

Review Article

Threatening Inferior Limb Ischemia: When to Consider Fasciotomy and What Principles to Apply?

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Inferior limb compartmental syndrome (CS) gathers a constellation of symptoms that traditionally refers to pathologically increased intramuscular and surrounding tissue pressure generally contained in nonexpansile leg spaces. It associates oftentimes reperfusion or traumatic injury. Intrinsic rigidity of these leg and foot closed compartments may enhance critical pressure risings with deleterious effects on specific vascular and nervous supply, with two main presentations: *acute* versus *chronic* display. For these situations, early fasciotomy plays the major role of releasing specific compartment hypertension and prevents deleterious tissue necrosis. Intervention is effective only if required upon precise indications (measured tissue pressure within 20 to 30 mm Hg of systemic diastolic pressure) and performed correctly in a timely fashioned approach. Any failure or delay in recognizing CS inevitably leads to adverse outcomes and jeopardy for secondary limb loss. When judiciously applied during or soon after limb-salvage revascularization technically successful fasciotomy may represent a major contributor in limb preservation. It accounts for a well-defined therapeutic proceeding available for any conscientious and well-briefed interventionist.

1. Introduction

The compartmental syndrome (CS) is a morbid entity that can arise in any area of the body that has little or no capacity for tissue expansion, such as the lower and upper limbs, the abdomen, and some described cervical regions [1, 2].

The vascular practitioner becomes increasingly confronted with revascularization side effects [3] since new techniques and contemporary devices venture more extreme and challenging peripheral ischemic situations. Among the whole evoked CS etiologies [1–3], the revascularization subgroup represents a frequent and possibly devastating pathology, requiring urgent and appropriate therapeutic measures [2, 3]. It is generally accepted that one of the most common locations of all peripheral CS manifestations is represented by the lower leg [1–3]; this arises upon relative nondistensibility of local bones and surrounding dense connective septa and tissues, facing reactive “postischemic” vasodilatation and swelling [1–4]. Intrinsic rigidity of these leg and foot closed

compartments in specific anatomical locations may enhance critical pressure risings with deleterious effects on the local vascular and nervous supply. Eventual failure or delay in recognizing CS inevitably leads to adverse outcomes for concerned patients and hinders early prognosis for limb preservation [1, 2].

Urgent fasciotomy undoubtedly represents in these situations the definitive treatment to address CS and avoid secondary ischemic destruction [1–6]. Promptness and accuracy in pressure releasing of *all compartments* of the affected leg seem to equate with key values of the advised interventionist [2, 5, 6].

2. The Compartmental Syndrome of the Inferior Limb: Etiology and Diagnosis

2.1. Anatomical Features. Previous works in this field evinced four distinct anatomical compartments in *the leg* and at

least eight in *the foot* as to be eventually considered for decompression [1, 2, 5–7]; although clearly stated for the leg, there is no current consensus regarding the exact number of compartments to be regularly treated in the foot [2, 3, 5–7].

From a practical perspective, the main anatomical structures [2, 7] with meaningful clinical implications in *the leg* and *foot*, (Figures 1 and 2) are summarized [1, 2, 7, 8] in Tables 1 and 2.

3. Pathophysiological Concerns

Blood perfusion is attainable within specific anatomical compartments *only* since diastolic pressure exceeds the intracompartmental given pressure [1, 2, 6, 8].

It has been shown that any important and sustained increase in tissue pressure contained by inelastic conjunctive structures may enhance CS in two possible presentations: *acute* versus *chronic* display [1, 2, 6]. Reperfusion CS mainly relies on *acute* pathophysiological layouts and hence represents the main focus of this clinical review.

Compartment *pressure elevation* was already emphasized by previous studies as to play alone a central mechanism in CS etiology [1–3, 6, 8].

Contemporary clinical evidence more accurately suggests that either abruptly increasing the volume (alike local edema) or decreasing the compartment's amplitude (e.g., following external compression) both may enhance notable raises of the intracompartmental pressure [2, 7].

Several morbid conditions were enounced as to eventually generate more often acute versus chronic CS, such as tissue swelling, local hematomas or pus collections, revascularization syndromes, burns, casts, repair of muscle hernias, vicious positions, circumferential dressings, exercise-related pain in young athletic patients, or even local venous insults (DVT) [1–6]. However, the precise pathophysiological mechanism of each CS (or types of CS) dwells unclear [2, 6].

Following congruent clinical observation, the most affected inferior limb compartment (including postischemic injury) cited in a majority of published works appears to be *the anterior leg compartment (AC)* [1, 2, 8].

In the same setting, one of the most cited etiologies generating the *acute* CS refers to the “ischemia-reperfusion mechanism” [1, 5] that triggers soft tissue swelling and maladaptive responses in confined leg and foot anatomical spaces.

Local Tissue Changes. Among all anatomical structures of the extremity compartments, nerve and skeletal muscles are the most exposed to ischemic aggression.

