8 Compartment Syndrome of the Extremities: Pitfalls in Diagnosis and Management

Luigi Branca Vergano and Philip F. Stahel

8.1 Historical Perspective

The sequelae of untreated compartment syndrome were first described by Volkmann in 1881; he described the clinical features of the syndrome as a paralytic contracture of a limb due to a tight bandage [1]. A better description of the syndrome was provided by Bywaters and BeallI based on a case series of British victims during World War II in 1941 [2]. The authors underlined the general consequences of the syndrome, described as a "crush" syndrome with impending gangrene of the limb, systemic shock, progressive renal failure, and ultimately death. A better understanding of the pathophysiology of the syndrome is attributed to Carter in 1949 [3]. Carter's description related to a muscle trauma leading to increased pressure within a muscular compartment, with consequent impairment of blood supply, ultimately leading to muscle necrosis.

The importance of time from the onset of the syndrome was well understood and described in the early literature. Most authors reported that tissue ischemia lasting for less than 1 h is associated with reversible neuropraxia, while prolonged ischemia over 4 h will induce irreversible axonotmesis. Irreversible muscle necrosis was described beyond 6–8 h of ischemia [4].

The historic basic principles related to the evolution of compartment syndrome and subsequent tissue injury remain valid until present [5].

Traumatic etiologies of acute compartment syndrome can be divided into three main groups: fracture related, soft tissue injury-related, and vascular injury-related [6].

Extremity fractures after high-energy trauma mechanisms represent the most frequent cause of acute compartment syndromes [7].

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Crush injuries are responsible for around 20% of acute compartment syndromes in absence of fractures.

Traumatic vascular injuries can induce acute compartment syndrome as a consequence of ischemia-reperfusion injury [8, 9].

Nontraumatic root causes include exertional compartment syndrome, thermal/burning injuries, constricting casts or wraps, bleeding disorders, soft tissue infections, and iatrogenic complications [5, 10, 11].

8.2 Definition

The definition of acute compartment syndrome of the extremities is an increase in pressure within a defined compartment of the limb, demarcated by a fascia. The increase in the intracompartmental pressure causes a decrease in perfusion pressure, leading to hypoxemia of the tissues. If this situation is prolonged, irreversible impairment of the muscles can occur, leading to tissue necrosis and devastating subsequent patient outcomes.

8.3 Pathophysiology

In the extremities, there are many anatomic compartments, containing muscle groups and neurovascular structures, separated and demarcated by fasciae. The fascia is composed by dense connective tissue; this fibrous tissue envelopes delimit anatomical space, with low compliance. This means that little increase in volume causes high-pressure elevation.

In case of traumatic event and in some nontraumatic accident (burns, ischemia and reperfusion), the injured tissue responds with precapillary vasodilation in the arteriole system of the muscles, along with collapsing venules and increased permeability of the capillary bed. This leads to increased capillary filtration and raise of interstitial fluid; the pressure of interstitial fluid (normally lower than 10 mmHg) raises in injured tissue, with the clinical aspect of edematous limb, and consequently the intracompartmental pressure raises [12–14].

In case of nontraumatic compartment syndrome (e.g., extravasation of drugs), there is a direct increase of extravascular fluid.

The increase in intracompartmental pressure has the direct consequence of external compression of the microvasculature. Efferent capillaries and venules, with their small diameter and lack of intramural musculature, are extremely sensitive to pressure changes, and collapse first. The compression of outflow system, along with venous congestion, diminishes the arterio-venous gradient. This reduction of the gradient leads to a decrease in local perfusion pressure and consequently to tissue ischemia [4, 12, 15, 16].

The congestion of the microcirculation causes an increase in the permeability of the vessel walls, worsening the fluid extravasation in the interstitial space. The increased fluid volume in third space produces tissue edema and increased interstitial

pressure, generating a feedback loop, which increases external pressure on the intracompartmental vasculature, with consequent worsening of tissue ischemia.

Lymphatics, which, in healthy tissues, assist with outflow and decompression, fail rapidly under the increased pressure [17].

Once perfusion pressure reaches a critically low level, severe tissue hypoxemia evolves.

When muscles are deprived of oxygen and metabolic supply, changes in cellular metabolism occur, resulting in the production of reactive oxidative species, which damage endothelial cells, further increasing vascular permeability.

The combination of hypoxia, increase in oxidant stress, and development of hypoglycemia in tissue causes cell edema, cellular swelling, and necrosis.

Furthermore, in reperfusion injury, after a prolonged period of ischemia, the production of oxygen radicals, lipid peroxidation, and calcium influx leads to disturbances of mitochondrial oxidative phosphorylation and, ultimately, cell-membrane destruction; this worsens the extravasation of the fluid in the interstitial space [4, 13, 14, 18].

