Atypical Presentation of Fat Embolism Syndrome After Gunshot Wound to the Foot

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Abstract

A 42-year-old man sustained a gunshot wound to the foot, which caused multiple fractures of the tarsal bones. Over the course of 6 hours, he became obtunded and began to experience respiratory compromise and neurologic symptoms. On magnetic resonance imaging, he was found to have innumerable tiny infarcts consistent with fat embolism syndrome. He was placed in a spanning external fixator. The patient remained in a coma for 6 weeks after injury but gradually regained function.

Fat embolism syndrome is classically described as resulting from injury to the diaphysis of a long bone, but it can occur secondary to trauma of the foot. Neurovascular status must be continually evaluated in the setting of any bony trauma, and index of suspicion should remain high when neurologic deficits are encountered.

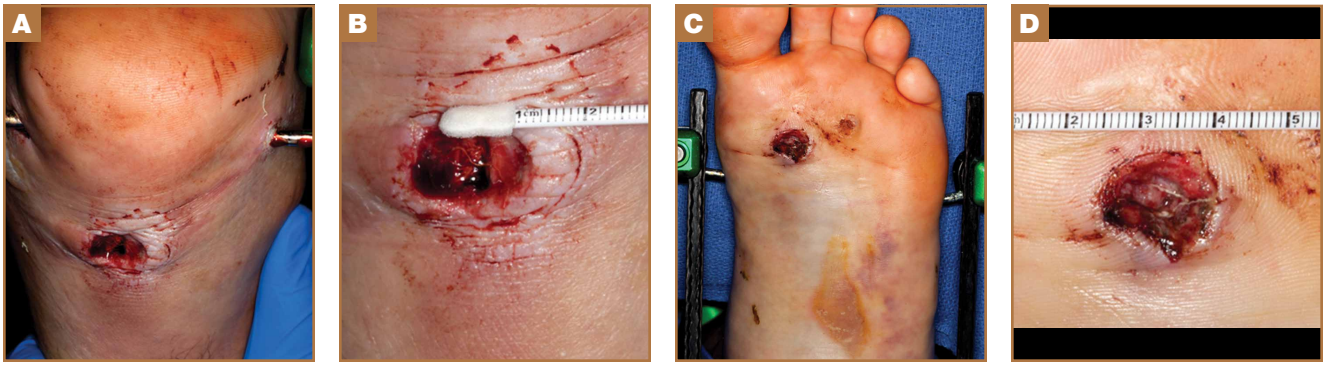
Fat embolism syndrome (FES) is a rare complication reported primarily after long bone fractures, with an incidence of 0.3% to 2.2%.[1-3] It is most commonly caused by trauma and is thought to result from movement of bone fragments or to occur during intramedullary reaming.[1] Both of these factors lead to a distortion of the bone marrow cavity, allowing marrow and fat to enter the circulatory system.[1] Although the true pathophysiology remains poorly understood, it is possible that, once in systemic circulation, the fat particles become lodged in the vascular system, inciting an inflammatory response, leading to organ dysfunction via mechanical or biochemical processes.[4] Typically, the diagnosis is made after clinical features are observed, including hypoxemia, petechial rash, and cerebral signs not related to a head injury or other conditions.[5,6]

Although FES is an uncommon complication after traumatic injuries, mortality after FES in a recent study was reported to be 10%.[1] FES is most commonly seen after fractures of the femur and tibia, although cases have been described involving fractures of the radius, ulna, and humerus.[1,3] We present an atypical case of cerebral FES after multiple fractures of the foot; to our knowledge, such a case has not been reported in the English-language literature. The patient provided written informed consent for print and electronic publication of this case report.

Case Report

A 42-year-old man was hunting with his son when he was accidentally shot in the left foot with a .270-caliber rifle bullet at close range. The patient sought care at a local hospital and,

Figure 1. Images of (A, B) entrance wound on the posterior aspect of left foot through the Achilles tendon and (C, D) exit wounds on the plantar aspect of left foot.