Viewed *from a cellular and molecular perspective*, local tissue ischemia enhances release of inflammatory *cytokines* responsible for vasodilatation, increased capillary permeability, and local edema [2, 6]. The common presence of systemic inflammatory response (although inescapable following initial surgical interventions) adds *leukocyte migration*, activation, and adhesion to the vascular endothelium and together with local *platelet* triggering seems to play a major role in initiating ischemia-reperfusion injury [6, 8]. Reactive *oxygen*

radicals were also described to produce DNA damage, being activated by local hypoxia and cell lysis [1, 6].

Beyond two hours of escalating muscle ischemia and after restoration of arterial flow, a “rapidly expanding” edema appears that inevitably leads to threatening local compressive effects in noncompliant osteofascial sectors [1, 2, 6–8].

According to relaxed *time and severity* of ischemia, serial *precocious* versus *retarded* CS enhanced tissue changes were described [1, 2, 4, 6].

(1) *Immediate ischemic effects*, generate three *specific zones* of tissue hypoperfusion:

- (a) a *proximal area* with no permanent damage and reversible affectation,
- (b) a *distal area* with very likely irreversible tissue necrosis, and
- (c) various amounts of “stunned tissues” potentially at risk for irremediable decay but still holding regenerative resources [2, 4].

The real extent and evolution of these staggered areas depend on the precocity and accuracy of initial CS diagnostic [2–4, 6–8]. Since *intracompartmental pressure equals the capillary pressure*, nutrient blood flow is reduced to zero and all these “borderline” territories are losing vital potentialities [1–7].

Venular pressure (normal values about 3–7 mm Hg) equally rises because of the venous outflow progressive blockage [1, 2, 6]; a vicious circle is consequently casted gathering relentlessly increasing capillary pressure, exacerbating fluid transudation, cell swelling, and impossible arteriolar and venular flow adaptation amidst the rigid and fibrous compartmental walls [2, 6–8].

Unless compartmental hyper pressure is relieved, cellular perfusion is abolished and tissue infarction enters the pathological cascade [2, 8].

(2) *Late ischemic consequences* were also cited. If untreated, CS may originate rhabdomyolysis with the release of multiple subsequent metabolic toxins [5]. Adjacent “myoglobinuric renal failure” was described, with high associated mortality rate [1, 5]. Other complications alike local infection or neurologic deprivation may contribute to retarded clinical recognition, adding auxiliary morbidity [1–5], and devastating limb injuries [1–3, 8].

(3) *The Iatrogenic CS*: regardless the time for presentation, *iatrogenic* CS may follow either transradial puncture for coronarography or PTCA [2], or more recently claim the transpedal accesses technique for retrograde tibial endovascular recanalization (personal team unpublished data) and should be equally mentioned.

Two main theories are postulated and still prevail as current pathophysiological hypotheses in reperfusion CS [1, 2].

- (a) The “*diminished arterial-venous gradient*” theory, due to secondary raises in compartmental and intraluminal venous pressures: these features may trigger both decrease in oxygenated blood delivery and consequent decline in drainage of deoxygenated venous blood [2].

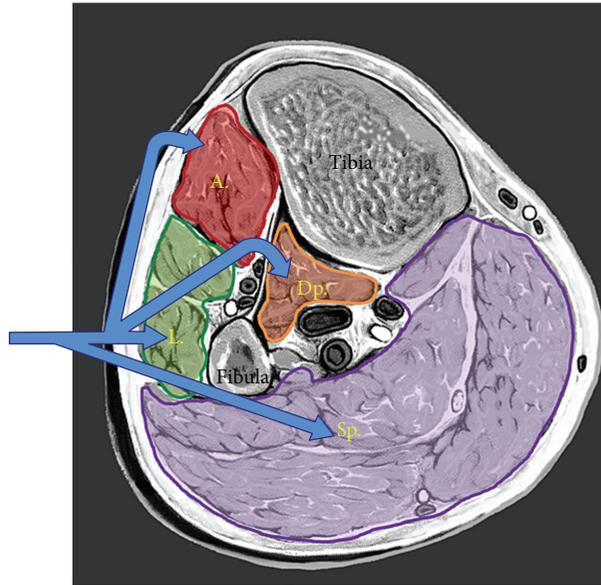


FIGURE 1: Leg fasciotomy: the “lateral approach” illustrated in a sagittal section of the lower leg. Throughout one longitudinal incision, the access is allowed in A = the anterior compartment. L = the lateral compartment. Dp = the deep posterior compartment. Sp = the superficial posterior compartment.