Neutrophils and other inflammatory cells are drawn to the ischemic regions and release cytokines and chemical mediators, which exacerbate the vascular permeability.

This cycle is perpetuated till complete necrosis of the tissues occurs unless a surgical procedure (fasciotomy) interrupts the loop.

The time from the initial event to the onset of the compartment syndrome can vary from minutes to hours, and different tissues respond in different manners to reduction of aerobic metabolism consequent to ischemia [17].

Peripheral nerves are highly susceptible to ischemia: after 1 h, reversible neurapraxia occurs, and after 4 h irreversible axonotmesis [4].

Muscles are slightly more resistant to the anaerobic metabolism due to compartment syndrome, but when ischemia persists for more than 8 h, irreversible changes are likely to occur. The inflammatory response, if not treated, evolves in irreversible changes and finally to necrosis and fibrotic tissue.

Rhabdomyolysis after acute compartment syndrome has been reported as more than 40%; the massive release of myoglobin in the circulation can lead to acute kidney injury and kidney failure [19].

8.4 Diagnostic Pitfalls: The "5 P's" Revisited

Acute extremity compartment syndrome can be diagnosed on the basis of clinical symptoms, by measurement of intracompartmental pressure, or both.

The clinical symptoms designated by the mnemonic of "5 P's" (pallor, poikilothermia, pulselessness, paresthesia, and paralysis) were historically considered to be the typical signs for the diagnosis of compartment syndrome [16, 17]. However, these clinical signs are typically the signs of arterial ischemia and delayed presentation of a "missed" compartment syndrome [20]. Thus, the guiding principle is represented by pain out of proportion as the only cardinal symptom of acute compartment syndrome of the extremities. In the current age, the classic "5 P's"

have been replaced by "Pain, pain, pain, pain, pain", and the burden is on the physician to either diagnose or rule out the presence of acute compartment syndrome, independent of the mechanism of injury [16, 17, 20].

Severe pain, not proportionated to the severity of the injury ("pain out of proportion"), that does not improve with i.v. painkillers and adequate analgesia should raise the suspicion of acute extremity compartment syndrome. Clinical symptoms appear in the early phases of the development of compartment syndrome in conscious and wakeful patients. Much attention has to be paid to overuse of painkillers, for the risk of hiding the subjective symptoms of the syndrome.

Another early sign is pain on passive stretching of the affected muscles, especially in case of compartment syndrome of the forearm (pain with extension of the fingers) and of the leg (pain with dorsi- or plantarflexion of the toes).

As mentioned before, nerves (especially sensory ones) are very susceptible to hypoxia. For this reason, neurological signs can appear in the onset of compartment syndrome: paresthesia in the affected extremity is common, while complete anesthesia or paresis appears late and not constantly. However, the absence of neurological signs must not rule out compartment syndrome; motor nerves have some resistance to ischemia, and waiting for complete motor deficits to make the diagnosis could be extremely risky [5, 6, 11].

Unless all these clinical signs have high sensitivity, they were shown to have low sensitivity and poor predictive value [16, 17].

All clinical symptoms lack completely in case of non-evaluable patients because of altered mental status: intubated, obtunded, or non-collaborative patients cannot communicate early symptoms of compartment syndrome. Even in awake patients sometimes the pain could be well tolerated, in presence of preexisting neurological disorders (neuropathy in diabetes or nephropathy) or, as mentioned before, due to pharmacological sedation (large use of painkillers, regional anesthesia, epidural pain catheters). Moreover, anxiety or other distracting injuries can contribute to misdiagnose an initial compartment syndrome [10, 12, 13].

On the basis of clinical examination, the limb involved appears swollen and distended and can be very hard at palpation (Fig. 8.1). The presence of distal pulses does not exclude acute compartment syndrome. In fact they can be completely normal for hours; blood flow through large arteries is preserved till the compartment

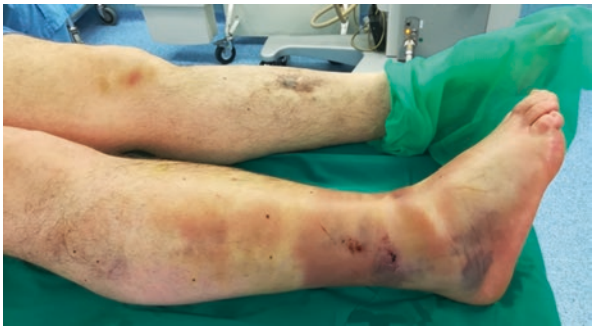

Fig. 8.1 Swollen leg after fracture of the distal third of tibia, with impending compartment syndrome

pressure rises above systolic blood pressure, which occurs in the late phase of compartment syndrome [17].

