Unconscious Patient After Elective Bilateral Total Knee Arthroplasty

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Case Description
We present the case of a 66-year-old coach of a national shooting team who had degenerative osteoarthritis in his both knees, but otherwise was healthy and took no regular medication. Neither he nor his family had any cardiovascular risk factors. He did not smoke or use alcohol. His ability to walk was restricted because of osteoarthritis.

He was scheduled for bilateral total knee arthroplasty in March 2013. Before surgery, the orthopedist found the range of motion in the knees to be good, no flexion contracture was observed, and flexion was 120 degrees. Both knees were stable; the varus deformity was 3 degrees. The bone quality in the radiographs was normal. Auscultation of heart and lungs was normal; ECG and laboratory values were within normal limits.

The operation was performed under combined spinal and epidural anesthesia, and vital signs were stable throughout the operation, which lasted for 3.5 hours. Normal, cemented, cruciate-retaining knee replacement was performed using intramedullary guides in femur and tibia in both knees. During the second knee surgery, the patient had a short period of confusion when the tourniquet was released. After the surgery, he was observed in the recovery room overnight. Blood pressure and oxygen concentration were normal, and enoxaparine 40 mg daily was started as thrombosis prophylaxis. In the morning, he spoke normally with his daughter and the anaesthesiologist.

He was moved to the ward. Thirty minutes later, he was found unconscious. He was extending his upper and flexing his lower limbs, and his head and neck were extended. The pupils were of equal size and reacted normally to light, but he had vertical nystagmus. The patient was sweating and had a fever of 38.5°C. Chest radiograph and brain and chest CT were normal.

After consulting the neurologist on call, the patient was transferred to the emergency room within the university hospital.

In the emergency room, the patient did not open his eyes, and his gaze was abnormal with the eyes moving from side to side. He did not speak or obey commands. He periodically extended his upper limbs, while flexing his lower limb. There was flexion to pain in all limbs, and the muscle tone was increased in the upper limbs. Glasgow Coma Scale score was 5. Babinski sign was present on both sides.

Next, a cerebral CT angiography was ordered, and the patient was intubated and sedated because of decreased level of consciousness.

Question 1
What would you suspect to find in the CT angiography? What further diagnostic evaluation would you consider?

Answer
One might have suspected a posterior circulation stroke and thrombosis of the basilar artery. CT angiography showed the basilar artery to be open.

To rule out a nonconvulsive status epilepticus, EEG monitoring was started, which showed burst suppression at the beginning of the recording. This was attributed to the general anesthetic (propofol) infusion. The patient received an intravenous load of fosphenytoin, and the EEG subsequently normalized. When basilar artery thrombosis and nonconvulsive status epilepticus had been ruled out, MRI of the brain was obtained with diffusion-weighted imaging, perfusion-weighted imaging, T2-weighted imaging, fluid-attenuated inversion recovery, and time-of-flight MR angiography. MRI showed dozens of small ischemic lesions bilaterally in both anterior and posterior circulation comprising cortex, cerebellum, brain stem, thalamus, and basal ganglion, with no intra- or extracranial vessel pathology.

Question 2
What stroke cause do you suspect and what further diagnostic evaluation is needed?

Answer
Cardioembolic mechanism was suspected because of multiple lesions in different arterial territories. Bedside transthoracal ECG was performed by a cardiologist, and no thrombi or
endocarditis was seen. Instead, pulmonary pressure seemed to be increased and pulmonary embolism was suspected. Pulmonary CT angiography showed pulmonary embolus in the right posterior lobe. The patient was anticoagulated and transferred to the stroke unit.

As there was no cardiogenic cause found and the MRI lesions were multiple, small, and located bilaterally in both anterior and posterior circulation with no intra- or extracranial vessel pathology, cerebral fat embolism was suspected. The patient was being treated in the stroke unit, intubated, mechanically ventilated, and sedated because he was restless. In 2 days, his platelet count decreased (120 E9/L), and he became anemic (hemoglobin, 8.9 g/dL; erythrocytes, 2.92 E12/L). Follow-up MRI 5 days later showed no new lesions or hemorrhage. Subsequently, the patient was extubated, and after 9 days in the stroke unit, he was transferred to general neurology floor for rehabilitation evaluation. His recovery was gradual; after 3 weeks, he was able to open his eyes spontaneously, answer questions with a yes, and follow simple commands such as showing his tongue and moving his toes. He was able to move all limbs, but could not lift them.

The patient was then transferred to a rehabilitation hospital. After 1 month, he was able to talk, stand up on his own, and walk without assistance. At that stage, he still had minor cognitive impairment and memory difficulties, but after 4 months, he was able to drive a car and return to his work.

**Discussion**

Cerebral fat embolism is a rare cause of stroke; however, it may be likely in patients with neurological deterioration after surgery. Fat embolism syndrome is a systemic inflammatory reaction of the microvasculature of the brain, lungs, and skin caused by small embolic fat globules from the bone matrix. Fat emboli are more common after bone fractures, with a frequency in the range of 0.9% to 2.2%, but can also be found after orthopedic procedures, including bilateral total knee arthroplasty, where the incidence was recently reported to be 0.17%. During surgery, fat passes into the venous system, then first to the right side of the heart and to the lung capillary bed, and finally into the brain, either through a patent foramen ovale, which our patient did not have, or arteriovenous anastomoses in the lung. The classical cerebral fat embolism triad includes hypoxemia, neurological abnormalities, and petechial rash. In addition, thrombocytopenia and anemia are common. Respiratory symptoms are most frequent intraoperatively or <12 to 72 hours. The rash is present up to 60% of cases, usually on the conjunctiva, oral mucous membranes, and skinfolds of neck and axillae.

Neurological manifestations range from drowsiness and confusion to coma and include limb weakness, aphasia, and pupillary abnormalities. Confusion and disturbances of consciousness may be secondary to systemic dysfunction as seen in hypoxia or hypotension, or, as in our case, caused by multiple ischemic lesions bilaterally in the thalamus and brain stem resembling clinically basilar artery thrombosis or nonconvulsive status epilepticus. Noncontrast CT is often normal, and the work-up includes MRI. The chest radiograph classically shows multiple bilateral patchy areas of consolidation typically in the middle and upper zones, giving rise to a snowstorm appearance. Thrombocytopenia and unexplained anemia are common (37% and 67%, respectively), and blood and urinary analysis may show fat globules, although all of these are nonspecific signs.

Treatment is nonspecific and supportive, with adequate oxygenation and mechanical ventilation in some cases. Of principal importance is early resuscitation and minimizing the stress response and hypovolemia, as was done with our patient at the emergency room and the stroke unit. Fortunately, the severe neurological symptoms of cerebral fat embolism frequently resolve, as was the case with our patient who was able to return to work and reported no cognitive or behavioral changes.

**TAKE-HOME POINTS**

- Early diagnostic evaluation in stroke is critical for acute intensive care and proper rehabilitation.
- Fat embolism should be considered in patients with impaired consciousness after bilateral knee surgery.
- In general, the treatment of cerebral fat embolism does not differ from stroke management.
- Cerebral fat embolism can mimic life-threatening neurological illnesses, such as basilar artery thrombosis or nonconvulsive status epilepticus.

**Disclosures**

None.

**References**


**KEY WORDS:** embolism, fat
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