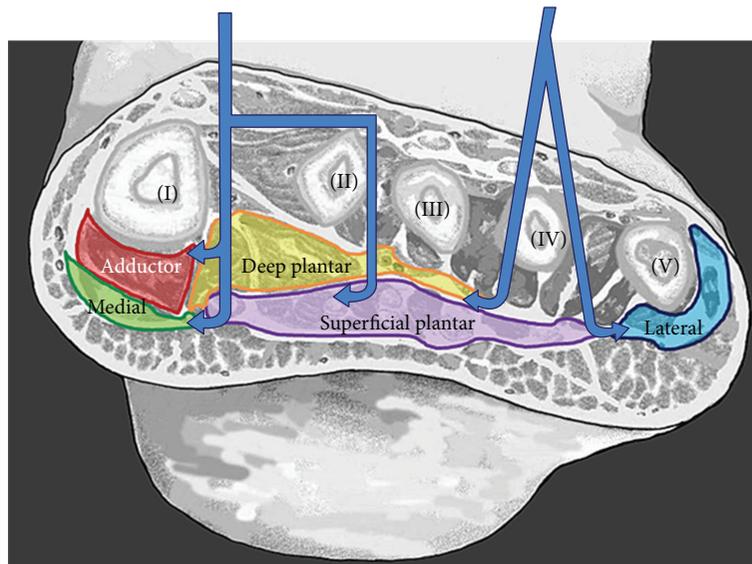


FIGURE 2: Foot fasciotomy: the “dorsal approach” using one or two incisions on the dorsum of the foot. It eases the access to the superficial plantar, the interosseus, the medial, the lateral, the deep plantar, and adductor foot’s compartments.

(b) The “critical closing pressure” hypothesis; this concept suggests that, because of small arteriolar flow passage (high amount of tissue tension and stiffened arteriolar walls), higher arteriolar perfusion is required to maintain tissue viability [2]. A progressively drop in the arteriolar-tissue gradient is further noted that inexorably leads to local arteriolar collapse, finally leading to tissue necrosis [2].

Interestingly, both mentioned hypotheses may in fact complete each other and reflect different patterns of CS deployment for peculiar local ischemic tissue affectations [1, 2, 9, 10].

Specifically concerning the acute hypoxic foot features, it could be stipulated that targeted arteriolar ischemic distress (specific forefoot, plantar, dorsal, or heel compartmental hyper pressure) may enhance predictable topographical decrease in the arteriolar-tissue gradient in these territories, since the collateral “rescue” flow is either severely deprived or abolished [2, 10].

Whatever the accurate pathogenic mechanism, it is now generally accepted that prompt surgical decompression and aggressive surveillance of these high-risk limbs remain the mainstay values in dedicated CS therapy [1-9].

TABLE 1

Anterior compartment (A) of the leg	Lateral compartment (L) of the leg	Superficial posterior compartment (SP) of the leg	Deep posterior compartment (DP) of the leg
Anterior tibial artery Tibialis anterior m. Extensor digiti longus m. Peroneus tertius m. Extensor hallucis longus m. Deep peroneal n.	Peroneus longus m. Peroneus brevis m. Common peroneal n. Superficial peroneal n.	Gastrocnemius m. Soleus m. Plantaris m.	Posterior tibial artery Peroneal artery Popliteus m. Flexor hallucis longus m. Flexor digitorum longus m. Tibialis posterior m. Tibial n.
Superficial veins		Superficial veins	Deep veins

The bold font specifies particularly the main arterial axes in the leg (the most important anatomic structures implicated in genuine ischemic pathology of CS).

TABLE 2

Medial compartment of the foot	Lateral compartment of the foot	Superficial + deep compartments of the foot	Interosseus compartment (×4) in the foot	Adductor compartment of the foot	Calcaneal compartment in the foot
(i) Flexor hallucis brevis (ii) Abductor hallucis	(i) Abductor digiti quinti (ii) Flexor digiti minimi	(i) Flexor digitorum brevis (ii) Flexor digitorum longus (iii) Lumbricals	Interossei muscles	Adductor muscle	Quadratus plantae
Superficial veins	Superficial veins	Superficial veins	Superficial veins	Superficial veins	(i) Posterior tibial artery + vein (ii) Lateral plantar artery + vein
Superficial nerves	Superficial nerves	Superficial nerves	Superficial nerves	Superficial nerves	(i) Posterior tibial nerve (ii) Medial + lateral plantar nerves

The bold font specifies particularly the main arterial axes in the leg (the most important anatomic structures implicated in genuine ischemic pathology of CS).

4. Clinical Presentation

4.1. *Clinical Symptoms.* The initial diagnostic of CS essentially relies on meticulous and repetitive *clinical observation* of the ischemic leg [1–3, 8].

It represents a timely fashion judgment where personal expertise and awareness of the leading interventionist complete repetitive team evaluation of the revascularized leg [6, 8].

Frequent examination is mandatory in the acute phase if CS is suspected [2, 6]. Undoubtedly, the most common and precocious symptoms (except previously neuropathic limbs) gather [1–7].

(a) Severe *pain*, that is currently disproportionate from main clinical appearances, almost always worsens progressively despite appropriate medication.

(b) Distal motor and sensory *neurological dysfunction* (numbness and weakness of specific nerves passing through the affected compartment) is frequently described. *Paresthesia* invariably demonstrates early nerve ischemia, which progresses to complete *anesthesia* without prompt intervention; it is commonly associated with the following:

(c) *Swelling* and *tenderness* are perceived at routine tissue palpation.

Because CS always occurs in specific tissue sectors where local elevated pressure remains however *below the systolic* blood pressure.

