risk to develop CS is higher when the bleeding is arterial and the interosseus membrane is intact. Proximal tibial fractures - epiphyseal or epi-metaphyseal fractures, since they are located in well vascularized areas, are frequently complicated by CS. (5,6,7)

B. Interstitial edema due to increased capillary permeability can be: post-traumatic, post-ischemic, including the haemostatic band (8), post-reperfussion – if the arterial restoration is late/ not followed by fasciotomy and the tissular reperfusion syndrome occurs. (9,10)

No matter the clinical form, etiology and location, the CS' physiopathology is the same, with two major components:

1. pathologic phenomena start at micro-circulation level, being initiated by the increased Intra-Compartmental Pressure (ICP) which overrides the hydrostatic pressure in the capillaries (which is usually 30 mmHg) (11,12), and thus leading to the impairment of the tissue irrigation up to the level where the oxygen's transfer to the cells is not possible anymore, even if the tissue perfusion pressure increases in order to compensate the ICP. If ICP increases, the compensatory mechanisms tend to increase the venous pressure, and when venous pressure exceeds CPP, capillary collapse occurs. (13)
   - The value where this occurs has not been certainly established yet, but is certainthat for an ICP of 30 mmHg, decompression surgery is required since, for this value, the blood flow in the capillaries disappear, the cells are not fed with oxygen anymore, and all phenomena subsequent to the cellular anaerobiosis break out. (14) There are two main consequences of capillary collapse: the irreversible ischemia, (which is therapeutically overrun, since it evolves centripetal while the periphery is compromised, and the muscles destroyed) and the extensive capillary thrombosis. (15)
2. through a self-maintaining chain, in the absence of an efficient therapy, the organism is "poisoned" with products resulted from the muscular ischemic necrosis,

especially myoglobine, thus leading to MSOF (Multiple System and Organ Failure), which ENDANGERS THE PATIENT'S LIFE. (16,17)

Diagnosis and Treatment

Compartment Syndrome has several phases:

1. debut phase (alarm phase);
2. status phase, and afterwards there are two possibilities, namely;
3. effective treatement together with healing resources converge to the healing phase, or
4. therapeutic resources are overwhelmed and one of the CS complications occurs, such as:
   - local complications without Acute Peripheral Ischemia (API);
   - local complications with API without MSOF;
   - general complications - API with MSOF.

Alarm phase

The alarm phase is characterized by - pain - there are two types of pain to be evaluated:

1. spontaneous pain, sometimes in disproportion with the amplitude of the trauma, is like a continuous deep tension, sometimes pulsating, cannot be correlated with the territories of sensitive innervation, does not completely disappear after analgesics, not even after opiates, and not even after proclivity. Most of the authors consider that the spontaneous pain occurs when ICP levels reach 30 mmHg. Special attention must be paid when the patient does not perceive the pain (severe neurological disorders with sensitive and motor dissociation, polyneuritis, other sensitivity disorders, post-spinal anesthesia) or he/she cannot express it (alteration of consciousness).
2. passively induced pain (stretching test), augmented by the passive elongation of the affected muscles (for example, if CS is located in the anterior compartment, the pain is augmented by the plantar flexion). This type of pain, although it represents a significant debut sign, is unspecific, as it can also appear after any trauma which generates muscular injuries. It should be highlighted that the stretching test is an indicator of the evolution towards muscular retraction.
   - increase of the perimeter (circumference) of the calf, compared to the contra lateral one;
   - minor/medium *increased consistency of the muscles* (the most specific sign for CS, generated by the increased ICP);
   - no neurological impairment;
   - *pulse present* distally, IF there is no arterial injury of the major axis associated with CS.(18)

Whenever these symptoms appear, the presumptive diagnosis of "Compartment Syndrome, alarm phase" will be "attached" as a complication of initial trauma . Differential diagnosis should be made with:

- oedema which normally appears after any trauma;
- deep vein thrombosis - the pain is in the calf, deep, aggravated by passive dorsiflexion, but also at the compression of the calf; the diagnosis is established after Doppler ultrasound examination;
- other causes which increase the volume of a segment of the limb: infections (where the infectious syndrome also appears), tumors (where the deformation precedes the trauma), chronic venous insufficiency (in this case, the oedema is influenced by proclivity, and the echo-Doppler investigation makes the differential diagnosis).