If the clinical diagnosis is equivocal, measurement of intracompartmental tissue pressure can be extremely useful for the diagnosis. The physiological compartment pressures in adults are around 8–10 mmHg. There are many portable devices for the measurement of intracompartmental pressure: the needle is inserted perpendicular to the skin into the muscle compartment under exam. Less than 1 mL of sterile saline is injected through the needle into the compartment. When equilibrium is reached, that it takes a few seconds, the compartmental pressure is then read on a digital screen. If these devices are not available, an arterial line transducer system with side-port needles can be effective as well. The needle should be inserted near the fracture site (not farther than 5 cm). Pressure measurements should be obtained in all compartments of the extremities involved, especially when analyzing forearm and leg. It is not infrequent to miss the development of acute extremity compartment syndrome in a neighboring compartment [16, 17, 21–24].

A measured pressure greater than 30 mmHg is thought to be an indication for emergency surgical decompression. The use of an absolute value, however, has been questioned because the perfusion pressure necessary for oxygenation is partly dependent on the patients' blood pressure. On one hand, this means that a relatively high pressure in a well-perfused compartment could suggest unnecessary fasciotomy. On the other hand, low compartment pressure in a poor perfused patient (shock) could be alarming and the muscle compartment should be considered hypoxygenated and at risk. Some researchers have suggested the use of differential pressure (Δp = diastolic blood pressure − intracompartmental pressure), with a proposed threshold of 30 mmHg. In a prospective study, McQueen et al. examined the use of a pressure differential ($\Delta p > 30$ mmHg) as diagnostic criteria for acute compartment syndrome. They showed no missed diagnoses of compartment syndrome with this value [14, 25–29].

Compartment syndrome is a dynamic process, and the limb can worsen its edema along minutes and hours. The compartment pressure, even if normal at the initial examination, can raise till high value. For this reason, it is imperative to repeat frequent examinations of the extremities of the patient, especially if consciousness. Continuous measurement of intracompartmental pressure can be made by attaching a catheter to an arterial transducer. This method is controversial, first of all because it measures only one compartment at a time, then because some studies have suggested that the use of continuous measurement can lead to unnecessary fasciotomy [30–32].

8.5 Management of Acute Compartment Syndrome: A Surgical Emergency

Once the diagnosis of compartment syndrome is sure, fasciotomy should be carried out emergently; it is proved that an extended time period between onset of compartment syndrome and surgical treatment worsens the outcome. Animal studies suggest that tissue necrosis occurs within 6–12 h of onset of hypoxemia. The optimum

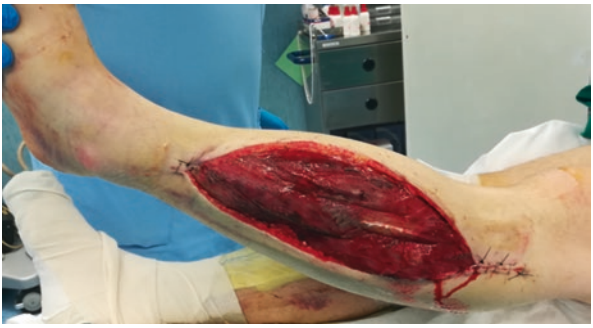

Fig. 8.2 Anterolateral fasciotomy of the leg

timing for fasciotomy is within 8 h from the development of acute extremity compartment syndrome, but the rationale is to perform fasciotomy as soon as possible [33–35] (Fig. 8.2).

In case of missed diagnosis of compartment syndrome for more than 24 h, it is questionable to perform fasciotomy. In fact, in such cases, muscle necrosis has already occurred and simple fasciotomy could pose the patients at high risk of bacterial colonization of necrotic tissues. If necrotic muscles become infected, repeated debridement is needed, and amputation might be necessary if the infection cannot be controlled. On the other hand, if acute extremity compartment syndrome has been missed for longer than 24–48 h without evidence of infection, nonsurgical management should be applied.

The fasciotomy must be complete in length, with decompression of all the muscle compartments affected. Multiple techniques exist that might be used for closing or dressing fasciotomy wounds. It is important to avoid any constrictive dressing: the muscles, especially in the first hours, can worsen their swelling, and they should be allowed to fully expand. The fascia, the subcutaneous tissue, and the skin should left be open. Loose tension-based suture, as shoelace technique with vessel loops, can be applied; this suture can be tightened the following days, when swelling decreases [35–38].

Wet gauzes dressing or vacuum-assisted medication can be alternatively used. Standard wet gauzes should be changed every 24 h. VAC therapy allows 2–4 days of permanence on the wound. The goal of fasciotomy dressing is to facilitate delayed primary closure of the wound. The literature does not show completely agreement on the superiority of VAC dressing over wet gauzes. However, many studies support the evidence that use of VAC is associated with significantly higher rates of primary closure than traditional dressings [37, 38] (Fig. 8.3).