(d) *Distal pulses are commonly unaffected.* This represents an important feature that may misguide the inexperienced clinician that might get trapped by apparently “regular” peripheral arterial perfusion (palpable pulses, comforting ABI, warm extremity, reassuring TcPO₂, etc.) [1, 2, 6, 7].

As stated by recent works, irreversible muscle necrosis may occur starting with the 3rd hour that follows the onset of ischemia [2, 6, 7].

Any delayed action in correct and expeditious fascial decompression may further jeopardize the affected limb, still at high risk for major tissue loss [2, 6, 7].

4.2. *Complementary Laboratory Assessment.* It appears unfortunately common to initially discount the CS diagnostic, by exclusively following the clinical symptoms [2, 6].

The regular physiopathology of the “acute” CS undoubtedly relies on three main triggering factors, to be commonly assessed in all presentations [1, 2, 4, 7]:

- (1) increased intercellular tissue pressure,
- (2) critical decay of venous outflow, and
- (3) diminished neural conduction in the affected territory.

Taking into account these changes, the following adjuvant diagnostic methods were proposed [2, 7].

(a) Measurements of the local tissue pressure in the affected leg compartment represent a cardinal point to assess CS. An important variable was found to express *the gradient* between diastolic blood pressure and compartments intrinsic tension [1–3, 6, 7]. Fasciotomy is recommended if tissue pressure (normally <10 mm Hg) raises within 20 mm Hg to 30 mm Hg of diastolic blood pressure [6]. Current indication for fasciotomy then stipulates the necessity to perform global compartmental decompression of the affected leg or foot, *if the differential pressure between compartment pressure and diastolic pressure is less than 30 mm Hg* [1, 2, 6].

In the daily practice, according to an initiatory CS context, tissues pressures are commonly elevated up to 40–60 mm Hg, or <30 mm Hg below diastolic blood pressure [1–3, 6].

Some authors stipulated in the early 70–80s that any tissue measurements greater than 30 mm Hg for more than 3–4 hours may represent valid indication for fasciotomy [1–3, 8].

It is nowadays more clearly established that isolated appraisal of compartmental pressure without diastolic blood pressure reference is neither sensitive nor specific [1, 6, 8].

(b) Other noninvasive imaging techniques were equally cited in the assessment of CS, such as the “near-infrared spectroscopy” or the “laser Doppler flowmetry,” with possible promising applications in the future [1, 2]. Their utility is at the present time under clinical investigation [2, 7].

(c) Venous Duplex scan of the affected leg is another practical and easy way to perform investigation as to ascertain notable slowing-down (or cessation) of regular venous outflow in specific compartmental veins [2, 6]. The presence of normal respiratory phasicity on tibial venous flow may then rule out eventual elevated tissue pressures in the concerned compartment [2, 6].

(d) Although more invasive and somehow cumbersome [6], the somatosensory evoked potentials method [2, 6, 7] was described in some studies to provide accurate information (however owing a certain delay) in CS clinical recognition [9].

Other complementary methods, such as the MRI, the Thallium-201 stress scan testing, and some biomarkers scoring (alike the white cell count, creatine kinase, or myoglobin levels), were equally described to eventually support the CS diagnostic, yet with debatable practical utility [2, 6].

4.3. Chronic Compartment Syndrome. While most of clinical presentations rely on “acute” onsets and outcomes of CS, another form of “chronic” CS was equally described, generally in young and otherwise healthy individuals having more often highly developed and bulky muscular mass (but also in normal genotypes) [2, 3, 6, 7, 11]. The clinician should be aware of this rare and however more insidious presentation, revealed by discrete swelling and local tenderness over the affected compartment, adding common loss of phasic Doppler tibial venous flow during exercise [2, 6, 11]. Although with prevailing physiotherapeutic benefits owing appropriate differential diagnosis [2, 6, 7, 11], in some embarrassing settings for current professional activities, the use of small

and esthetically acceptable skin incisions for endoscopic fasciotomies (mostly concerning the anterior compartment of the lower leg) may be individually considered.

5. Principles of Treatment

Animal studies and retrospective human data indicate that duration of elevated pressure condition dictates the outcome in CS [3, 7]. Early decompression of reperfused tissue previously affected by severe ischemia represents a cardinal priority for the vascular interventionist [6–9]. It is unanimously accepted that the sole method as to release compartment hyperpressure and prevent tissue necrosis is represented by expeditious fasciotomy [1–9, 11–13].

5.1. Open Surgical Fasciotomy

Technique. Fasciotomy (incision of the underlying fascias) is indicated when intracompartmental pressure rises within 20 to 30 mm Hg of systemic diastolic pressure [2, 6]. Pressures in CS typically exhibit 40 to 60 mm Hg, or <30 mm Hg below diastolic blood pressure [1, 3, 6, 11].

To be considered as “technically successful,” current “open” surgical fasciotomy should include the following requirements [6, 11].

- (a) complete opening of the skin overlying the affected compartments (Figures 1 and 2),
- (b) longitudinal incision of the entire fascia investing each of the described compartments (Figure 3),
- (c) careful postoperative wound care, followed by complete tissue coverage since swelling subsides.

