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Case Reports and Series

Acute Compartment Syndrome of the Foot Due To Frostbite: Literature Review and Case Report

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ABSTRACT

Acute compartment syndrome of the foot and ankle is a relatively rare clinical finding. Lower extremity compartment syndrome is customarily due to vascular or orthopedic traumatic limb-threatening pathologic issues. Clinical correlation and measurement of intracompartmental pressure are paramount to efficient diagnosis and treatment. Delayed treatment can lead to local and systemically adverse consequences. Frostbite, a comparatively more common pathologic entity of the distal extremities, occurs when tissues are exposed to freezing temperatures. Previously found in military populations, frostbite has become increasingly prevalent in the general population, leading to more clinical presentations to foot and ankle specialists. We present a review of the published data of acute foot compartment syndrome and pedal frostbite, with pathogenesis, treatment, and subsequent sequelae. A case report illustrating 1 example of bilateral foot, atraumatic compartment syndrome, is highlighted in the present report. The patient presented with changes consistent with distal bilateral forefoot frostbite, along with gangrenous changes to the distal tuft of each hallux. At admission and evaluation, the patient had increasing rhabdomyolysis with no other clear etiology. Compartment pressures were measured in the emergency room and were >100 mm Hg in the medial compartment and 50 mm Hg dorsally. The patient was taken to the operating room urgently for bilateral pedal compartment release. Both pathologic entities have detrimental outcomes if not treated in a timely and appropriate manner, with amputation rates increasing with increasing delay.

Compartment syndrome is an increased compartmental pressure that occludes appropriate vascular supply at the capillary level, resulting in myoneural necrosis (1–3). Compartment syndrome of the lower leg and foot is frequently associated with high-impact traumatic fractures, soft tissue compromise, arterial reperfusion injury, and procedural positions, including lithotomy (3–8). Compartment syndrome of the foot has also been described secondary to atraumatic causes such as infection and repetitive micro-trauma, including aerobics, running, and basketball (9–13).

Acute compartment syndrome of the foot can be associated with crush injuries, including motorcycle accidents, with an incidence rate of 2% to 6% (3,14). Approximately 23% will be found with nonfracture-associated soft tissue injuries (6). This uncommon clinical pathologic entity can result in several long-term sequelae, including digital contractures of the short flexors known as "Volkmann's contractures," which were originally described in the hand and most commonly accompany calcaneal fractures in the foot (14–18). Compartment syndrome of the foot was largely underappreciated in medical reports until the works by Myerson (15) and Fakhouri and Manoli (2), outlining the anatomy and surgical approaches for pedal compartment decompression.

In contrast, an often-encountered disease affecting the foot and ankle is thermal necrosis secondary to frostbite. Frostbite is the result of freezing of the tissue and is often found in the extremities; however, it can occur in any area exposed to freezing temperatures. Once mainly found in military personnel, frostbite has become a frequently treated abnormality found in the general population (19). Frostbite typically presents after cold exposure from weather conditions but has also been described with the application of ice packs and cryotherapy after injury and surgery (20,21). Frostbite is typically a clinical diagnosis, the severity of which is often not apparent for weeks after

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the initial injury and rewarming period (22). Frostbite is most commonly associated with a spectrum of symptoms, ranging from an area of central whitening to complete necrosis of the muscle and bone with tissue loss. Short-term complications include infection, pain, gangrene, and hyperhidrosis. More long-term complications include an increased risk of repeat cold injury, scarring, arthritis, tissue atrophy, peripheral neuropathy, and the need for surgical amputation (19). Rarely reported is the subsequent development of compartment syndrome, as highlighted in the presented case report. Only isolated case reports have correlated frostbite with the acute onset of compartment syndrome (23).

The primary goal of the present work was to report on the pathologic entity and the effective treatment strategies for acute compartment syndrome of the foot. In addition, we report on the various etiologies and highlight a unique case of frostbite-induced compartment syndrome of bilateral feet at a level 1 trauma center.

Materials and Methods

Search Criteria

We performed a literature review using MEDLINE® via PubMed® (US National Institutes of Health, National Library of Medicine; available at: http://www.nlm.nih.gov/bsd/pmresources.html), without limitations, except to the English language. We used the keywords, "compartment syndrome," and "acute compartment syndrome," with the word "foot," resulting in 426 and 106 related reports, respectively. When "acute compartment syndrome of the foot" was combined with "review," the search revealed 21 associated studies. Combining these terms with "case report" identified 6 studies. The combination of "compartment syndrome" and "foot," with "frostbite" led to 2 studies. In addition, searching "compartment syndrome" with "foot" and "atraumatic" or "non-traumatic" resulted in only 5 studies, with 4 directly affecting the foot and 1 the posterior muscle group.