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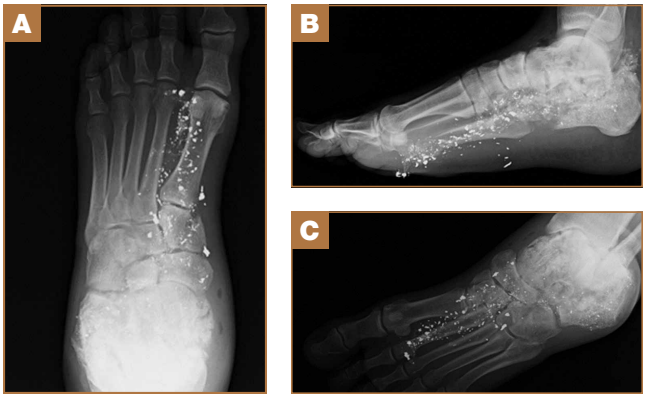

Figure 2. (A) Anteroposterior, (B) lateral, and (C) oblique radiographs of left foot.

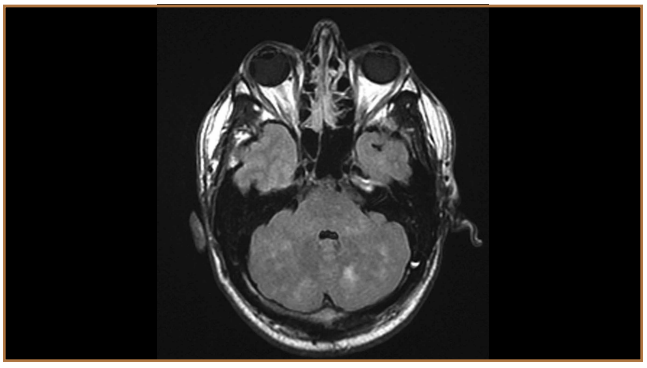

Figure 3. Axial T2-flair magnetic resonance imaging of the brain, showing diffuse tiny infarcts in the "starfield" pattern.

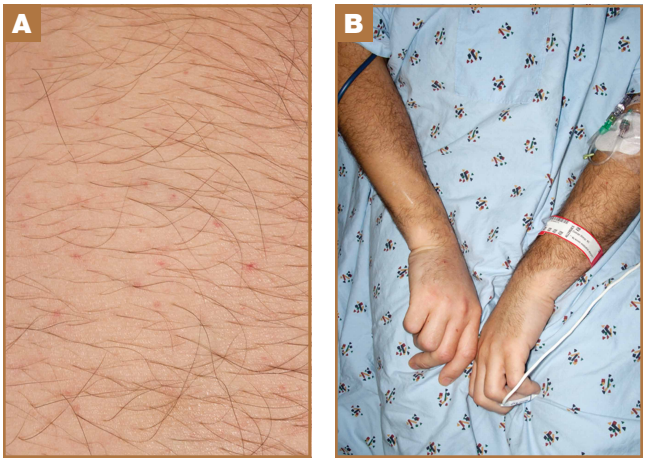

Figure 4. (A) Physical examination findings of subtle petechial rash and (B) extensor posturing because of brain injury.

in the ensuing 3 hours, his mentation appeared normal. He reported pain and numbness distal to the injury in the tibial nerve distribution, but he remained vascularly intact, alert, and oriented. He was given 7 mg of hydromorphone hydrochloride over 2 hours for pain control and was transferred to our hospital via ambulance approximately 6 hours after injury. Upon arrival, he was noted to be extremely sedated and obtunded, responding only to pain with spontaneous eye opening. He was unable to follow commands. He was given 1.2 mg of naloxone intravenously to reverse what was presumed to be acute opioid intoxication; however, his mental status did not improve.

On examination, the patient was noted to have a small entrance wound through the Achilles tendon (**Figures 1A, 1B**) and an exit wound on the plantar aspect of the foot near the heads of the first and second metatarsals (**Figures 1C, 1D**) with minimal bleeding and no gross contamination. There was significant edema on the medial and proximal aspects of the left foot, 3+ dorsalis pedis pulse, and a capillary refill of 4 seconds. Radiographs showed traumatic fracture deformities of the calcaneus, navicular, medial cuneiform, and first and second metatarsal bases, as well as an intra-articular fracture deformity of the left talus extending to the talar dome (**Figures 2A-2C**). Neurologic examination could not be reliably obtained because of the patient's mental status. He was determined to be unstable for immediate surgery, and his left leg was splinted pending neurologic evaluation.