The three described *thigh* compartments, adding the other four located in *the leg* and around nine *foot* musculotendinous compartments [2, 11], can be approached for fasciotomies either through a *single* or by *multiple* incisions [2, 6, 7, 11].

5.1.1. Thigh Fasciotomy. Although rarely described in dedicated clinical experience (Figure 3(a)), the thigh may be only seldom concerned by “isolated” CS and more often by global inferior limb compartmental injuries [1, 2, 6, 8]. There were described three main compartments in the thigh: the anterior (the quadriceps muscle), the posterior (hamstrings), and the medial (adductors) compartments. Fasciotomy could be performed either through a single lateral incision that allows adequate decompression of all three thigh sectors (Figure 3(a)) or by separate approaches [12].

5.1.2. Lower Leg Fasciotomy. The lower leg represents the most cited location for fascial decompression (Figures 1 and 3(b), 3(c), and 3(d)):

(1) The “*single incision*” technique (Figure 1): the leg is disinfected and draped circumferentially following the standard fashion. A *lateral incision* is performed overlying the upper fibula (Figures 1 and 3(d)). This cut-down should be prolonged downstream to the lateral malleolus; it should



FIGURE 3: *Inferior limb*. A succinct illustration of the main recommended incisions: (a) the lateral aspect of the thigh, (b), (c), and (d) the medial and lateral approaches for leg decompression, and (e) the dorsal access for foot fasciotomies.

involve the skin, the subcutaneous tissue up to the fascia encasing the muscles [2, 7].

- (a) Exposure and incision of the fascia encasing laterally the peroneal muscles are mandatory; this will decompresses *the lateral* compartment of the ischemic leg. Identification of the lesser saphenous vein or its variants may avoid hazardous bleeding. The interventionist should keep focused exclusively on the fascial plane [2, 6, 7, 11].
- (b) A short anterior skin flap is then gently created (without notable displacement of ischemic tissues); further identification of specific fascias that enclose the other compartments of the leg is substantial. Incision of the anterior fascia longitudinally as to decompress *the anterior compartment* (Figure 1) until the distal third of the leg can be performed next [2, 7]. A particular attention should be allowed not to injure the superficial peroneal nerve lying between the anterior and lateral compartments of the leg; this was advised in urgent circumstances featuring swollen and more difficult to recognize structures [6–8].

- (c) Via the same lateral incision, a posterior skin flap should be then created as to approach *the superficial posterior* leg compartment (Figure 1). The adjacent superficial fascia enclosing the posterior compartment should be carefully incised over 10 to 25 centimeters as to access the soleus and the gastrocnemius muscles [2, 7]. After posterior retraction of these two muscles, the *deep posterior* compartment could be equally addressed (Figure 1); the underlying deep fascia should be then meticulously sectioned, avoiding eventual damage either of the common peroneal nerve or the neighboring posterior tibial neuro-vascular bundle, sometimes strongly attached to the profound fascial structures [2, 6, 7].

It is important for the interventionist to identify severely ischemic from “still viable” muscle that has characteristic pink-red color, contracts when mechanically or electrically stimulated (electrocautery), and normally bleeds while checked. Muscular structures that do not react and show questionable viability should be better debrided [2, 6].

(2) The “double incision” technique (Figure 3(b)): the “double incision” technique allows decompression of all four leg compartments accessed by two skin approaches, without

creation of skin flaps [2, 7]. Although technically with greater “expeditiousness” and less tissue dissection, some authors characterize this approach as “more aggressive,” claiming wider surgical exposures [1, 6]. The leg is regularly disinfected and draped following the standard fashion.

- (a) A *lateral incision* is performed as exposed in the previous paragraph, owing similar access to *the lateral and anterior* leg compartments (Figure 3(d)). Both lateral and anterior fascias are delivered until the malleolar regions, bearing same anatomical precautions previously depicted in the text.
- (b) During the second stage of the procedure, a *medial incision* is undertaken along the posterior edge of the tibia (Figure 3(c)). The great saphenous vein should be routinely identified and carefully preserved, as well as its satellite saphenous nerve. This complementary medial access enables direct control to *the superficial and deep posterior* compartments and further warrants specific local fascial incisions [2, 7]. After cutting down the superficial fascia that encloses the gastrocnemius and soleus muscles, entrance to the deep posterior compartment is gained by dissecting the proximal tendons of these two easy-to-recognize muscular landmarks. The *deep posterior compartment* is then accessed and the underlying deep fascia attentively sectioned, seemingly avoiding injuries of the common peroneal nerve, also the nearby posterior tibial neurovascular bundle [2, 6, 7].

As mentioned before, muscle’s viability assessment is paramount and should be routinely evaluated in all compartments during the procedure [1, 2, 6, 7]. However, sole visual evaluation of skeletal muscle necrosis may be intriguing and exposed tissues should be reassessed every one or two days, until complete wound closure is achieved [1, 6, 7, 9].