The positive diagnosis is established by measuring the ICP with different types of manometers (modern manometers are digital - *Fig. 2*): when the ICP value is HIGHER than normal (20 mm Hg) but UNDER 30 mmHg, we can be sure about the alarm phase of CS.

Treatment in the alarm phase, has two major components:

1. treatment of the skeletal injury (fracture, dislocation, sprain, contusion) – stabilization of the fracture, reduction of the dislocation, cast for sprains, etc;
2. treatment of the CS. The main idea is that the ICP has to be monitored throughout the treatment in order to be shore that the CS remains in the alarm phase, and does not evolve. Local NSAID, cast and elevation of the injured limb are useful in order to decrease oedema; systemic NSAID, anti-thrombotic therapy (preferably with LMWH), hydration and non-specific drugs (platelet or vaso-active) can be used, under strict control of ICP. (19)

If the treatment of the skeletal injury is surgical and the alarm phase of CS is diagnosed prior to surgery, ICP should be monitored during surgery: if ICP remains UNDER 30 mm Hg, surgical treatment of the skeletal injury is performed as planned, followed by medical treatment mentioned above; if ICP increases OVER 30 mmHg (during or at the end of surgery), fasciotomy will be performed, since CS has evolved from the alarm phase to the status phase (which will be discussed in the following).



Figure 2. *Stryker manometer used in our Clinic*

Status phase

The *clinical panel* of the post-traumatic CS, acute form, in status phase, comprises the following elements:

- pain in the affected muscular groups, with the above-mentioned characteristics, will quickly extend distally from the place of the initial injury, to the toes, where it becomes intense;
- the volume of the shank increases significantly, compared to the healthy one, with oedema of the foot and ankle, so measuring the perimeter in the medium third of the shank, bilaterally will show the difference;
- *increased consistency of the muscular groups*, which is *pathognomonic* for CS; in severe cases, the consistency has an "wooden" aspect;
- the skin is glossy, under tension, with obvious venous design. *(Fig. 3, 4)*
- neurological impairment: sensitive and motor The sensitive disturbances are paresthesia, followed by hypoesthesia, localized at first *distally*, at the toes' level, and which subsequently expands centripetally. Sensitive disturbance is the first sign of ischemia of the peripheral nerves, meaning that the ICP exceeds 30 mm Hg (which is considered to be the "border" of peripheral nerve resistance to ischemia). Motor impairment consists of the impossibility to perform actively the type of movement normally gernerated by the contraction of the muscles in the affected area (for example: for the anterior compartment, the affected movements are dorsiflexion of the foot and toes, etc);
- *pulse* is *present* distally up to the onset of irreversible API. In most of the situations, oedema of the ankle and foot makes the evaluation of peripheral pulse to be difficult, but Doppler ultrasonography clarifies the problem. It is very important to underline the fact that *the presence of the peripheral pulse does not exclude the diagnosis of compartment syndrom*, but it shows that the major arterial axes were not yet compromised by capillary thrombosis extended centripetally

Positive diagnosis is based upon measuring of ICP; there are various opinions with regard to the value of ICP value that should be considered as significant for CS, namely:

1. some authors believe that ICP should be higher than 30 mmHg for positive diagnosis of CS, but it should not exceed 45 mmHg for diagnostic purposes,
2. there is also the opinion according to which the absolute value of ICP is not significant, but the differential pressure (DP); DP represents the difference between the diastolic pressure and ICP. According to some authors DP should be of LESS then 20 mmHg in order to diagnose CS in the status phase. In other words, in order to allow a normal circulation within both the capillary territory and microcirculation, ICP should not "get closer" to the diastolic pressure by less than 20 mmHg, otherwise the functionality of the microcirculation will be severly altered and the metabolic exchanges will collapse.

