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Non-Necrotizing Streptococcal Cellulitis as a Cause of Acute, Atraumatic Compartment Syndrome of the Foot: A Case Report

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ABSTRACT

Acute compartment syndrome is widely accepted as a surgical emergency. Most cases of acute compartment syndrome occur after high-energy trauma, especially crush injuries. We present a unique case of acute, atraumatic compartment syndrome of the foot associated with infectious cellulitis. A 53-year-old male, with a medical history significant for human immunodeficiency virus, presented to the emergency department secondary to an insidious onset of intense foot pain, swelling, and an inability to bear weight on the affected extremity. He had no history of recent trauma. He was admitted to the hospital because of a suspected infection and subsequently was given intravenous antibiotics. During the admission, he developed a severe infection, and blood cultures demonstrated growth of group A streptococcus. No abscess or hematoma was identified on magnetic resonance imaging or during exploratory surgery. The findings from intraoperative cultures were negative. Despite proper medical care for his infection, the lower extremity pain worsened; therefore, compartmental pressures were obtained at the bedside. Multiple compartment pressures were measured and were >40 mm Hg. Compartment syndrome was diagnosed, and the patient was taken to the operating room for emergent fasciotomies. Surgical release of the medial, lateral, interosseous, and adductor compartments revealed copious amounts of serosanguinous drainage. Again, no definitive hematoma or purulence was identified. The patient's symptoms resolved after the fasciotomies, and he healed uneventfully. Our case highlights the need to consider acute compartment syndrome in the differential diagnosis for pain out of proportion to the clinical situation, even when a traditional etiology is absent.

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Compartment syndrome occurs when the intracompartmental pressure is increased within a closed space. This increased pressure is typically caused by a buildup of fluid, from either bleeding or edema (1). Two types of compartment syndrome have been reported: acute and chronic. Acute compartment syndrome has often been associated with high-energy trauma, especially crush injuries (2,3). Other causes include non–high-energy trauma, such as burns, animal bites, muscle tears, and atraumatic causes, such as bleeding disorders and tight circumferential dressings. If left untreated, acute compartment syndrome can lead to debilitating side effects, including neurovascular damage and rapid tissue compromise. Thus, acute compartment syndrome is widely accepted as a surgical emergency (4–6). Chronic compartment syndrome, also referred to as exercise-induced compartment syndrome, is more commonly diagnosed and typically occurs in the lower extremity. Chronic compartment syndrome occurs from a temporary increase in the intracompartmental pressures as a response to increased blood flow to the muscles during activity.

Compartment syndrome of the foot has been an infrequently identified, incidental finding (1,2,7–10). Acute, atraumatic cases are considered extremely rare in the lower extremity and, therefore, run the risk of being overlooked. We present a unique case of a patient with acute, atraumatic compartment syndrome of the foot due to circumferential non-necrotizing cellulitis.

Case Report

Clinical Presentation

A 53-year-old male presented to the emergency department in November 2012 with an insidious onset of severe left foot pain, swelling, and an inability to bear weight on the affected limb. He

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denied any history of injury or acute trauma. He had presented to his primary care physician 1 week earlier for heel pain, which was diagnosed as plantar fasciitis, and he had been recommended to see a specialist for additional care. The symptoms became markedly worse before his scheduled follow-up appointment, and he presented to the emergency department and was admitted to the hospital. When questioned, he rated his pain as >10, on the 0 to 10 numeric pain rating scale (11). His medical history was significant for dyslipidemia and human immunodeficiency virus. His human immunodeficiency virus was well-controlled with the combination drug efavirenz/emtricitabine/tenofovir disoproxil fumarate, with a CD4 T-lymphocyte count of 561 (healthy range 500 to 1200) cells/mm^3, an undetectable viral load, and no recent opportunistic infections.

The podiatry service was consulted on the second day of admission. During our physical examination, the patient was febrile and tachycardic, with a maximum oral temperature of 103.5°F and a maximum heart rate of 135 beats/min. His dorsalis pedis and posterior tibial pulses were palpable bilaterally, with marked nonpitting edema over the dorsal left foot and anterior ankle region. Increased warmth of the dorsal foot and anterior ankle was also present compared with the contralateral limb. The aspects of the neurologic examination that were performed, including light touch and proprioception, showed normal findings. However, 2-point discrimination and other more specific neurologic testing for compartment syndrome were not completed initially, secondary to a lack of consideration for compartment syndrome. Diffuse pain was present on palpation of the dorsal foot and anterior ankle, with pain also experienced with passive range of motion of the pedal joints. His muscle strength could not be assessed secondary to guarding; however, the patient was able to passively flex and extend his toes. Significant erythema was also present over the left foot and ankle. His initial leukocyte count was 8.2 × 10^9/L in the emergency department, but this had increased to 14.2 × 10^9/L within the first 24 hours of admission. The C-reactive protein level and erythrocyte sedimentation rate was elevated at 71 mg/L and 18 mm/hr, respectively. The prothrombin time was 13.0 seconds and the international normalized ratio was 1.3; the partial thromboplastin time was not obtained. Blood cultures were obtained on the second day of admission and demonstrated growth of group A *Streptococcus pyogenes*. Our hospital sepsis protocol was initiated because 3 of 4 systemic inflammatory response syndrome criteria were positive and bacteremia was present. Plain film radiographs of the foot and ankle obtained in the emergency department revealed diffuse soft tissue swelling with no soft tissue emphysema (Fig. 1). Magnetic resonance imaging with contrast revealed marked soft tissue swelling, skin thickening, and dense subcutaneous edema about the ankle, hindfoot, and dorsum of the midfoot (Fig. 2). Small joint effusions were also noted along the posterior subtalar joint and dorsal talonavicular joint. Considering the clinical, laboratory, and imaging findings, this patient was treated using a working diagnosis of cellulitis. The differential diagnosis at this time also included septic arthritis and inflammatory arthropathy.