5.1.3. Foot Fasciotomy. Following Ling and Kumar [13], several foot compartments were described and considered for eventual fascial decompression, according to parallel *ex vivo* observations (Figure 2):

(1) the posterior *half of the foot* is separated into *three fascial sectors* [13]:

- (a) the medial compartment that contains the abductor hallucis; it is surrounded medially by skin and subcutaneous fat and laterally by the medial septum;
- (b) the intermediate compartment that encloses the flexor digitorum brevis and more deeply the quadratus plantae; it is bordered by the medial septum medially, the intermediate septum laterally, and the main plantar aponeurosis on its plantar aspect;
- (c) the lateral compartment holding the abductor digiti minimi; it is surrounded medially by the intermediate septum, laterally by the lateral septum, and on its plantar aspect by the lateral band of the main plantar aponeurosis.

(2) No distinct myofascial compartments seem to be individualized *in the forefoot* [13].

Hind and Forefoot Fasciotomies. Matching the same study [13], “*single hind foot incision*” for posterior compartment’s fasciotomy through a *medial plantar approach*, associated (or not) to correspondent “single” or “bilateral” forefoot fasciotomies (Figure 2), would be sufficient to globally decompress the ischemic foot [13].

(I) The separate “*Medial Plantar*” approach (Figure 2): in cases of isolated calcaneal or plantar compartment syndromes with compression of medial and lateral plantar nerves and appended vessels [10], a single plantar incision (usually on the medial aspect of the foot) can be considered [11, 13]. Since lateral compartments are difficult to decompress using this way, it is however recommended to add a lateral incision to this approach [11]. The “*Medial Plantar*” way starts with a short cut-down following the plantar aspect of the 1st metatarsal. The medial compartment becomes visible and is split longitudinally. In order to reach the other compartments (towards the lateral aspect of the foot), the abductor hallucis should be gently retracted [11, 13].

Although without general consensus regarding the forefoot [2, 13], several surgical approaches were proposed (Figures 2 and 3(e)) and are succinctly depicted as follows [2, 11, 13].

(II) The “*Dorsal*” approach: one or two dorsal foot incisions are performed over the 2nd and the 4th metatarsals (Figures 2 and 3(e)), owing specific openings of the dorsal fascia of each interosseous compartment [11].

This approach also facilitates the deeper access throughout the medial, the lateral, the superficial, the deep plantar, and also the adductor foot’s sectors (Figure 2).

(III) The “*Lateral Plantar*” approach: it is less exerted in current clinical practice alone [11, 13], starts with an incision on the 5th metatarsal (Figures 2 and 3(e)), and continues to the median aspect of the plantar foot [11]. It is usually cumulated to either the dorsal or the medial incisions [11, 13].

(IV) The combined “*Lateral Foot and Ankle*” approach (Figures 2 and 3(b), 3(e)): it should be considered in specific circumstances, (especially in younger patients with firm skin and unaltered tissue regeneration capacities). A sole incision is performed in this case that begins at the lateral malleolus and extends to the forefoot between the 4th and 5th metatarsals allowing both dorsal and lateral plantar fascial decompression [11].

5.2. Endoscopic Leg and Foot Fasciotomy. Contemporary clinical expertise supports rising evidence about the feasibility and potential benefits of minimally invasive endoscopic fasciotomies in extremities affected by CS [14, 15].

Technique. a short skin incision is made toward the targeted musculotendinous compartment of the leg [14] or foot [15] (Figures 1 and 2) as to allow single [14] or double portal accesses [15]. An arthroscope is then introduced allowing visualization of the entire leg or the plantar fascia in distinct cases [14, 15]. A hook knife is interposed through the lateral opening of the slotted cannula and the targeted fascia is

divided by pulling the knife from remote points to the nearest fascial edge [14]. The blade should remain under direct vision at all times. Usually two or more passes are required as to achieve complete division of the fibrous layers [14, 15]. Meticulous hemostasis and accurate visualization of all vasculonervous structures (despite local swelling) are mandatory as to avoid iatrogenic damage [14–16]. The advantages of endoscopic, as compared to open compartmental release, include shorter operative time, quicker return to normal footwear, and less risk of postoperative wound complications [14–16]. Disadvantages rely on poor visualisation and accesses of remote vasculonervous structures, added to complementary skin tension in obese or severe inflammatory and ischemic presentations [14–16].

5.2.1. Possible Clinical Associations between Topographic Foot Revascularization (Angiosome-Oriented) and the Regional Display of CS. It has been suggested that, in specific cases, foot compartments may be subjects to focal ischemic injuries, especially in CLI patients with deprived collateral network (alike diabetics or end-stage renal patients) [2, 10, 17–19]. In these selected presentations, the CS may follow the topography of chronic inflammation and ischemic foot wounds [20, 21]. Since collateral compensation is abolished among different anatomical foot sectors, specific angiosome revascularization may trigger confined regional reperfusion syndromes (according the preexisting fibrous tracts) [10, 22, 23]. Among the whole of the cited foot compartments [2, 7, 13], the interosseous sectors seem to be the most affected zones by CS, after releasing critical ischemic foot injuries [10, 18, 20, 24].