The wound should be closed when feasible: the fascia could left be open in case of excessive tension of the suture. Early closure of fasciotomy wounds has been associated with recurrence of compartment syndrome. When direct suture is not possible, skin graft may be used. Even if primary closure without tension is the gold standard, some authors strongly recommend the use of skin graft for fasciotomy closure: Johnson et al. show less infection and significantly less pain in grafted patients compared to them with primary closure of the wound [39, 40].

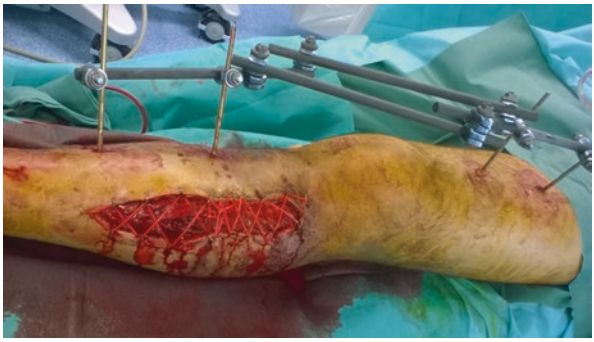

Fig. 8.3 Shoelace technique for closure of an anterolateral fasciotomy of the leg

Complications to soft tissues and skin after fasciotomy are not rare. Nearly one-third of patients have postoperative complication: soft tissue necrosis, wound dehiscence, skin graft infection or necrosis, or need for tissue debridement. Even without occurrence of complications, patients treated with fasciotomy often complain of altered sensation and dry skin with pruritus, besides cosmetic issues [41, 42].

8.6 Specific Compartment Syndrome Locations

Acute compartment syndrome can occur in any location of the upper and lower extremities, from the shoulder to the hands and from the gluteal compartment to the foot. The lower leg represents the most frequent and vulnerable location for acute compartment syndrome, followed by the forearm, thigh, and upper arm [10].

8.6.1 Lower Extremity

8.6.1.1 Gluteal Region

Gluteal compartment syndrome is rare when compared to other anatomical regions. Only about 20% of the cases of gluteal compartment syndrome is secondary to pelvic trauma. More frequently the syndrome is the consequence of nontraumatic conditions, such as prolonged immobilization due to loss of consciousness in patients with history of drug or alcohol abuse, incorrect position during orthopedic or urological surgeries with long operative time, infections, intramuscular drug abuse [43–45].

There are three compartments in this region: tensor fascia lata laterally, gluteus medium and minimus deeper, gluteus maximus posteriorly. Sciatic nerve runs through these compartments, between gluteus maximus and pelvis external rotator complex. When these muscles are swollen, the nerve is at high risk of compression. The consequence might be a neuropathy, with paresthesia in the early stage, and complete palsy if the syndrome remains untreated.

In case of massive necrosis of the gluteus muscles, high quantity of myoglobin is released in the blood stream. The consequences of the rhabdomyolysis are well known, carrying finally to acute kidney failure.

The literature does not reveal precise indications for surgery. Nearly 30% of the patients were treated conservatively, with careful monitoring. The other 70% were treated with emergent decompression of the three compartments [45, 46].

8.6.1.2 Thigh

Acute compartment syndrome of the thigh is a potentially devastating condition. The causes of the syndrome can be traumatic (femur fractures with or without vascular injury, contusion with muscular hematoma) or nontraumatic (external compression in consciousness people caused by narcotic overdose, incorrect intraoperative positioning during orthopedic procedures in patients with epidural anesthesia) [47–49].

The thigh has three compartments: anterior, medial, and posterior.

Clinical presentation of acute compartment syndrome is characterized by excessive painful and tensely swollen thigh. Measurement of the thigh circumference may be helpful to monitoring the condition, determining progression of the swelling.

Some studies strongly recommend the treatment of acute compartment syndrome of the thigh with emergent fasciotomy. Often large inter or intramuscular hematoma is evacuated during surgery. Delayed fasciotomy was associated with increased complications. But has to be noticed that fasciotomy of the thigh results in large scars, with wound infection reported in up to 67% of the cases. On the other hand, thigh musculature can tolerate elevated compartment pressure for long period, if compared to other muscular regions [16, 48–50]. In fact, the compartment has large volume and its fasciae have a relatively elasticity.

For the reasons above, the course of the thigh compartment syndrome is variable. Some patients have high morbidity and mortality, others have an uncomplicated course with excellent outcomes. Consequently, there is not a strict recommendation for the treatment of acute thigh compartment syndrome (surgical versus conservative).

