

Clinical Reasoning: Patient With Prior Spinal Cord Injury Who Developed Altered Mental Status After a Fall

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Abstract

An 18-year-old man with a history of complete traumatic spinal cord injury (SCI) at C5-C7 3 years before presented with unresponsiveness and hypoxia after a fall. There were no overt signs of bruising or swelling. After extensive and unrevealing initial workup, MRI brain without contrast showed numerous diffusely scattered punctate foci of diffusion restriction and evidence of numerous microhemorrhages. A full body skeletal survey revealed mildly affected, non-displaced, incomplete fractures in the distal femoral metaphyses bilaterally. This case presentation discusses specific considerations for patients with SCI, reviewing the differential diagnosis, workup, and management of altered mental status after minor falls or other trauma in this population.

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Section 1

An 18-year-old man presented to the hospital with altered mental status after a fall. He experienced a complete traumatic spinal cord injury (SCI) at C5-C7 3 years before presentation. At baseline, there was some preserved strength in the proximal arms, though he required help with transfers. He was able to feed himself with adaptive utensils. His bilateral legs were plegic. He had autonomic dysreflexia and neurogenic bowel and bladder, but was cognitively intact. On the day of presentation, securement straps were around his legs when he fell while being moved from a sitting to standing position. His head struck the floor, but he did not initially lose consciousness. Thirty minutes after injury, he appeared uncomfortable, reporting difficulty in breathing and tingling in his hands. Family administered 10 mg of oral diazepam, which he took regularly for anxiety.

On arrival of Emergency Medical Services, oxygen saturation was 78%, so supplemental oxygen was administered. The patient arrived to the emergency room unresponsive, not opening his eyes nor following commands, with only flexor

movements of bilateral arms to noxious stimuli. There were no signs of bruising or swelling. He received the benzodiazepine antagonist flumazenil without improvement.

Arterial blood gas showed normal pCO₂ levels. Chest x-ray was unremarkable. Noncontrast head CT revealed no abnormalities and was stable from 3 years before. CT angiography of the head and neck and CT perfusion revealed no large vessel occlusion, dissection, or perfusion deficits to suggest acute stroke. CT cervical spine showed C4-C7 fusion with intact hardware. Rapid response EEG demonstrated beta activity consistent with known diazepam administration; no seizures or epileptiform activity. Urine toxicology screening revealed benzodiazepines, tetrahydrocannabinol, opiates, and cocaine. Family reported patient use of acetaminophen with codeine and marijuana, but no known history of cocaine or intravenous drug use. The patient was admitted to the Neuro-intensive care unit.

Questions for Consideration:

1. Where do his symptoms localize?
2. What is the differential diagnosis?
3. What are the most important next steps in management?

GO TO SECTION 2

Section 2

Coma and decorticate posturing localize to the midbrain, thalami, or bilateral hemispheres, so more global neurologic processes were initially considered. Naloxone was administered for possible intoxication with no change in examination. The toxicology service believed that the known medications and substances could not sufficiently explain all symptoms. Video EEG was obtained because seizure remained a consideration and revealed generalized slowing with periods of burst suppression. Postconcussive syndrome was considered unlikely, given the duration and severity of symptoms.

He subsequently developed worsened hypoxia and tachypnea, requiring intubation. Chest x-ray revealed aspiration pneumonia and bilateral pulmonary edema, prompting transthoracic echocardiography, which demonstrated an ejection fraction of 25% with global hypokinesis of both ventricles. Electrocardiogram revealed normal sinus rhythm and incomplete right bundle branch block. Laboratory studies showed a new leukocytosis of 20,400/ μ L (neutrophilic predominance and bandemia) and thrombocytopenia to 92,000/ μ L. Elevated procalcitonin, 2.64