Taking in account the dominance of *posterior tibial* (PT) arterial flow in the hind foot angiosomes [10, 20–24], the vascular interventionist should be aware of possible revascularization CS arising in this region. Specifically for these *three posterior compartments*, sole PT artery reopening in circumstances of severe local sepsis and collateral decay (e.g., for diabetic foot wounds) [21, 23] may enhance confined flexors tracts swelling [13, 24] requiring post- or perioperative (simultaneously to tissue debridement) decompression [10, 13, 18, 24].

Following a similar reasoning, for characteristic *anterior tibial* and single dorsal arch arterial reconstruction, the appended forefoot interosseous and fascial sheaths (although without specific anatomical marks) [13] may be targeted for peculiar “*single*” versus “*bilateral*” forefoot fasciotomies [2, 13, 24] (Figures 2 and 3(e)).

5.3. Local Wound Care and Closure. Complete closure of the fasciotomy site represents a critical step in maintaining a viable and functional limb [2, 6, 8, 16, 18]. The fasciotomy access also the revascularization-related complications rise impressively since wound closure is delayed [2, 6–8, 24].

Skin recovery should be stimulated by early debridement and punctual eradication of local infection [1, 18, 21]. Regular applications of sterile moist dressings adding specific antibiotic or physiotherapy are mandatory in most of the cases [1, 6, 20, 21]. The vascular interventionist should be aware that

precipitate skin closures may invariably enhance tegumentary necrosis or recurrent compartmental hyperpressure [6, 11]. Many of these wounds should require to be left open until secondary tissue recovery is obtained [1, 2, 11, 21].

It has been proved that definitive closure is only achievable after whole subsidence of local swelling [2, 18, 21]. According to every individual presentation, it is also recommended using temporary mesh, vacuum-assisted closure systems, or skin grafts, in order to obtain secure healing of all tissue accesses [1, 2, 21, 24].

Concerning the lower extremity, it was stipulated that loss of one or two muscle compartments can be physiologically tolerated, assuming that successful skin closure is achieved [2–6, 8, 11, 25].

Irreversible and massive muscular damage (more than two muscle compartments in the leg) inevitably leads to major tissue loss and aggressive prosthesis rehabilitation [1, 6, 8, 18, 25].

6. Important Points to Hold

- (i) CS has a *variable time for onset*; the severity of initial ischemic features and the evidence of postoperative peripheral neuropathy represent important indicators for expeditious fasciotomies.
- (ii) *The duration* of compartmental elevated pressure principally dictates the outcome in CS.
- (iii) Persistent *pain* on passive range of the revascularized limb may suggest incipient CS.
- (iv) Distal “*normal*” *pulses* may mislead the clinician in precocious diagnostic of CS after limb-salvage revascularization.
- (v) Fasciotomy is recommended if tissue pressure (normally <10 mm Hg) raises within 20 mm Hg to 30 mm Hg of *diastolic blood pressure*, or if the *differential pressure between compartment pressure and diastolic pressure shows less than 30 mm Hg*.
- (vi) *Definitive closure of fasciotomy is mandatory* and is only achievable after the swelling has subsided.
- (vii) In diabetic or end-stage renal disease patients that exhibit threatening CLI with deprived collateral resources, foot CS may express complementary *ischemic and focal neurologic injuries*, enhanced by topographic arterial distribution.

7. Conclusion

Compartmental syndrome is a highly threatening complication of the revascularized limb following critical ischemic condition. The lower leg and foot are the most common zones affected by the reperfusion syndrome. Early recognition of CS is mandatory in avoiding local complications and major tissue loss. Whatever the technique (uni- or multilateral approaches), prompt fasciotomy represents the definitive way of treatment of this pathology.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

