Review: Acute Compartment Syndrome of the Foot

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ABSTRACT

Foot compartment syndrome is a serious potential complication of foot crush injury, fractures, surgery, and vascular injury. The purpose of this article is to summarize and review the existing literature on this entity. Long-term sequelae of foot compartment syndrome (FCS) include contractures, deformity, weakness, paralysis, and sensory neuropathy. These complications are poorly tolerated, and often necessitate multiple procedures for rehabilitation. Therefore, the threshold for considering compartment syndrome and performing fasciotomy must be low to minimize such outcomes. The existence of nine foot compartments and frequent presence of complicating injuries necessitate multi-stick needle catheterization for direct measurement of compartment pressures.

Fasciotomy is indicated when compartment pressure exceeds 30 mmHg, or if compartment pressure is greater than 10-30 mmHg below diastolic pressure. The approaches for compartment decompression generally include two dorsal incisions for access to forefoot compartments, and one medial incision for decompression of the calcaneal, medial, superficial, and lateral compartments.

INTRODUCTION

The pain and swelling associated with foot injuries frequently obscure the presentation of compartment syndrome.27,28 Considering the potential for development of debilitating complications, failure to recognize an elevated compartment pressure in the foot is a serious pitfall. Until Myerson’s description of foot compartment syndrome in 1988, this entity was largely unrecognized with only infrequent case reports appearing in the literature.3,29,30 Since then, much has been learned about the anatomy of foot compartments, clinical presentation, accurate diagnosis, and proper treatment of foot compartment syndrome. Long-term, significant disability resulting from unrecognized foot compartment syndrome underscores the necessity of prompt diagnosis and treatment. This is a review article intended to summarize existing foot compartment syndrome literature.

History

In 1881, Richard von Volkmann described the development of contractures and paralysis following application of restrictive dressings to an injured limb.37 Hildebrand was the first to suggest the relationship of Volkmann’s ischemic contracture with elevated tissue pressure in 1906.13 Murphy later described fasciotomy for relief of elevated compartment pressures following hemorrhage into a fascial compartment.24 In 1926, Jepson demonstrated the benefits of early fasciotomy in the presence of elevated compartment pressures.15 Much investigation has been devoted to compartment syndrome since these early studies. It is now generally accepted that elevated compartment pressure leads to impaired microcirculation in affected compartments.6,35,36 Elevated compartment pressure induces ischemia, compromises muscle and nerve tissue which may result in limb dysfunction and deformity.4,11,32

Prior to the late 1980s, foot compartment syndrome was largely unrecognized in the literature.29,30 More thorough investigation followed recognition of fixed foot deformities such as claw toe, pes cavus, and equinus as sequelae of severe foot trauma.4,11,18,32,36 The pathophysiology of these entities is identical to the classic Volkmann’s ischemic contracture of the hand following forearm compartment syndrome.4

Pathophysiology

The end-point of a compartment syndrome is permanent myoneural tissue damage within a compartment. It is secondary to compromised perfusion induced by increased intracompartmental pressure.21,37,41,4 The etiology of compartment syndrome includes any event that decreases compartment volume or increases content of a relatively fixed volume.4,41 Typically, an inciting event
results in hemorrhage, edema, or a combination thereof within a compartment.4,7,9,11,14,17,22,23,28 Such events include crush injury, fracture, reperfusion injury, surgical procedures, or occlusive dressings. Inelastic osseofascial planes provide limits to volume expansion so that compartmental pressure elevates with continued hemorrhage and edema.16,15 This increase in interstitial pressure decreases the transmural pressure gradient between the microcirculation and interstitium, thus inducing ischemia within the affected compartment.21,25

Under normal physiologic conditions, compartmental pressure is less than 8 mmHg.4 Experiments have demonstrated that perfusion within a compartment ceases when intracompartmental pressure equals diastolic pressure — the critical closing pressure, as proposed by Burton.6 In the presence of normal vascular tone and blood pressure, complete cessation of perfusion occurs well below diastolic pressure in the forearm and calf (64 and 55 mmHg, respectively).21 Under conditions of vasoconstriction or hypertension, complete ischemia was observed at even lower pressures.21 The exact pressure at which tissue damage occurs is subject to debate, and previous conclusions were derived from studies of the leg and forearm. However, it is agreed that fasciotomy is always indicated well before compartmental pressure equals diastolic pressure.4,7,9,11,21,25-27 Although pressure studies of foot compartments have not yet been performed, it is prudent to perform foot compartment fasciotomies if the suspicion of compartment syndrome arises.