In the literature, the overall death rate in patients with acute thigh compartment syndrome was reported to be very high (till 47%). Other authors have instead reported mortality of 11%, caused by associated injuries rather than by direct consequences of the syndrome [47–49].

8.6.1.3 Lower Leg

The lower leg is the most common location of acute extremity compartment syndrome. In general, up to 70% of all cases of compartment syndrome are associated with fractures, and nearly 40% of all cases are secondary to diaphyseal fractures of the tibia [6].

The lower leg consists of four compartments: anterior (containing ankle extensor muscles), lateral (peronei muscles), superficial posterior (sural triceps), and deep posterior (ankle flexor muscles). The anterior and lateral compartments are the most frequently affected by compartment syndrome.

Fractures of the diaphysis of the tibia are most commonly associated with acute extremity compartment syndrome of the lower leg (from 2 to 9% reported in the literature). Even fractures of proximal and distal metaphysis of the tibia can

provocate compartment syndrome, with different rate of occurrence described in the literature (1.4–17%). The predominance of compartment syndrome occurring in the middle third of the tibia can be attributed to the fact that diaphyseal portion of the tibia is surrounded by bulky muscle mass. Among tibial shaft fractures, displaced, comminuted, and segmental patterns are the most prone to develop compartment syndrome. Even then high-energy tibia fractures are associated with increased risk of developing compartment syndrome, a significant association between fracture type (A, B, or C sec. Ao classification) and compartment syndrome is yet debated. An open tibia fracture does not exclude the possibility of developing acute extremity compartment syndrome [6, 29, 34, 51–54].

Younger patients have higher risk to develop lower leg compartment syndrome, compared to old ones. This may be explained by the difference in muscle mass between young and old patients; younger have more muscle mass that can potentially swell against relatively noncompliant fascia in limited space. Even the property of the fascia can change during the years because of the different proportion and characteristics of collagen fibers between young and old people [51, 52, 55, 56].

The definitive treatment is emergency fasciotomy and decompression of the four compartments. This is usually achieved with two incisions, one centered over the intermuscular septum laterally (decompression of anterolateral and lateral compartments) and the other posterior to the subcutaneous posteromedial border of the tibia (decompression of superficial and deep posterior compartments). A four-compartment decompression by a single incision has been described [57]. The incision is lateral, and can be done with or without fibulectomy. The procedure is usually performed by general or epidural anesthesia, but even bedside fasciotomy under local anesthesia could be an option in very unstable patients.

8.6.1.4 Foot

Each foot has nine compartments: medial, lateral, four interossei, and three central. Measurement of intracompartmental pressure of all compartments is virtually impossible because of the difficulty to target every compartment with the misuration needle. Although emergency fasciotomy is indicated in practically all other acute extremity compartment syndromes, its recommendation in the foot lacks consensus and is still debated. To make multiple large incisions on swollen and injured foot posed the extremity at high risk of wound complications; some surgeons thus prefer to manage the sequelae of untreated compartment syndrome [58–62].

8.6.2 Upper Extremity

8.6.2.1 Shoulder

Deltoid compartment syndrome is extremely rare, with few cases described. The reported cause is usually nontraumatic: prolonged compression in obtunded patients, drug or anabolic abuse with intracompartmental injection of heroin or hormone, long operation time in lateral decubitus. The fasciotomy is mandatory, with decompression of the three deltoid compartments [63–65].

8.6.2.2 Upper Arm

Upper arm includes two compartments, anterior (flexor) and posterior (extensor). The brachial fascia surrounds the two compartments that are separated by medial and lateral intermuscular septa.

Upper arm compartment syndrome is very rare; the few reported causes are traumatic (fracture, tendon rupture) and nontraumatic (anticoagulants, prolonged use of tourniquet).

Decompression can be achieved by a lateral incision (in case of concomitant fracture fixation) or by a medial incision (in case of revision of vascular lesion). There is no consensus on whether a release of both the anterior and posterior compartments should be performed when only one compartment is affected [17, 66, 67].

8.6.2.3 Forearm

The forearm is the most common site of compartment syndrome in the upper extremity. In the forearm there are four compartments, divided by fascia layers: superficial and deep volar compartments, dorsal compartment, and the mobile wad (posterolateral compartment).

Deep volar compartment is the most susceptible to ischemic and compression injury. It lays between the interosseous membrane, virtually inextensible, and the superficial volar compartment. It contains flexor pollicis longus and flexor digitorum profundus.

Superficial volar compartment, divided by fascia from the deep volar one, includes pronator teres, palmaris longus, flexor digitorum superficialis, flexor carpi radialis, and flexor carpi ulnaris.