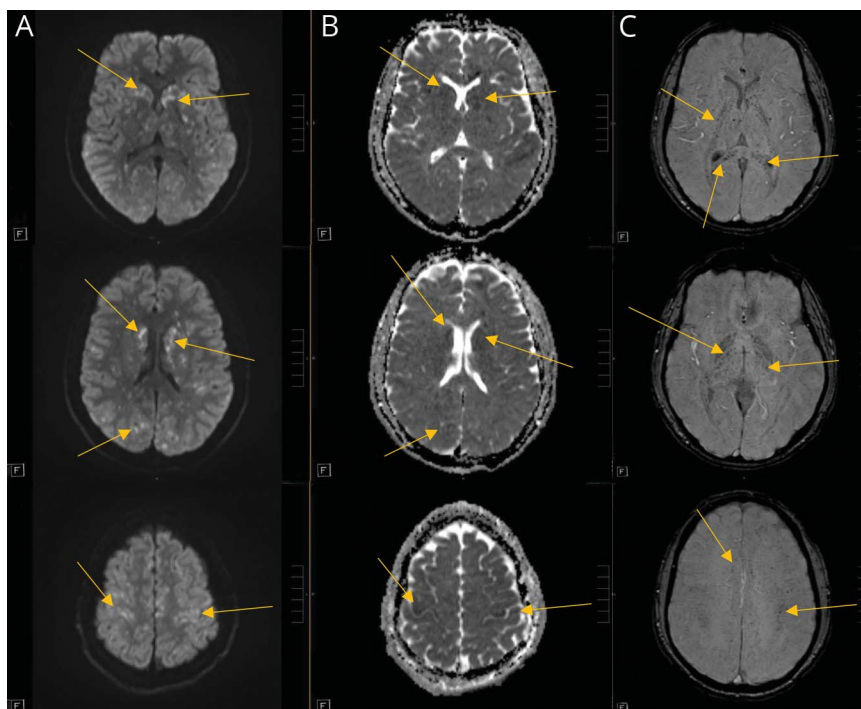
ng/mL (<0.1), suggested the presence of an infection. Empiric cefepime and vancomycin were initiated for possible sepsis. C-reactive protein to 42 mg/L (<10) and D-dimer more than 10,000 ng/mL (<255) suggested an inflammatory response. Cardiac injury was suggested by elevated brain natriuretic peptide, 142 pg/mL (<100), and troponin I, 0.32 ng/mL (<0.04). Urine culture revealed 20,000 CFU/mL *Klebsiella pneumoniae*; sputum and blood cultures were negative. CSF was normal, with 100 mg/dL of glucose, 51 mg/dL of protein, 1 nucleated cell, and negative cultures and meningitis-encephalitis panel.

Consistent with the initial possible localization of bilateral hemispheres, MRI brain without contrast on postfall day 1 revealed evidence of widespread bihemispheric injury, with diffusely scattered punctate foci of diffusion restriction (Figure 1) and evidence of numerous bihemispheric microhemorrhages (Figure 2).

Questions for Consideration:

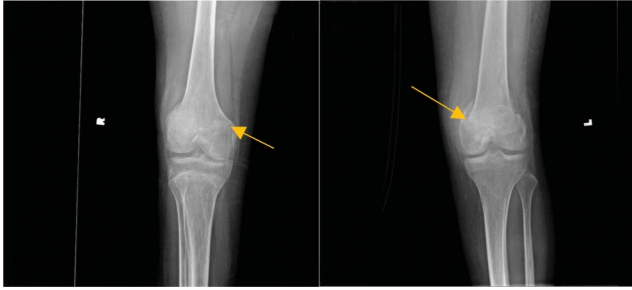
1. Given the MRI findings, what is the new differential diagnosis?
2. What should be the next steps in evaluation?

Figure 1 MRI Brain Showing Diffusion Restriction



Diffusion-weighted imaging hypertintensities (DWI, A) and apparent diffusion coefficient sequence hypointensities (ADC, B) showing diffuse punctate foci of diffusion restriction, with susceptibility-weighted imaging hypointensities (SWI, C) showing numerous microhemorrhages.

Figure 2 Bilateral Knee Radiographs



Radiographs of bilateral knees showing mildly impacted, nondisplaced, incomplete fractures in the distal femoral metaphyses.

GO TO SECTION 3

Section 3

MRI findings of punctate foci of restricted diffusion and microhemorrhages suggested a process involving small blood vessels, with the differential diagnosis including vasculitis, microangiopathic processes, or an embolic cause. Vasculitis appeared unlikely, given the acute time course. There were no schistocytes on peripheral smear to suggest thrombotic thrombocytopenic purpura or disseminated intravascular coagulation. The pattern of very small infarcts with diffuse punctate microhemorrhages was inconsistent with cardiac emboli, which would normally cause larger infarcts and not have bihemispheric microhemorrhages. Transesophageal echocardiogram revealed no intracardiac source of embolism or patent foramen ovale. Four-limb venous duplex studies revealed no evidence of deep venous thrombosis.

Air or fat emboli causing acute micropulmonary emboli followed by brain emboli seemed plausible, given that respiratory complaints preceded the acute neurologic change after the patient's fall. However, initially, no embolic source was evident. There were no overt signs of bruising to suggest fracture. On hospital day 2, petechiae developed in the axilla and groin. A skeletal survey to evaluate for occult fracture revealed new cortical step-offs along the lateral aspects of the distal femoral metaphyses bilaterally, suspicious for mildly affected, nondisplaced, incomplete fractures. Subsequent femur and tibia fibula x-rays confirmed these findings. Orthopedic surgery placed knee immobilizers. The patient was diagnosed with fat embolism syndrome (including cerebral fat embolism). Although limited data for benefit are available, methylprednisolone (100 mg IV) was administered daily for 1 week, followed by a 1-week taper. His course was complicated by cerebral edema and hydrocephalus, requiring osmotherapy and external ventricular drain placement.