The exact mechanism of compartment syndrome has yet to be elucidated, but the most accepted theory is that of local venous hypertension. Under normal conditions, intraluminal venous pressure exceeds interstitial pressure thereby maintaining vein patency.21 As tissue pressure increases, venous luminal pressure is exceeded, causing the veins to collapse and blood flow to cease. The ensuing loss of a vascular-interstitium gradient results in cessation of capillary blood flow. As demonstrated in animal studies, three hours of ischemia in the rabbit leg results in increased capillary endothelial permeability sufficient to produce post-ischemic swelling of 30 to 60%.3 Swelling compounds the ischemic insult, and further increases intracompartmental pressure.

Other proposed theories implicate arterial spasm following elevation of compartment pressure,4 or collapse of arterioles once critical closing pressure within the compartment is reached.4 Regardless of which theory or combination thereof is responsible, the end result is ischemic necrosis of compartmental tissue in the absence of prompt intervention.21

Investigations using animal models suggest that irreversible nerve and muscle damage begins after five to six hours of ischemia.21 Muscle is particularly sensitive to changes in oxygen tension, with cicatrix (scar) formation following the development of muscle necrosis.31 Harmon and Guinn have shown that 90% of muscle fibers show evidence of injury after eight hours of ischemia.31,34 Subsequent longitudinal and horizontal scarring leads to contracture formation and adhesion to surrounding tissues, thereby compounding deficits in musculotendinous excursion.31 Animal models suggest ischemia time of 12 hours is required to produce contractures.25 Functional neural deficits begin within 30 minutes of ischemia onset. These become irreversible after 12 to 24 hours of ischemia. Muscle dysfunction occurs after two to four hours of ischemia, and becomes irreversible at four to 12 hours. The relatively early onset of neural dysfunction may allow for early diagnosis and timely treatment of compartment syndrome. Clinical studies of compartment syndrome sequelae have also suggested the 12-hour delay between the onset of compartment syndrome and irreversible damage.21 Cellular injury to muscle often results in myoglobinuria, potentially resulting in renal injury.33

Neural deficits precede the onset of myoneural necrosis by a few hours. This fortunately allows a time window for diagnosis on the basis of neurological findings. However, functional abnormality of nerve tissue does not occur until at least 30 minutes after ischemia onset, making this finding intermediate in the time course of compartment syndrome.25 Treatment before the onset of neurological deficit is essential. One review of literature found that only 13% of patients with drop foot at the time of diagnosis recovered function after late fasciotomy.5 As the scarring process develops over a period of six to 12 months, nerve compression often develops, leading to varying degrees of nerve injury beyond that suffered during the initial ischemic insult.31,34 Both duration and degree of compression determine the extent of nerve injury.4 Nerve and muscle injury both lead to contracture formation.

Contracture deformities are particularly troublesome in the foot considering the severe functional deficits that may occur with foot compartment syndrome. The most common deformities include equinus, equinovarus, claw toe deformity, and pes cavus. Foot compartment syndrome most commonly leads to claw toe deformities (intrinsic minus) as the intact extrinsic toe flexors and extensors overpower the damaged intrinsic foot muscles.1,2,17 Cavus may be caused by leg compartment syndrome with contracture of tibialis posterior. Another mechanism of cavus foot deformity is post foot compartment syndrome fibrosis of intrinsic foot planter structures. With contracture of the intrinsic muscles and tendons, “intrinsic plus” deformity is characterized by flexion of the metatarsophalangeal joints, and extension of the interphalangeal joints.4 This is not a true intrinsic
plus deformity since the term implies continued function of intrinsic foot muscles rather than deformity resulting from contracture. Development of an insensate foot predisposes patients to complications of neuropathic extremities such as formation of chronic ulcers and joint destruction.4

Anatomy

Early anatomical studies of foot compartments divided the foot into four compartments; medial, lateral, central, and interosseus.10 In 1990, Manoli and Weber described three patients with calcaneal fractures who later developed progressive claw-toe deformities, prompting further investigation into the entity of foot compartment syndrome.20 Their cadaver dye injection studies revealed the presence of nine compartments in the foot, rather than four compartments as previously thought (Table 1). Three compartments run the entire length of the foot (medial, lateral, superficial). Five compartments are contained within the forefoot (adductor and four interossei). The calcaneal compartment is confined to the hindfoot, but does communicate with the deep posterior compartment of the leg as demonstrated by injection studies. Some have suggested the possible presence of a tenth dorsal compartment containing the extensor digitorum brevis.21