Within the volar compartments run the median nerve, the anterior interosseous nerve, and the ulnar nerve. The median nerve runs in the forearm between the flexor digitorum superficialis and the flexor digitorum profundus, and is the most commonly injured in forearm compartment syndrome. It can also be compressed under the transverse carpal ligament.

The dorsal compartment lays on the interosseous membrane and contains extensor of the thumb and extensor of the long fingers.

The mobile wad includes the brachioradialis, extensor carpi radialis longus, and extensor carpi radialis brevis.

Fractures of the forearm, both open and closed, are the most common causes of forearm compartment syndrome. There are also nontraumatic causes of forearm compartment syndrome, including reperfusion injury, angioplasty or angiography, intravenous line extravasations, injection of drugs, coagulopathies, or bleeding disorders [68, 69] (Fig. 8.4).

In awake patients, pain out of proportion and pain with passive stretching of the fingers in a swollen forearm are considered very sensitive signs of compartment syndrome. In the early phases, it is fundamental to remove any bandages or splints that may be causing external compression on the forearm. The fractures should be realigned. If the suspect of compartment syndrome persists, fasciotomy should be urgently executed.

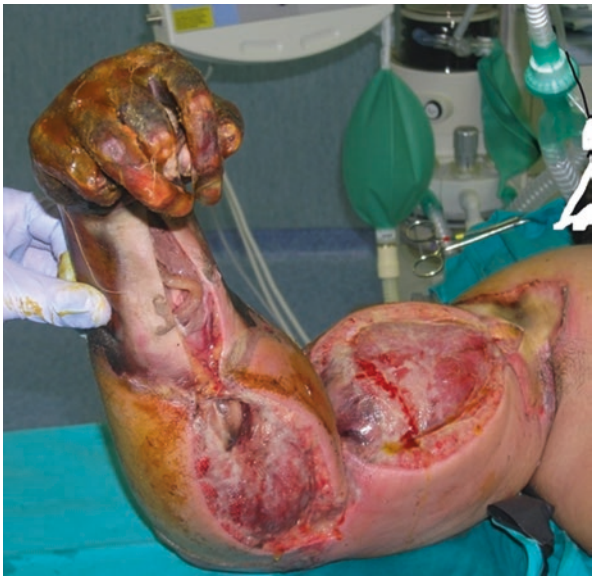

Fig. 8.4 Compartment syndrome and fasciotomies of the upper extremity after an electrocution

Even if the compartment could involve only one or two compartments, many authors suggest to decompress all the four compartments: this is accomplished with two incisions, one on the volar aspect and one on the dorsal aspect of the forearm. The volar incision begins proximal and medial to the antecubital fossa and extends distally in a curvilinear fashion till the midline, at the level of the carpal tunnel (that should be released). The dorsal compartment and mobile wad are decompressed with a single incision beginning at the level of the lateral epicondyle and extending to the distal radioulnar joint in line with Lister tubercle [68–70].

Delay to decompress the forearm is predictive of long-term complications. If patients underwent fasciotomy after 6 h of presentation, then they were significantly more likely to develop complications. The overall complication rate, as described in a recent systematic review, is about 40%, including neurologic deficits, contracture, delayed fracture union, muscle necrosis, complex regional pain syndrome, and tethering of skin graft to tendon limiting motion [71, 72].

8.6.2.4 Hand

The hand is composed of ten myotendinous compartments. Although anatomic studies suggest the presence of variability among individuals, the hand compartments are the thenar, hypothenar, adductor, and seven interosseous compartments. The carpal tunnel is mostly considered part of the forearm. Each of these compartments is bound by its own fascial envelope. The digits, despite the absence of muscle bellies, are considered by some authors to contain individual compartments, delimited by the boundaries of Grayson and Cleland ligaments [73].

Due to the low incidence of hand compartment syndrome, few large studies exist, with the literature describing small case series or case report. The underlying

causes slightly differ from other compartment syndrome of the extremities: complications related to intravenous infiltrations, crush injuries, fractures, prolonged external compression, insect bites, snake envenomation, high-pressure injections, infection, and burns [74, 75].

In the event of suspected compartment syndrome, any dressing should be removed. If the suspicious persists, decompression must be carried out urgently. The release of the dorsal and volar interossei is obtained with separate longitudinal dorsal incisions over the second and fourth metacarpal. Dissection is carried down along the sides of each metacarpal, and the fascia is incised. Deeper dissection is continued along the radial aspect of the second metacarpal to release the adductor compartment. A similar technique is used along the radial and ulnar side of each metacarpal to decompress the volar interossei. To release the thenar and hypothenar compartments, volar incisions are needed. Digital decompression is performed using midaxial incisions, releasing the Cleland ligaments, taking caution to avoid the neurovascular bundles [74–76].