In this phase, the treatment has to achieve the rapid decrease of the severely augmented ICP which will otherwise produce irreversible changes; that means that, besides the treatment of the skeletal injury, we have to rapidly re-establish the balance by: eliminating the cause which tends to increase the volume of the content and by increasing the volume of the container and "eliminating" the inextensible structure, which is the fascia.

The incision of the fascia is called *fasciotomy*, it can be performed on one or two incisions, in order to fully open the affected compartments. The mandatory conditions for fasciotomy are: (20).

- to be long enough, starting from the head of the peroneum to the external maleolus (external) and from the internal maleolus to the internal articular space (internal) *(Fig. 5, Fig. 6)* because if does not completely decompress a muscle, the part of the muscle which is decompressed simply herniates and becomes ischemic after some time *(Fig. 7, 8)*;
- to open all the muscular spaces;
- to perform evacuations of hematoma, lavage, surgical debridement;
- to determine release of the muscular ICP, otherwise fasciotomy is uneffective;
- to be covered only after complete release of the



Figure 3. *Compartment syndrome (alarm phase)*



Figure 4. *Compartment syndrome- status phase*



Figure 5. *Correct fasciotomy - extensive hematoma*



Figure 6. *Correct fasciotomy*



Figure 7. *Incorrect fasciotomy, with necrosis of anterior and posterior muscles*



Figure 8. *Incorrect fasciotomy, with necrosis of anterior and posterior muscles*

symptoms, after some days of treatment, overwise the process will be reiterated; so, secondary closure (by suture or skin grafts - *Fig.9, 10)* - is recommended, and not the primary one.

It ie extremely important to underline that *clinical suspicion* is the major point in CS, since in the absence of the manometer, objective ICP evaluation is imposible. In these situations, *under no circumstances* should fasciotomy be delayed when there is *the slightest suspicion* of Compartment Syndrome, regardless of whether we can measure the ICP or not.

Phase of local complication without acute peripheral ischemia

a. Volkmann ischemia retraction. When CS involves one or more muscles which become ischemic, without API of the limb, ischemia transforms the muscles in fibrotic structures, which are inextensible, so it looks like the muscle is shortened, retracted. Most frequently, this evolving form of CS occurs to the posterior logia and results in a vicious equine position, pursuant to the cicatricial fibrosis of the sural triceps.



Figure 9. *Secondary cover- skin graft*

b. paralysis of the popliteal external sciatic (peroneal) nerve can complicate the CS when the decompression is late, so the peripheral nerves have already been affected by ischemia; in other words - when the time of ICP exceeding 30 mmHg is large enough to allow the



Figure 10. *Secondary suture*

irreversible neurological injuries to settle down, but without producing the necrosis of calf's entire muscular structure. (21)

4. Acute peripheral ischemia

Acute peripheral ischemia can complicate the CS, as it was shown. When the impact of the ischemia is systemic, MSOF develops, and it represents the most severe evolution stage of CS. It is determined by the body's "poisoning" with toxic products resulted from the anaerobic muscular metabolism.

In this phase, the treatment is meant to support the vital functions and to compensate the severe unbalances induced by MSOF, and is of competence of Intensive Care. Unfortunately, amputation above the knee is indicated in these cases as a life-saving procedure

Conclusions

Even if the main phenomena take place in the inferior limbs, usually in the calf, Compartment Syndrome is a post-traumatic disorder which can affect the whole body. Due to the increased Intra-Compartmental Pressure (ICP), irreversible damage can occur if treatment does not perform decompression early enough, affecting the muscles, the nerves and the blood vessels. Once presumptive diagnosis is established, fasciotomy should be performed if ICP cannot be monitored. If it can be, ICP pressure over 30 mm Hg is a clear indication of fasciotomy. Correct and early fasciotomy is the guarantee of the favorable outcome of the patient. (22). Once irreversible thrombosis appeared in microcirculation, CS becomes a *life-threatening disorder*, since the pathological process is self-maintaing and leads to irreversible ischemia, with all its' local and general consequences.

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