The calcaneal compartment contains the quadratus plantae muscle and the lateral plantar neurovascular bundle. In some patients, the medial plantar nerve is located in the distal portion of the calcaneal compartment.21 Previously, the quadratus plantae was thought to be contained in the forefoot as proposed by Grodinsky in his studies of infection spread within the foot compartments.10 However, a transverse septum extending between the medial and lateral intermuscular septae just superficial to the quadratus plantae divides the superficial and calcaneal compartments.21 The posterior tibial nerve and vessels also traverse the calcaneal compartment proximally, allowing communication between the calcaneal and deep posterior leg compartments.18,21,29 Injuries such as calcaneus fractures, tibia fractures and crush injuries to either compartment have resulted in combined compartment syndromes of the calcaneal and deep posterior leg compartments.18,21 This necessitates monitoring of the deep posterior leg compartment in conjunction with calcaneal compartment measurements. Adequate release of the calcaneal compartment may include release of the distal tarsal tunnel through extension of the medial incision posteriorly.

Clinical Presentation and Diagnosis

Myerson’s retrospective study of 12 patients treated for compartment syndrome following trauma to the foot clarified the presentation of foot compartment syndrome.28 Present in all patients was foot pain. However, many of these patients suffered severe crush injuries to the foot, thus introducing a confounding factor in using pain as a means of diagnosis. Presence of these injuries should introduce a high index of suspicion for the development of compartment syndrome, even in the presence of associated open injuries.28,29,31 Open injuries do not necessarily release compartments as it is improbable that all nine compartments will be released with open injury. Small fascial defects associated with foot trauma may not be sufficient to significantly alter compartment volume.21,28,33

Despite the potential for confusion with massive foot trauma, pain in the presence of compartment syndrome is usually out of proportion to injury, and will not abate with adequate immobilization of the injured foot.21,27 A developing compartment syndrome should also be considered if progressive doses of analgesics are required.

Table 1: Nine compartments of the foot. Key structures of each compartment are also listed.

<table>
<thead>
<tr>
<th>Hindfoot (1)</th>
<th>Forefoot (5)</th>
<th>Full length (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CALCANEAL</td>
<td>INTEROSSEUS (x 4)</td>
<td>MEDIAL</td>
</tr>
<tr>
<td>Muscle:</td>
<td>• interossei</td>
<td>• flexor hallucis</td>
</tr>
<tr>
<td>• quadratus plantae</td>
<td>ADDUCTOR</td>
<td>• abductor hallucis</td>
</tr>
<tr>
<td>Neurovascular structures:</td>
<td>• adductor</td>
<td>LATERAL</td>
</tr>
<tr>
<td>• posterior tibial nerve, artery, vein</td>
<td>• abductor digitii quinti</td>
<td></td>
</tr>
<tr>
<td>(site of communication with deep posterior leg compartment)</td>
<td>• flexor digiti minimi</td>
<td>SUPERFICIAL</td>
</tr>
<tr>
<td>• lateral plantar nerve, artery, vein</td>
<td>• flexor digitorum brevis</td>
<td></td>
</tr>
<tr>
<td>• +/- medial plantar nerve</td>
<td>• lumbricals (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• flexor digitorum longus tendons</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• +/- medial plantar nerve</td>
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to relieve pain following extremity trauma. Pain with passive motion has also proved to be of use in making the clinical diagnosis of a foot compartment syndrome. A simple bedside test is to passively dorsiflex the toes to stretch the intrinsic muscles of the foot, thereby leading to decreased volume and exacerbation of pain. Myerson found that 86% of patients in his series with compartment syndrome of the foot experienced pain with passive motion, making it the most sensitive subjective clinical finding. Pain with passive stretch is not specific for compartment syndrome as some degree of pain is present with any degree of muscle swelling or ischemia.

Decreased two-point discrimination and light touch deficits on the plantar aspect and toes are more reliable than decreased pinprick sensation. Serial sensory exams are necessary as relative decreases in sensation over time are more sensitive findings than a single sensory exam. Motor deficits in foot trauma patients are the least useful as motor strength is difficult to grade objectively, and patients are often limited by pain. It is important to bear in mind that neurological deficits are relatively late findings, and should be acted upon quickly.