The patient's oxygen saturation was 94%, and his temperature was 38.2°C (100.76°F). Although his heart rate was in the 90s upon arrival, he became tachycardic over the next 4 hours, with heart rate ranging from the 110s to 130s; he remained tachycardic for approximately 72 hours. Laboratory values upon arrival showed a hemoglobin value of 12.8 g/dL and platelets of 249,000/μL. He developed anemia and thrombocytopenia within 72 hours of the injury, with a low of 6.6 g/dL and 88,000/μL, respectively, by postinjury day 4. Computed tomography of the head, electroencephalography, urine drug screen, and lumbar puncture were unremarkable. The patient never became hypoxemic. Within 14 hours after injury, he was completely comatose with extensor posturing. In the intensive care unit (ICU), the patient was intubated for airway protection.

The next day, the patient underwent magnetic resonance imaging (MRI) of the brain, which showed innumerable tiny infarcts throughout cerebral hemispheres, cerebellum, and brainstem in a characteristic "starfield" pattern on T2-weighted images (**Figures 3**). This was radiographically consistent with fat emboli related to the left lower extremity gunshot wound. An echocardiogram showed small right-to-left shunt and a possible intrapulmonary shunt, although this was never confirmed. The echocardiogram was technically challenging secondary to his persistent tachycardia. He also developed a subtle petechial rash (**Figure 4A**).

The patient underwent percutaneous gastrostomy-tube placement for nutrition on postinjury day 4 and remained intubated, unable to protect his airway, and nonresponsive with extensor posturing (**Figure 4B**). He was also taken to the operating room for spanning external fixator placement on postinjury day 3 to restore calcaneal height and length as well as foot stability (**Figures 5A, 5B**).

The patient was treated with supportive care and was discharged from the hospital in a comatose state on hospital day 17 to a rehabilitation facility. He began to emerge from the coma 6 weeks after injury, and his external fixator was removed and a cast applied to his lower extremity. His entrance and exit wounds healed as expected. Initial agitation was treated with propranolol and quetiapine. Because he continued to have difficulty with spasticity and increased tone, he was given botulinum toxin type A injections in the pectoral muscles, biceps, and forearms. He made continued and rapid improvement in response to intensive multidisciplinary therapy and returned home 4½ months after injury. Eight months after the injury, he is now walking independently with a cane and independent with his activities of daily living. Unfortunately, he has substantial pain in his foot, which appears to be a combination of both neuropathic and posttraumatic arthrosis causes. He is undergoing consultation for a possible amputation. Radiographs show consolidation of the hind and midfoot fractures with retained bullet fragments (**Figures 6A-6C**). He continues to receive multidisciplinary care to address cognitive limitations and is making progress.

Discussion

FES is a life-threatening disease affecting multiple organ systems.[7] Classically, the pulmonary, central nervous, and dermatologic systems are affected.[5,6,8] While FES is most recognizable after long bone fractures and orthopedic procedures involving the intramedullary canal, to our knowledge, FES after gunshot wound and concomitant fractures of the foot has never been reported.

The syndrome is defined by major and minor criteria as outlined by Gurd.[5] Major criteria include hypoxia, deteriorating mental status, and petechiae. This case represents a somewhat atypical presentation of FES, because dermatologic manifestations and pulmonary compromise were subtle. The minor criteria consisting of tachycardia, fever, anemia, and thrombocytopenia were present in our patient, although at different phases during the progression of the syndrome. This emphasizes the difficulty in diagnosing FES because the symptoms do not occur simultaneously.

In the classic syndrome, after an initial asymptomatic interval of 12 to 72 hours, pulmonary, neurologic, and/or dermatologic changes usually ensue.[9] Altered mental status, including headache, confusion, stupor, coma, rigidity, or convulsions, has been documented in 86% of patients.[10] In our case, the neurologic symptoms presented earlier, at around 6 hours after injury, and respiratory symptoms, including hypoxia, tachypnea, and dyspnea, reported in 75% of cases,[2,11] did not occur at all. In fact, continued intubation was only required in this case for neuromuscular airway protection. Classic dermatologic manifestations, a reddish-brown nonpalpable petechial rash diffusely covering the upper torso and extremities, normally appears within 12 to 36 hours.[12,13] Nevertheless, in our case, these findings were subtle compared with others previously reported.[14,15] In fact, despite being seen by numerous physicians, including neurologists and ICU intensivists, only the orthopedists' notes made reference to this modest finding (**Figure 4A**).