During hospitalization, the patient remained in a minimally conscious state, ventilator dependent, requiring tracheostomy and percutaneous gastrostomy tubes. After staying in an acute rehabilitation facility and with ongoing therapies, he is now alert and oriented, using an alphabet board, communicating by mouthing words and working on sustaining phonation with the tracheostomy on room air. He has an intrathecal baclofen pump for spasticity in the arms. He can pull himself upright but requires maximal assistance to maintain posture.

Discussion

Patients with SCI are known to be at a high risk for long bone fractures, which occur with an annual incidence rate of 2%–6% in this population.^{1,2} In this case, x-rays revealed only mildly affected, nondisplaced fractures, raising the question of whether patients with SCI are at a risk of fat embolism from even minor fractures.

After SCI, patients become vulnerable to fracture due to severe reduction in bone density below the level of the lesion.

Bone loss is characterized by demineralization of the trabecular lattice structure in the distal femur and proximal tibia epiphyses, which is replaced by fatty marrow.³ Factors influencing bone mass in these patients include the degree and location of injury, muscle spasticity, age, sex, weight-bearing activity, and duration after injury.^{4,5} As in this case, common causes of fractures include wheelchair transfers, bumping unseen objects, and other low-effect activities; significant trauma is not necessary.^{1,2} The knee is often the first point of contact in these injuries, making it particularly susceptible to fracture.³ Bilateral femoral fractures increase the risk of fat embolism, with incidence shown to be 33% compared with 1%–3%, after single long bone fractures.⁶ Fat emboli usually develop within 24–72 hours of injury.⁶ Fat embolism syndrome (FES) occurs when fat emboli lead to multisystem organ failure, typically including pulmonary, dermatologic, and neurologic complications. Neurologic manifestations include headache, behavior changes, seizures, and altered consciousness. More rare manifestations include decorticate posturing, cerebral edema, hydrocephalus, and paroxysmal sympathetic hyperactivity.⁷ Systemic effects include fever, tachycardia, retinal involvement, jaundice, renal dysfunction, anemia, thrombocytopenia, and elevated inflammatory markers.⁸ In this case, fat emboli injury to the myocardium likely resulted in new heart failure.

One theory of fat embolism pathophysiology suggests that trauma increases intramedullary pressure, causing the bone marrow to enter venous sinusoids, releasing fat droplets into the veins that embolize and occlude blood flow in small vessels. Accordingly, an increased incidence of fat emboli would be expected after trauma in patients with SCI, given the increased bone marrow fat content.³ However, the data regarding fat embolism after minor trauma in patients with SCI is limited. Fat embolism has more commonly been reported in patients with Duchenne muscular dystrophy (DMD)—another population with significant loss of bone density replaced with fatty marrow. In one case series, 5 patients aged 14–23 years were found to experience FES with respiratory and neurologic symptoms after minor trauma. There are also reports of fatal FES after minor trauma among patients with DMD.^{9,10} Notably, none had clear fractures on imaging and/or autopsy.⁹

Cerebral fat embolism often presents as a self-limiting illness with eventual complete recovery. Supportive therapy and symptomatic management remain the mainstays of treatment. Corticosteroids are often used for the prevention of fat embolism, and some authors have reported benefit as treatment to limit the postinjury rise in free fatty acid levels and blunt the inflammatory response.¹¹ Other treatments have been proposed with limited or conflicting evidence, including anticoagulation, 20% sodium dehydrocholate, low molecular dextran, albumin, 5% alcohol glucose solution, dehydrating agents, cooling therapy, and hyperbaric oxygen.⁷ Unfortunately, poor outcomes are common, with a mortality rate of up to 10%.⁷ Thus, it is imperative to consider fat embolism

in patients at a high risk of decreased bone density—including patients with SCI—who present with fitting neurologic and respiratory symptoms after minor falls or trauma, even when no overt signs of trauma are present.

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Name	Location	Contribution
Alexa Dessy, MD	Department of Neurology, NYU Langone Health	Drafting/revision of the article for content, including medical writing for content; major role in the acquisition of data; analysis or interpretation of data
Dhristie Bhagat, MD	Department of Neurology, NYU Langone Health	Drafting/revision of the article for content, including medical writing for content; major role in the acquisition of data; and analysis or interpretation of data

Appendix (continued)

Name	Location	Contribution
Barry M. Czeisler, MD	Department of Neurology, NYU Langone Health	Drafting/revision of the article for content, including medical writing for content

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