If not treated, acute compartment syndrome of the hand will evolve in intrinsic contracture. The muscle bellies, after ischemia and necrosis, become fibrotic and shortened. The hand will assume an intrinsic minus position, with the metacarpophalangeal joints in extension and the interphalangeal joints in flexion. The contracture of the first webspace, due to retraction of adductor muscles, can be associated to the other deformities. The prognosis for functional recovery is extremely low [74, 75].

8.7 Considerations in the Pediatric Population

Acute compartment syndrome in children is a rare but potentially devastating condition affecting orthopedic patients. The reported causes are the same of those of the adult population, with traumatic as the most common. In the upper extremity supracondylar humerus fractures and both bone forearm fractures are the most frequently involved in acute compartment syndrome; in the lower limb, tibia fractures are the main causes. Nontraumatic causes can be iatrogenic, due to casting complications, intravenous infiltrations, or intravenous medication administration [26, 77].

The diagnosis of acute extremity compartment syndrome in children is particularly difficult and is often delayed because the classic signs commonly described in adults are not constant. The three A's can be helpful when formulate a suspicious of compartment syndrome in children: anxiety, agitation, and increasing analgesic requirement. As described, the pressure threshold that mandates fasciotomy is debatable. In adults an absolute pressure of 30–45 mmHg has been suggested as an indication for decompression. Because normal compartment pressures are higher in children, and these values cannot be used as reliable standards in children. Furthermore, direct measurement of intracompartmental pressure using a needle and catheter is invasive and can be difficult in children. With this, compartment syndrome remains fundamentally a clinical diagnosis even in children [26, 77, 78].

When compartment syndrome is suspected, circumferential dressings should be split and casts should be bi-valved. If the clinical diagnosis of compartment syndrome is made, emergent fasciotomy and decompression is indicated. Despite a long period from injury to surgery in many cases reported in the literature, excellent results were achieved with fasciotomy in most patients. The reason is that children can tolerate increased intracompartmental pressure for longer periods of time than adults before tissue necrosis becomes irreversible [77, 79].

8.8 "Crush" Syndrome

Crush injury, literally, is a lesion resulting from direct physical crushing of the muscles due to something heavy. When the limbs are subject to prolonged pressure or are tightly restrained, rupture of muscle cells releases myoglobin. Myoglobin is filtered out of the glomerulus, but once the renal threshold is exceeded, it precipitates in the distal convoluted tubules causing obstruction. Furthermore, other substances such as potassium, magnesium, phosphate, acids, enzymes like creatine phosphokinase and lactate dehydrogenase are released into the blood stream. These are essential for cell function, but are toxic when released into circulation in large amounts. The consequent metabolic changes rapidly leads to the so-called crush syndrome [2, 18, 19, 80].

The muscles are grossly swollen, hard, cold, insensitive, and necrotic (Fig. 8.5). Kidneys also tend to be edematous and increase in volume. The released potassium in the circulation causes alteration in cardiac rhythm; respiratory gas exchange due to lung edema. Ultimately, shock develops, followed by ARDS, SIRS, sepsis, and finally death.

The majority of crush syndrome reported in literature are associated with disaster (earthquake, explosion, terroristic attack), with a large number of victims. In daily practice, crush syndrome can be occasionally seen in comatose patients, after prolonged rescue in traffic accident, in patients receiving surgery in tight position [80, 81].

Most of these patients are conscious at rescue; crush injuries, in fact, are not common after head and chest injuries because the prolonged pressure necessary to cause this syndrome often results in death for massive brain injury or for hypoxia.

After rescue and initial resuscitation, the key is to recognize the signs and symptoms of the syndrome: petechiae, blisters, muscle bruising, and superficial injuries

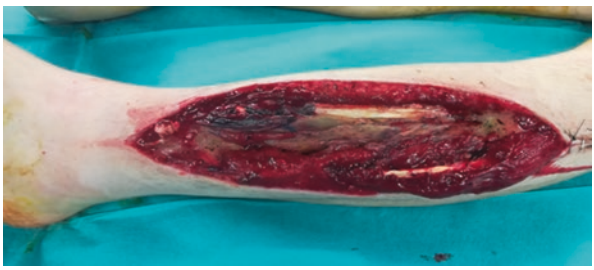

Fig. 8.5 Muscle necrosis after crush syndrome of the leg

are common. Myalgia, muscle paralysis and sensory deficit, fever, cardiac arrhythmia, pneumonia, oliguria, and renal failure are the natural consequence of crush injuries. After rescue of these patients, reperfusion of the muscles can worsen the general status. Large amounts of potentially toxic substance are suddenly released into the blood stream, reaching rapidly brain, heart, lung, and kidneys. The keys of treatment are replacement of fluid, maintenance of effective kidney function, and decompression of the suffering muscular compartments [80, 81].

