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 in 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
endoendocarditis 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 extracra-
nial vessel pathology, cerebral fat embolism was suspected.
The patient was being treated in the stroke unit, intubated,
mechanically ventilated, and sedated because he was rest-
less. 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 spontane-
ously, answer questions with a yes, and follow simple com-
mands 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 cog-
nitive 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 inflam-
matory 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 anaemia 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 skin-
folds 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 con-
sciousness may be secondary to systemic dysfunction as seen
in hypoxia or hypotension, or, as in our case, caused by mul-
tiple ischemic lesions bilaterally in the thalamus and brain
tem stemming clinically basal artery thrombosis or non-
convulsive status epilepticus. Noncontrast CT is often normal,
and the work-up includes MRI. The chest radiograph classi-
cally shows multiple bilateral patchy areas of consolidation
typically in the middle and upper zones, giving rise to a snow
storm appearance. Thrombocytopenia and unexplained ane-
mia 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 oxy-
genation and mechanical ventilation in some cases. Of prin-
cipal 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 throm-
  bosis or nonconvulsive status epilepticus.

Disclosures

None.

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Unconscious Patient After Elective Bilateral Total Knee Arthroplasty
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