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DIAGNOSIS AND TREATMENT OF COMPARTMENT SYNDROME OF THE FOOT

Mark Myerson, MD

The entire spectrum of compartment syndromes of the leg is now well recognized as a complication of lower extremity trauma, and much experimental work has been done to define its pathophysiology and treatment. If diagnosis and treatment are carried out expeditiously, many of the disastrous complications of myoneural ischemia can be avoided. However, it has been the author’s experience that little is understood about compartment syndrome of the foot; these devastating injuries are managed cursorially or not at all. The literature records only scattered reports of compartment syndrome of the foot, although one study outlined the experimental basis for fasciotomy in the acute stage. This article outlines the pathophysiology and anatomic basis of compartment syndrome, as well as its clinical presentation, diagnosis, and treatment.

PATHOPHYSIOLOGY

A compartment syndrome is a complex constellation of events that results from an elevation of tissue fluid pressure within a closed space. The foot has four, separate, easily identifiable spaces or compartments rigidly bound by various osseofascial structures (see “Anatomic Basis,” next page). Following injury, bleeding into these closed fascial compartments elevates the local tissue fluid pressure. The compartment syndrome complex develops as the local tissue interstitial fluid pressure increases and capillary perfusion drops below that which is required to maintain tissue viability. Due to the inelastic surrounding osseofascial structures, the elevated tissue pressure is not dissipated and, unless reduced, will eventually cause vascular occlusion and myoneural ischemia.

Many theories have been proposed to account for the sequence of events leading from tissue injury to compartment syndrome ischemia. Experimental studies have demonstrated that tissue perfusion will progressively decrease as tissue compression increases, and that blood flow in the microcirculation will stop when local tissue pressure equals the diastolic blood pressure—the concept of critical closing pressure. In other words, blood flow may cease at levels below the mean arterial pressure secondary to passive capillary collapse when local tissue pressure increases above intracapillary pressure. An alternative hypothesis is that increased pressure actively closes small arterioles, which then creates increased vasomotor tone when transmural pressure is lower.

A currently popular and accepted theory for the pathogenesis of compartmental ischemia involves local venous hypertension. Under normal circumstances, intravenous pressure must exceed the surrounding tissue pressure for the veins to remain patent. Local increases in venous pressure will reduce the arterial-venous gradient and will therefore reduce capillary blood flow. In the foot, when the local tissue pressure continues to rise, it compromises the arteriole-venular gradient, causing capillary blood flow to cease. When pressures are below diastolic levels, blood flow can then no longer sustain local metabolic demands. Therefore, myoneural ischemia can occur in the presence of arterial flow.

Once established, compartment syndrome complex will lead to vascular occlusion and myoneural ischemia. If this ischemic process continues, irreparable damage to tissue ensues and myoneural necrosis occurs. The end result of an untreated compartment syndrome of the
Fig 1: This patient's isolated crushing injury to the foot was treated with immobilization and casting. He presented for evaluation 3 years later with all the manifestations of an untreated compartment syndrome.

Fig 2: Note the equinovarus position of this patient's foot and the marked atrophy of all the intrinsic muscles—complications of untreated ischemia of the foot and leg.

doorsally, the plantar aponeurosis inferiorly and laterally, and an intermuscular septum medially. The intersosseous compartment contains the seven interossei and is bounded by the intersosseous fascia and the metatarsals.

These well-defined osseofascial spaces provide the anatomic setting for a compartment syndrome. Although each compartment comprises a separate entity, communication between them (i.e., leakage of dye from one space into another) has been noted under certain circumstances, such as when pressures were experimentally induced to more than 40 mm Hg. Under normal physiologic pressures, however, there is no communication or direct extension between the fascial spaces of the foot and the leg, other than via the neurovascular bundle.

CLINICAL PRESENTATION

By virtue of its location, the foot is extremely vulnerable to isolated extremity injury. Most of the devastating injuries of the feet treated by the author are caused by crushing forces, typically presenting with massive swelling (Fig 5). Under these circumstances, the clinician should approach initial assessment with a high index of suspicion for potential compartment syndrome, even when associated with open injuries. Because "the eye sees only what the mind knows," clinicians must be made aware of this potential. Open injuries do not automatically decompress compartmental spaces. Major compartment syndromes of the leg have been identified with open tibial fractures and, similarly, the author has treated numerous open foot injuries associated with compartment syndromes.

The diagnosis of a compartment syndrome is clinical, based on the history of the injury and the findings of signs and symptoms compatible with myoneural ischemia. Fortunately, functional losses precede myoneural necrosis by a few hours, allowing clinicians some latitude for earlier diagnosis of ischemia and prevention of tissue necrosis.