One of us (R.A.B.) reviewed the abstracts for the 134 studies relating to acute, atraumatic compartment syndrome of the foot and the associated case reports for relevance. This process yielded approximately 23 studies. We excluded studies requiring translation and those related to exertional compartment syndrome, compartment syndrome of the leg, and pediatric compartment syndrome.

Results

Anatomy

Understanding the fascial anatomy of the foot is critical to the diagnosis and decompression treatment of compartment syndrome. The number of compartments in the foot has varied from 3 to 10 in the reported data (2,3,24,25). The 3 major compartments (medial, central, and lateral) run longitudinally from proximally to distally (3,26). Manoli and Weber (25) reported one of the most commonly cited works describing the compartment levels of the foot in cadavers in 1990. They performed infusion techniques to demonstrate the presence of 9 total compartments of the foot, with special attention to the medial compartment after calcaneal fractures (2,25). They divided the central compartment, highlighted the smaller dorsal interossei compartment and advised on new methods for decompression. Reach et al (26) discovered an additional 10th compartment, which contains the skin (Table). The hindfoot compartment is also identified as the "calcaneal compartment" and contains the quadrates plantae muscle. This compartment communicates with the deep posterior leg compartment and includes the neurovascular structures of the posterior tibial, lateral plantar nerve, artery, and vein and, occasionally, the medial plantar nerve. The 5 forefoot compartments include 4 interosseous compartments with the interossei muscles and the adductor compartment consisting of the adductor hallucis. The remaining 3 full-length compartments are the medial, lateral, and superficial compartments (9). The medial compartment contains the flexor hallucis and abductor hallucis muscles, and the lateral compartments contain the abductor digiti quinti and the flexor digiti minimi muscles. The superficial compartment holds the flexor digitorum brevis and the 4 lumbrical muscles, the tendons to the flexor digitorum longus, and, sometimes, the medial plantar nerve (9,18).

Frostbite can affect any of these compartments with varying severity. Initially presenting distally on the digit, injury can cause pressure to increase, extending to the interossei and medial compartments. Frostbite was classified by Cauchy et al (27) into 4 different degrees of severity, ranging from superficial skin damage to deep full-thickness damage to bone.

Pathophysiology

Frostbite most often affects the fingers, toes, ears, and nose as a result of exposure to subfreezing temperatures. The tissue injury that occurs in frostbite has been attributed to 2 processes. First, cell death occurs during exposure, which results in progressive dermal ischemia, causing necrosis (19). Cell death during the exposure period is due to ice crystal formation. Extracellular ice formation damages the cell membrane, causing electrolyte abnormalities and initiating cell death. The intracellular ice crystal formation causes cell death through mechanical cell disruption (19).

The progressive ischemia occurring after cold exposure has been thought to be the result of increasing inflammatory mediators, such as prostaglandin F2 and thromboxane A2. These mediators subsequently increase edema, decrease dermal blood flow, and lead to endothelial damage (28). Rewarming during treatment can cause emboli in the microvascular network, resulting in tissue death secondary to thrombotic hypoxia (29). Some evidence has shown that control of these mediators can decrease tissue death after the rewarming period (30).

Compartment syndrome develops from vascular occlusion secondary to increased pressure within a muscular compartment, resulting in ischemia of the muscle and nervous tissue in the affected region with subsequent dysfunction or permanent damage. Increased compartmental pressures can be caused by internal bleeding, often secondary to trauma, a commonly associated inciting event (3,25). Tissue damage is caused not only by direct vascular occlusion, but also by apparent occlusion from an abnormal pressure gradient that results in tissue ischemia and capillary wall damage with associated increase in permeability that further increases the edema. The increased edema can further occlude venous return, reducing arterial inflow and causing more ischemia, resulting in a vicious circle and large increases in intracompartmental pressures (31). When this circle occurs, significant and potentially permanent loss of function to nerve and muscle can develop in the involved compartment or compartments.

Clinical Diagnosis of Compartment Syndrome

A timely and appropriate diagnosis is imperative to prevent systemic and local effects related to compartment syndrome. Classically, the 5 P's associated with compartment syndrome include pain (out of proportion), paralysis, paresthesia, pallor, and pulselessness (32,33). In most cases, not all symptoms will be present during serial examinations of the patient. Some investigators have reported that pain out of proportion compared with the extent of the injury is typically the "cardinal" presenting symptom of acute compartment syndrome (33). Sensory and motor nerve damage can present as paresthesia, followed by paresis and end-state paralysis. Paresthesia can be evaluated using pinprick, 2-point discrimination, and light touch tests (33). End-stage motor dysfunction is indicative of permanent function loss. The most significant late-stage finding is pulselessness with color changes affecting the limb. In the patient highlighted in the present report, the