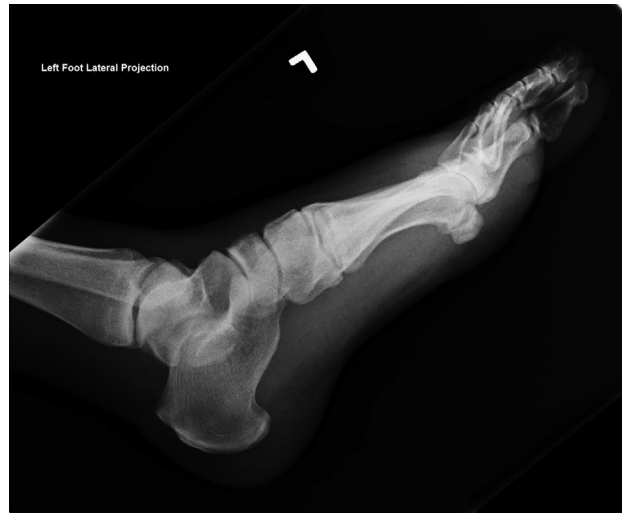

Fig. 1. Lateral radiograph of left foot demonstrating soft tissue edema.

Treatment and Surgical Technique

The initial antibiotic treatment consisted of intravenous ceftriaxone (1 g every 24 hours) for the first 3 days, which was then switched to intravenous clindamycin (900 mg every 8 hours) on the basis of the blood culture sensitivities. Analgesia consisted of oral hydrocodone/acetaminophen 5 mg/325 mg every 4 hours, as needed, and intravenous hydromorphone (2 mg every 4 hours) at scheduled intervals. After reviewing the magnetic resonance imaging studies, we recommended continuation of medical therapy with no surgical intervention. An infectious disease specialist was also consulted. Over the next 2 days, the patient's pain worsened and became unresponsive to the previously effective analgesic regimen. Owing to the patient's worsening clinical condition, we determined that surgical exploration was warranted.

On day 5 of the hospital admission, the patient underwent surgical exploration of the left foot and ankle. He was placed on the table in the supine position, and general anesthesia was administered. Exploration was directed at the fluid accumulations seen on the magnetic resonance imaging scans and the areas of most severe pain reported by the patient. After sterile preparation, an attempt was made to perform percutaneous aspiration of the subtalar joint. An inadequate amount of synovial fluid was withdrawn; therefore, a 2-cm oblique incision was made overlying the sinus tarsi. The incision was deepened using blunt dissection to the level of the deep fascia, and a hemostat was used to enter the subtalar joint, expressing a serous drainage. A swab was used to collect synovial fluid from the joint for aerobic and anaerobic culture with sensitivity testing. The peroneal tendon sheath, near the base of the fifth metatarsal, was

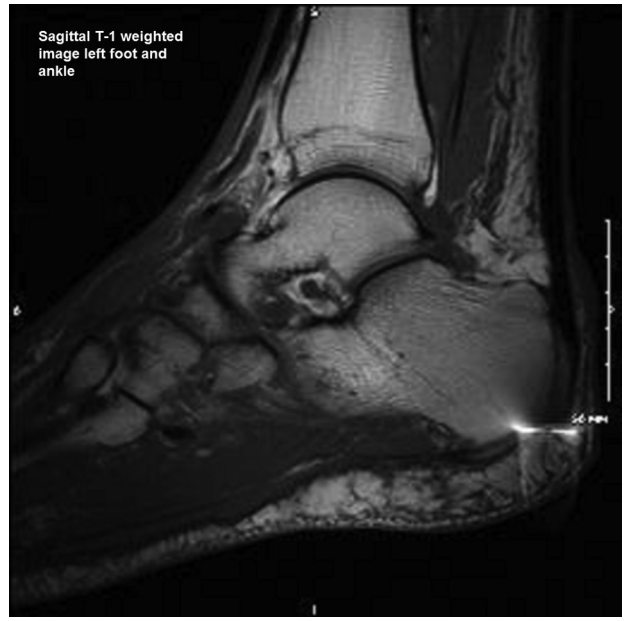

Fig. 2. Sagittal T_1-weighted magnetic resonance image illustrating left dorsal foot and anterior ankle soft tissue edema.

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