The clinical presentation of muscle and nerve ischemia is pain. Because a severely injured foot, often with multiple fractures, is in itself painful, it may be difficult to distinguish the pain of injury from that of a developing compartment syndrome. However, the pain of a compartment syndrome and ischemia does not abate with adequate immobilization of the foot and is usually out of proportion to the injury. The author has found that pain elicited on gentle, passive dorsiflexion of the toes, stretching the intrinsic muscles of the foot, is a sensitive index of compartmental ischemia.
(The equivalent test in the hand is passive abduction and adduction of the fingers.)

Lack of sensation is generally accepted as an important sign of nerve ischemia, but the author has not found this indicator very reliable in the foot; a diminution in sensation over time may be more helpful. In the foot, two-point discrimination and light touch over the plantar aspect and toes are more reliable than loss of pin-prick sensation.

The presence or absence of a dorsalis pedis or posterior tibial pulse is notoriously unreliable in diagnosing an early compartment syndrome. The digital pulse should never be used as a measure of locally compromised tissue. The author has repeatedly documented the presence of both the pulses and of normal capillary refill time, even in a foot with compartmental ischemia. The pathogenesis of the compartment syndrome clearly underscores the potential for misdiagnosis when based on the presence or absence of pulses. Nevertheless, a thorough vascular evaluation, including a routine Doppler examination, should be an integral part of managing these injuries, as it can assist one in preoperative planning.

During the initial evaluation, all dressings should be removed. No circumferential dressings, particularly splints or casts, should be used in the initial management of these injuries where the potential for compartmental ischemia exists. In equivocal cases, high-risk patients should be checked frequently, since compartment syndromes are usually progressive. The foot should not be maximally elevated under these circumstances but, rather, placed at heart level to ensure venous drainage without compromising local arteriolar pressures any further. In some patients who present late in the course of events, irreversible myoneural necrosis may have occurred by the time the ischemia is diagnosed. The diagnosis of compartment syndrome cannot be clinically made in patients with head, cord, or peripheral nerve injuries, although it seldom represents a problem for the isolated foot injury. Nevertheless, for patients with multisystem trauma, as well as in patients in whom the diagnosis is equivocal, more objective tests are available for measuring compartment pressures. Compartment pressure can be measured in a variety of ways, including those proposed by Mubarak et al., Matsen et al., Whitesides et al., and Rorabeck et al. Each method has potential advantages and pitfalls.

In practice, however, intracompartmental pressure measuring devices should be used liberally, with the understanding that changes in compartment pressures often precede the clinical signs of an incipient compartment syndrome. The method the author prefers is utilizing a small, digital, hand-held monitor based on the slit-catheter system (Stryker Quick Pressure Monitor Set). An 18-gauge needle is inserted first into the central and then the interosseous compartments. To measure the pressure in the central compartment, the surface landmark is the base of the first metatarsal, and the needle is passed between the metatarsal and the abductor hallucis muscle. The needle is advanced 1.5 in., taking care to obtain the reading when the needle can be felt to be in soft tissue and not up against bone or tendon. The central compartment can also be monitored from a dorsal approach, measuring the interosseous compartment pressure when passing through it. The interosseous compartment pressure should be measured in two positions by introducing the needle through the intermetatarsal space. The second and fourth web spaces are preferable, as these avoid inadvertent puncture.
Fig 5: This patient sustained a crush fracture dislocation of the tarsometatarsal joint complex when a truck backed up over his foot. Note the massive swelling associated with this injury (A) and the typical type B1 dislocation pattern (B). Reprinted with permission of Foot Ankle.

Fig 6: The double dorsal incision approach to fasciotomy. The incisions should be deepened to bone without any unnecessary subcutaneous dissection. Reprinted with permission of Foot Ankle.

of the dorsalis pedis and its branches. It has been shown in experimentally induced compartment syndromes that the central and interosseous compartment pressures correlate well with each other. 7

As with compartment syndromes elsewhere in the body, strict adherence to clinical findings or reliance on interstitial pressure measurements can be misleading. Treatment should be based on a combination of clinical findings and pressure measurements, particularly in the presence of an upward trend in pressures.

TREATMENT

The treatment for an established acute compartment syndrome is decompressive fasciotomy. Some clinicians advocate fasciotomy when the pressure is greater than 45 mm Hg, however, most clinical and animal studies show the necessity for fasciotomy when pressures greater than 30 mm Hg persist for more than 8 hours. 3,22,23

Because the clinician cannot know exactly when the pressures begin to elevate unless they are measured continuously, the author recommends fasciotomy for an acute foot injury with pressure greater than 30 mm Hg. Even this dictum, however, is subject to the element of time (reliance on pressure recordings for a treatment plan may cause irrevocable delay in expediting treatment) and the individual’s tolerance for increased tissue pressure. 19 This tolerance level varies a great deal from patient to patient, and the same degree of pressure may cause neuromuscular deficits in some patients but not in others. In addition, a hypotensive patient with poor peripheral perfusion may require fasciotomy at a lower intracompartmental pressure. Therefore, the author recommends using a general trend in pressure (if available) as well as the patient’s overall condition to guide treatment in borderline or uncertain cases.