References

- [1] R. E. Moore III and R. J. Friedman, "Current concepts in pathophysiology and diagnosis of compartment syndromes," *Journal of Emergency Medicine*, vol. 7, no. 6, pp. 657–662, 1989.
- [2] N. T. Mabvuure, M. Malahias, S. Hindocha, W. Khan, and A. Juma, "Acute compartment syndrome of the limbs: current concepts and management," *The Open Orthopaedics Journal*, vol. 6, pp. 535–543, 2012.
- [3] P. M. Walker, "Ischemia/reperfusion injury in skeletal muscle," *Annals of Vascular Surgery*, vol. 5, no. 4, pp. 399–402, 1991.
- [4] X. Li, D. Liang, and X. Liu, "Compartment syndrome in burn patients: a report of five cases," *Burns*, vol. 28, no. 8, pp. 787–789, 2002.
- [5] H. Haimovici, "Muscular, renal, and metabolic complications of acute arterial occlusions: myoneuropathic-metabolic syndrome," *Surgery*, vol. 85, no. 4, pp. 461–468, 1979.
- [6] J. C. Watson and K. H. Johansen, "Compartment syndrome: pathophysiology, recognition and management," in *Rutherford's Vascular Surgery*, pp. 1058–1065, Elsevier Saunders, Philadelphia, Pa, USA, 6th edition, 2005.
- [7] K. B. Simon, "Fasciotomy," in *Operative Anatomy*, pp. 675–680, Lippincott Williams & Wilkins, Philadelphia, Pa, USA, 2nd edition, 2003.
- [8] T. E. Whitesides Jr., T. C. Haney, K. Morimoto, and H. Harada, "Tissue pressure measurements as a determinant for the need of fasciotomy," *Clinical Orthopaedics and Related Research*, vol. 113, pp. 43–51, 1975.
- [9] D. A. Present, N. K. Nainzede, A. Ben-Yishay, and J. T. Mazzara, "The evaluation of compartmental syndromes using somatosensory evoked potentials in monkeys," *Clinical Orthopaedics and Related Research*, no. 287, pp. 276–285, 1993.
- [10] V. Alexandrescu, "The angiosome concept: anatomical background and physiopathological landmarks in CLI," in *Angiosome Applications in Critical Limb Ischemia, in Search for Relevance*, pp. 12–21, Minerva Medica, Torino, Italy, 2013.
- [11] M. Frink, F. Hildebrand, C. Krettek, J. Brand, and S. Hanke-meier, "Compartment syndrome of the lower leg and foot," *Clinical Orthopaedics and Related Research*, vol. 468, no. 4, pp. 940–950, 2010.
- [12] R. D. Burghardt, S. Siebenlist, S. Döbele, M. Lucke, and U. Stöckle, "Compartment syndrome of the thigh. A case report with delayed onset after stable pelvic ring fracture and chronic anticoagulation therapie," *BMC Geriatrics*, vol. 10, article 51, 2010.
- [13] Z. X. Ling and V. P. Kumar, "The myofascial compartments of the foot: a cadaver study," *Journal of Bone and Joint Surgery B*, vol. 90, no. 8, pp. 1114–1118, 2008.
- [14] I. Kitajima, S. Tachibana, Y. Hirota, K. Nakamichi, and K. Miura, "One-portal technique of endoscopic fasciotomy: chronic compartment syndrome of the lower leg," *Arthroscopy*, vol. 17, no. 8, article 33, 2001.
- [15] S. Kinley, S. Frascione, D. Calderone, S. J. Wertheimer, M. A. Squire, and F. A. Wiseman, "Endoscopic plantar fasciotomy versus traditional heel spur surgery: a prospective study," *Journal of Foot and Ankle Surgery*, vol. 32, no. 6, pp. 595–603, 1993.
- [16] S. L. Barrett and S. V. Day, "Endoscopic plantar fasciotomy: two portal endoscopic surgical techniques—clinical results of 65 procedures," *Journal of Foot and Ankle Surgery*, vol. 32, no. 3, pp. 248–256, 1993.
- [17] G. I. Taylor and W. R. Pan, "Angiosomes of the leg: anatomic study and clinical implications," *Plastic and Reconstructive Surgery*, vol. 102, no. 3, pp. 599–616, 1998.
- [18] C. Attinger, P. Cooper, P. Blume, and E. Bulan, "The safest surgical incisions and amputations applying the angiosome principles and using the Doppler to assess the arterial-arterial connections of the foot and ankle," *Foot and Ankle Clinics*, vol. 6, no. 4, pp. 745–799, 2001.
- [19] V.-A. Alexandrescu, G. Hubermont, Y. Philips et al., "Selective primary angioplasty following an angiosome model of reperfusion in the treatment of Wagner 1-4 diabetic foot lesions: practice in a multidisciplinary diabetic limb service," *Journal of Endovascular Therapy*, vol. 15, no. 5, pp. 580–593, 2008.
- [20] M. Edmonds, "A natural history and framework for managing diabetic foot ulcers," *British Journal of Nursing*, vol. 17, no. 11, pp. S20–S29, 2008.
- [21] A. J. Boulton and D. G. Armstrong, "The diabetic foot," in *Clinical Diabetes, Translating Research into Practice*, V. A. Fonseca, Ed., pp. 179–195, Saunders Elsevier, Philadelphia, Pa, USA, 2006.
- [22] O. Iida, S. Nanto, M. Uematsu et al., "Importance of the angiosome concept for endovascular therapy in patients with critical limb ischemia," *Catheterization and Cardiovascular Interventions*, vol. 75, no. 6, pp. 830–836, 2010.
- [23] V. Alexandrescu and G. Hubermont, "Primary infragenicular angioplasty diabetic neuroischemic foot ulcers following the angiosome distribution: a new paradigm for the vascular interventionist?" *Diabetes, Metabolic Syndrome and Obesity*, vol. 19, pp. 327–336, 2011.
- [24] C. E. Attinger, K. K. Evans, E. Bulan, P. Blume, and P. Cooper, "Angiosomes of the foot and ankle and clinical implications for limb salvage: reconstruction, incisions, and revascularization," *Plastic and Reconstructive Surgery*, vol. 117, supplement 7, pp. 261S–293S, 2006.
- [25] F. A. Frans, R. Met, M. J. Koelemay et al., "Changes in functional status after treatment of critical limb ischemia," *Journal of Vascular Surgery*, vol. 58, no. 4, pp. 957–965, 2013.



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