8.9 Acute Exertional Compartment Syndrome: A Rare Occurrence That Is Frequently Missed

Nontraumatic root causes of acute compartment syndrome of the extremities were described earlier in this chapter. One of the most concerning entities is related to acute exertional compartment syndrome which can occur after any extent of physical exercise in absence of a preceding trauma mechanism [16, 20]. A multiplicity of case reports have been published on the rare entity of acute exertional compartment syndrome of the leg, foot, and upper extremity [82–88]. All these reports unequivocally emphasize the notion that a missed diagnosis leading to delayed surgical treatment is associated with dismal patient outcomes, and most frequently related to the absence of a trauma mechanism which decreases the level of suspicion by the treating physician [82–88]. These findings corroborate the notion that the presence of pain out of proportion, reflected by the revised "5 P's" (Pain, pain, pain, pain, pain!), frequently represents the exclusive cardinal symptom suggestive of presence of acute compartment syndrome of the extremities, independent of the underlying etiology and absence of a preceding trauma mechanism [20, 89]. Thus, as a general rule of thumb, it is highly prudent for any physician, independent of the medical specialty, to consider presence of acute compartment syndrome in ANY patient with pain to the extremities, independent of the root cause, until proven otherwise [20, 89].

8.10 Outcomes of Compartment Syndrome and Sequelae

Acute compartment syndrome is related to high costs for the community: hospitalization increased threefold and overall costs are 2.3 times higher than for uncomplicated patients. Acute compartment syndrome can be a reason for legal dispute; late diagnosis and subsequent late treatment are the most important factors related to indemnity compensation [90–92].

The overall mortality after compartment syndrome has been reported to be as high as 15%, but the correlation is suggested to be more with the concomitant traumatic injury than with the extremity lesion itself. Among survived patients, loss of limb is obviously the worst complication after development of compartment syndrome. The reported amputation rate after compartment syndrome is 5.7–12.9%. Risk factors for amputation include male gender, associated vascular injury, and delayed fasciotomy [16, 20, 89].

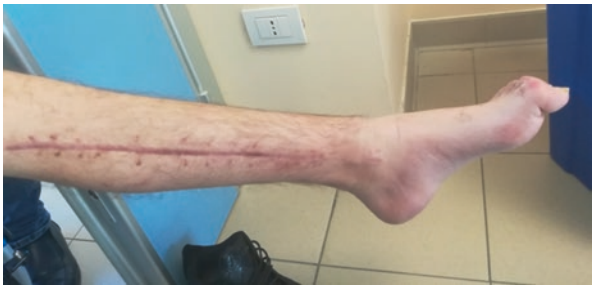

Fig. 8.6 Typical deformity of ankle and foot after compartment syndrome of the lower leg

When fasciotomies are performed more than 8 h after injury, rate of complications, and consequently bad outcomes, progressively increases. Consequently, even in case of saving the limb, the patient can still develop tremendous disability. A delay in the diagnosis of acute compartment syndrome can have devastating consequences for the function of the extremity of the patient. Physicians and patients might believe that a delay in diagnosis is due to abnormal presentation and symptoms; on the other hand, inadequate training and poor culture of trauma could represent important risk factors for missed diagnosis [91, 92].

When acute extremity compartment syndrome of the lower leg is missed or treatment is delayed, late functional disabilities mostly consists in limited range of movement of the ankle, reduced functional strength of the foot extensors, contractures of the foot flexors, abnormal superficial sensibility, and chronic pain. The ischemic insult to the nerves might result in decreased proprioception and sensation. Lesser-toe deformities (claw toes) and cavus foot deformity are common sequelae as well. It has to be noted that the degree of subsequent functional disability is strongly influenced by the severity of the primary soft tissue trauma [38, 56, 59, 60] (Fig. 8.6).

In the upper limb, Volkmann contracture is the result of irreversible tissue ischemia, muscle necrosis with consequent fibrotic evolution and retraction. In the forearm, the typical posture that develops includes elbow flexion, forearm pronation, wrist flexion, and thumb adduction with the metacarpophalangeal joints in extension and the interphalangeal joints in flexion. The median nerve is often affected because it lays in the deeper zone of the forearm, that is more severely compromised by ischemia. The main goal in the management of Volkmann contracture is to restore function; however, normal function of the upper extremity should not be expected. Affected muscles are exposed, fibrotic tissue is removed, tenolysis and neurolysis of the median and ulnar nerves should be performed. Tendon transfer may be a solution to improve the long-term residual function [68, 69, 72, 74].

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