Vascular examination is particularly unreliable for diagnosing compartment syndrome. Myerson has repeatedly documented findings of palpable dorsalis pedis and posterior tibial pulses in addition to satisfactory capillary refill time in feet with compartmental ischemia. Not only are the common sites for palpating foot pulses extra-compartmental, but diminution of palpable pulses and loss of capillary refill are very late findings in compartment syndrome. This is due to the high transmural pressure gradient in large arteries at near systolic pressure as compared with near diastolic transmural pressure in the microcirculation. Therefore, vascular exams are not reliable to exclude the diagnosis of compartment syndrome. Nevertheless, accurate management of foot injuries requires a thorough vascular examination.

Multi-stick invasive catheterization is essential for accurate diagnosis of compartment syndrome in the presence of clinical suspicion. This allows for direct quantification of intracompartmental pressures, and is the most sensitive means of detecting occult compartment syndrome. However, deciding on whether operative intervention is indicated should be based on the combination of compartmental pressures and supporting clinical findings. Due to the identification of at least nine foot compartments, multiple sticks are necessary in measuring foot compartment pressures. Selective catheterization of traumatized feet suggests the calcaneal compartment is subject to relatively higher compartmental pressures than other foot compartments.

Thus, every attempt should be made to determine calcaneal compartment pressure. The threshold for performing fasciotomy in the foot is based on extrapolation from compartment syndromes of the forearm and leg. As recommended by multiple studies, fasciotomy should be performed at 30 mmHg or 10-30 mmHg below diastolic pressure. The latter recommendation emphasizes the importance of taking into account the presence of hypertension or hypotension, as is commonly seen in trauma patients. These pressures are derived from leg and forearm studies. However, the pathophysiology of compartment syndrome is likely identical in foot compartments. Until studies specific to foot compartment pressures are performed, it is prudent to treat foot compartment syndrome in the same manner as for more extensively studied compartments.
Catheterization may be performed using commercially available pressure monitors. Medial compartment pressure is measured by inserting the needle approximately 4 cm inferior to the medial maleolus over the abductor hallucis muscle (Fig. 1). From this point, the needle can be advanced into the calcaneal compartment. Pressure in the superficial compartment is measured by reinserting the needle into the arch thereby penetrating the flexor digitorum brevis (Fig. 2). Subsequently, insertion of the needle into the lateral aspect of the foot just inferior to the fifth metatarsal allows pressure measurements of the lateral compartment (Fig. 3). Separate measurements are made for each of the interosseous compartments. Finally, by advancing the needle deep to the interosseous compartments, adductor compartment pressure measurement is obtained (Fig. 4).

The calcaneal compartment warrants special consideration, as it is a small compartment traversed by the neurovascular bundles of the foot. Severe disability is likely if calcaneal compartment syndrome is left untreated. Calcaneal compartment syndrome may also follow relatively low energy injuries as compared to those seen with typical high-energy injuries of the forefoot. The large surface area of cancellous bone bleeding into a small compartment after traumatic injury may explain this discrepancy. Patients with calcaneal fractures frequently experience more severe, unrelenting, acute pain than seen in other fractures. Swelling is frequently significant, but its absence is not a reliable means of predicting development of compartment syndrome.

Treatment

Once the diagnosis of acute compartment syndrome has been made, the goal of relieving pressure in the affected compartment(s) must be met. Fasciotomy is the definitive treatment for acute compartment syndrome. Numerous techniques have evolved with the discovery of additional foot compartments. The choice of fasciotomy is governed by surgeon preference, other planned procedures, and preexisting soft-tissue injury. Elevation of the affected extremity may reduce swelling, but should not exceed the level of the heart as doing so could further compromise tissue perfusion.

Plantar Approach

The plantar approach begins with incision along the plantar aspect of the first metatarsal to expose the medial compartment. The abductor hallucis is retracted inferiorly to allow access to remaining compartments. Blunt dissection is used to complete compartment release. Care must be taken to avoid damage to the plantar neurovascular bundles. This approach is not generally used as some compartments are difficult to access safely by this means. The sensory branch of the medial plantar nerve supplying the medial aspect of the pulp of the hallux is prone to injury, especially with significant swelling. Release of the adductor compartment requires deep forefoot dissection.