Further complicating the diagnosis is that, during the onset of symptoms, patients are typically victims of polytrauma and/or routinely given narcotics to help with significant pain. Therefore, it is appropriate to rule out opioid overdose and other metabolic sources of mental-status change. This can be done fairly expeditiously with laboratory testing and narcotic reversal. After these have been eliminated, FES should be considered in a patient with rapid neurologic deterioration, because a delay in treatment can affect outcomes.[2,4,16]

Because continuous showering of emboli to the brain and other organs occurs without fracture stabilization, rapid diagnosis with high clinical suspicion of FES is essential and can be aided immensely with MRI. In fact, MRI is the most sensitive test for this diagnosis and correlates with clinical severity of brain injury.[17] T2-weighted images show regions of high-signal intensity and "starfield" pattern, which are sensitive markers for FES (**Figure 3**).[18] These tests can be done concomitantly with a well-splinted extremity, and definitive stabilization should be carried out promptly because early splinting and fixation of orthopedic fractures improves outcomes.[17]

Perhaps the most important reason to make an expeditious diagnosis is to help counsel families, who are undoubtedly in shock

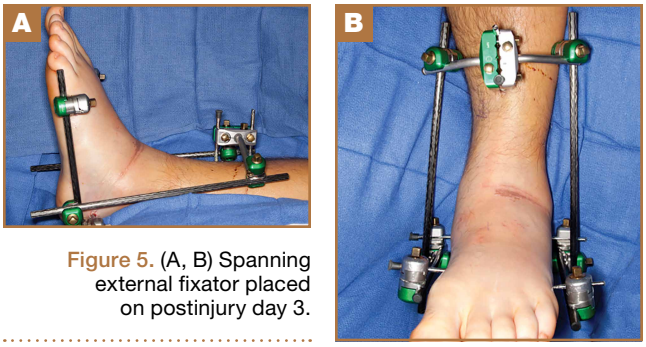

Figure 5. (A, B) Spanning external fixator placed on postinjury day 3.

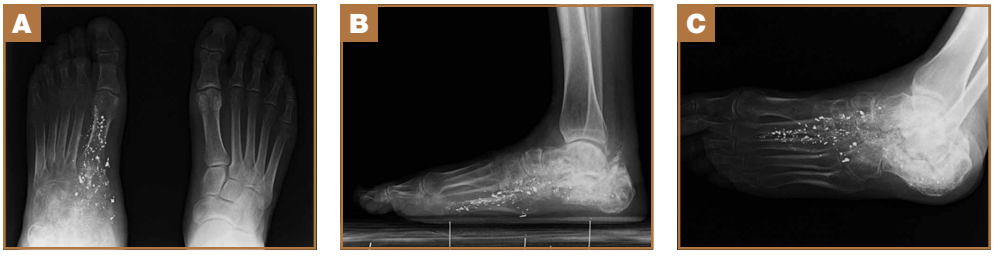

Figure 6. Eight-month postinjury (A) anteroposterior, (B) lateral, and (C) oblique radiographs with consolidation of fractures and retained bullet fragments.

and disbelief. Recovery times can vary widely, with the patient often continuing to regain cognitive and motor function over the course of months to years.[2] Without knowledge of signs of improvement in neurologic outcome, families cannot be accurately counseled regarding potential for recovery. The practicing orthopedist should be aware of this disorder, because initial neurologic deterioration may seem hopeless. Furthermore, supportive care should be initiated early with multidisciplinary teams and extensive rehabilitation because these offer the best outcomes in patients with FES.[4,18] Although our patient continues to have cognitive impairment, his recovery in the preceding 8 months has been aided by rapid diagnosis and multidisciplinary care and should offer hope to other patients faced with this situation.

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This paper will be judged for the Resident Writer's Award.