There are several fasciotomy techniques for reliably decompressing intracompartmental pressures to a level which will prevent myoneurial ischemia. Based on experience with the hand, Mubarak and Owen have suggested two longitudinal dorsal incisions over the metatarsals. 24 Grodinski, on the other hand, described a long plantar medial incision for drainage of established infections of the plantar fascia.
spaces of the foot.\textsuperscript{15} This incision, recommended by Loeffler and Ballard\textsuperscript{16} and, more recently, by Bonutti and Bell,\textsuperscript{5} provides access to all the fascial spaces and could also be used to decompress a compartment syndrome.

The anatomic basis for fasciotomy in acute compartment syndromes of the foot was established recently in experimentally induced compartment syndromes.\textsuperscript{7} Compartment syndromes were created in the feet of fresh cadaver below-the-knee amputation specimens and then decompressed via either dorsal or medial incisions. The length of time that it took for pressures to normalize was noted for each approach. Both approaches provided excellent access for decompressing the foot, but pressures took far longer to normalize with the dorsal than with the medial approach.\textsuperscript{7}

The dorsal approach for fasciotomy utilizes two longitudinal incisions (Figs 6-7) based on the second and fourth metatarsals; the author places them slightly medial to the second metatarsal and lateral to the fourth to ensure as wide a bridge of skin as possible. Although seemingly narrow, this bridge of skin seldom undergoes necrosis (Fig 8). Once the skin incision is made, it is deepened to the bone, using a small hemostat to spread the tissue longitudinally; no subcutaneous dissection is performed, minimizing compromise of already tenuous dorsal skin. Once the bone is reached, further longitudinal dissection is performed in each interosseous space (Fig 6). From these dorsal incisions, reaching the medial and lateral compartments requires precise dissection.

The medial approach follows the length of the inferior surface of the first metatarsal, entering the medial compartment between the metatarsal and the abductor hallucis muscle, and providing direct access into the other compartments (Figs 9-10). Once the abductor hallucis muscle is inferiorly retracted, the author uses blunt finger dissection and gentle longitudinal tissue spreading with a hemostat. To avoid injuring the neurovascular bundle when cutting across the central compartment, do not use scissors or sharp instruments. The skin incision can be extended more proximally to decompress the entire posterior tibial neurovascular bundle. The same principles that apply to fasciotomy elsewhere in the extremities apply to fasciotomy in the foot: no tourniquet is used, generous incisions are made, and subcutaneous fasciotomy is not advised. No debridement of muscle at the time of fasciotomy is performed because it is difficult to determine muscle contractility in the foot and, once decompressed, the muscle may recover postoperatively.

Fig 7: When the skin is incised, copious edema fluid is found. Once the fascial compartments have been opened, it is not uncommon to release a massive hematoma under pressure.

Fig 8: The bridge of skin between the fasciotomy incisions needs to be carefully protected. In spite of the narrow width of this dorsal skin bridge, it seldom undergoes necrosis.

Fig 9: The medial fasciotomy incision follows the length of the base of the first metatarsal and enters the foot by retracting the abductor hallucis muscle plantarward. Reprinted with permission of foot Ankle.

The decision to perform the fasciotomy dorsally or medially is based on the presence or absence of fractures amenable to open reduction and internal fixation. Skeletal stabilization, whether by external or internal fixation, or a combination of the two, facilitates wound healing. Rigid skeletal stability enhances the environment for soft-tissue healing, decreases pain, and allows more rapid mobilization of the extremity.

All incisions are left open and are routinely covered with allograft or synthetic skin (Epigard Synthes). This temporary skin coverage minimizes postoperative wound edema and simultaneously supplies a bacteriostatic coverage.
Fig 10: This patient had his foot caught between the couplings of a train. No fractures were present, but compartment pressure recordings measured 64 mm Hg, and he underwent decompression fasciotomy by medial incision.

Fig 11: Allograft skin is used to cover the fasciotomy for the initial management of the wound. Split-thickness skin grafts are utilized on about the fifth day (A). These grafts contract during healing, as shown in this patient 6 months post-injury (B).

CONCLUSION

The treatment of severe crushing injuries of the foot is challenging. The critical factor in management, however, is to recognize and treat these injuries early before the devastating sequelae of ischemia have occurred.

Most crushing injuries are associated with midfoot fractures and dislocations, and it is preferable to treat them after fasciotomy with open reduction and internal fixation. Under these circumstances, the double dorsal incision approach is utilized for fasciotomy, as it provides simultaneous access for fracture reduction and fixation. When a compartment syndrome is associated with a fracture pattern unsuitable for internal fixation, or when crushing occurs without fracture dislocation, then the medial incision is preferable.

REFERENCES


13. Duffield FA, Harris I. Increase of pressure in veins to level of arterial pressure caused by constrasting the limb in which the venous pressure is recorded. J Physiol. 1934; 81:283.