Plantar and Lateral Approach

Adding a lateral incision for release of the lateral and interosseous compartments partially minimizes deep dissection, although the adductor compartment must still be released via deep forefoot dissection. The lateral approach may double in utility by serving as the approach for fixation of calcaneus fractures.

Dorsal Approach

Previously, dorsal release was recommended on the basis of the belief that the interosseus compartments were the only ones requiring release. Myerson later...
modified this approach to incisions over the second and fourth metatarsals. These incisions allow access for blunt dissection of the remaining foot compartments deep to the metatarsals. Additional benefits to be reaped by these incisions include access for fixation of Lisfranc fracture-dislocation and other forefoot fractures. Making these incisions medial to the second metatarsal and lateral to the fourth metatarsal minimize risk of skin bridge necrosis.

**Medial Plantar and Dorsal Approach**

With discovery of the calcaneal compartment, investigators realized that a purely dorsal approach might be inadequate for release of this compartment. To ensure adequate release of forefoot and hindfoot compartments, Myerson recommended combined dorsal and medial plantar incisions (Fig. 5). The medial incision begins at the medial side of the calcaneus just anterior to the abductor hallucis origin (4 cm from the posterior aspect of the heel and 3 cm superior to the plantar surface), extending distally parallel to the plantar surface for 6 cm. Following medial compartment fasciotomy, the abductor hallucis is reflected superiorly to expose the intermuscular septum. Care must be taken at this point to avoid damage to the neurovascular bundle (lateral plantar nerve and vessels) just deep to the fascia. Splitting the intermuscular septum medial to the abductor hallucis allows entry into the calcaneal compartment and executes its release. Initiating entry into the compartment at the intermuscular septum, and extending the fasciotomy the length of the skin incision accomplish this. Caution must also be exercised at the distal end of the incision, as the medial plantar nerve is sometimes present at the distal end of the calcaneal compartment. Blunt dissection is recommended for release of the distal septum to avoid injury to these structures. Bulging of the quadratus plantae through the fasciotomy is suggestive of an adequate calcaneal compartment release.

By reflecting the medial compartment superiorly, the superficial compartment is identified just lateral to the medial compartment. The superficial compartment is then opened longitudinally, thus releasing the flexor digitorum brevis. This is followed by inferior retraction of the flexor digitorum brevis with the underlying transverse septum of the hindfoot. This allows exposure of the medial aspect of the lateral septum. Using a sharp elevator or dissecting scissors, the lateral septum is opened from its posterior origin to its anterior limit for release of the lateral compartment. The lateral compartment is considered fully released when the abductor digiti quinti and flexor digiti minimi are both visible (Fig. 6).

Dorsal incisions are made as described above for release of forefoot compartments. This is done through two separate incisions located medial to the second metatarsal, and lateral to the fourth metatarsal. The thin dorsal fascia is opened, followed by elevation of the interspace muscles from the medial aspect of the second metatarsal. This exposes the fascia of the adductor, which is then incised longitudinally. Release of the lateral interosseous compartments is accomplished through the lateral dorsal incision via longitudinal fasciotomies (Fig. 7).

Secondary closure of wounds is generally indicated after five to seven days post-fasciotomy. If skin closure is not feasible, skin grafting may be necessary. For calcaneus fractures, operative fixation should not be attempted at the time of fasciotomy due to the risk of
Foot compartment syndrome is an entity with profound implications for functional morbidity if it is not promptly recognized and treated. Frequently, these patients have suffered severe foot trauma, including crush injuries, calcaneal fractures, Lisfranc fracture-dislocations, and lower leg trauma. Elevated tissue pressure is the only truly accurate means of making a diagnosis as compartment syndrome usually occurs in the presence of concomitant injuries that often confuse subjective clinical findings. Multi-stick catheterization may itself present a challenge, as there are nine compartments in the foot, each of which carries the potential for development of compartment syndrome. Of the foot compartments, the calcaneal compartment carries the greatest risk of long-term deficits if left untreated. The underlying principle is that morbidity suffered from a fasciotomy is minimal when compared to the potential complications of a missed compartment syndrome. If clinical suspicion suggests the development of a compartment syndrome, serial pressure monitoring followed by fasciotomy if compartmental pressure continues to rise is recommended. Until compartment pressure studies specific to foot compartments are performed, it is advisable to perform fasciotomies under the same guidelines as for forearm and leg compartment syndrome.

CONCLUSION

REFERENCES