Netherlands Journal of Critical Care
Submitted January 2017; Accepted April 2017

CLINICAL IMAGE

Cerebral fat embolism

E.H.J. Hamoen[1], R.A. Waalewijn[2], J.W. Gratama[3], B. van Kooten[4], P.E. Spronk[5], A. Braber[5]
Departments of [1]Surgery, [2]Cardiology, [3]Radiology, [4]Neurology, and [5]Intensive Care, Gelre Hospital, Apeldoorn, the Netherlands

Correspondence
A. Braber – a.braber@gelre.nl

Keywords - **trauma, fat embolism, MRI**

Case

An 83-year-old man was found with a Glasgow Coma Score of 6 (E2M3V1), one day after a partial hip replacement because of a femoral neck fracture after high-energy trauma one day earlier. Before surgery, no abnormalities in consciousness were present. He did not receive any opiates during the 12 hours before the event. The oxygen saturation was 93% with 3 litres O2/min. As it was not immediately clear that the patient had not received any opioids, 0.4 mg of naloxone was administered intravenously. However, this did not alter the comatose condition. Blood tests did not show any metabolic disturbances, results of an arterial blood sample were within the normal range. A computed tomography (CT) and CT-angiography scan of the brain showed no abnormalities. The patient was admitted to the intensive care unit for monitoring. Because his condition did not improve, an MRI scan of the brain was performed. This demonstrated diffuse small dot-shaped diffusion restrictions, predominant in the white matter of both hemispheres *(figure 1)*. The clinical course resulting in a comatose state combined with these diffuse punctate lesions was strongly suggestive for fat embolism. Seven days later, transthoracic cardiac ultrasonography was performed to confirm the presence of a patent foramen ovale *(figure 2)*. During his two-week admission on the ICU, the patient gradually recovered neurologically. However, eventually he succumbed to respiratory insufficiency because of pneumonia and mucus stasis. Pulmonary CT angiography was performed, which showed no signs of pulmonary emboli.

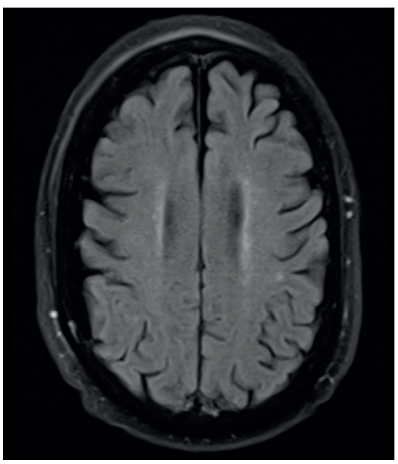

Figure 1A. Fluid attenuated inversion recovery (FLAIR) sequence on the second day after trauma, showing no relevant abnormalities

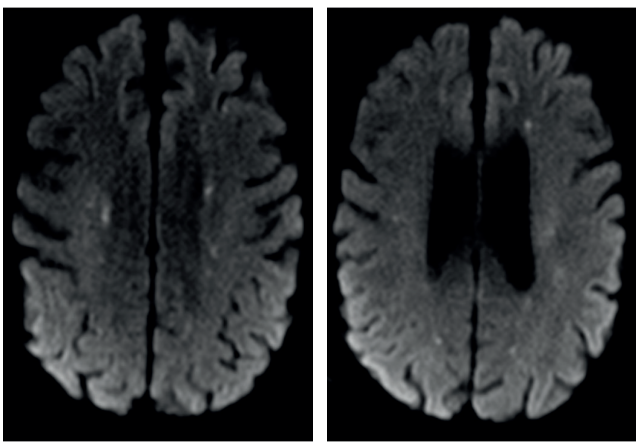

Figure 1B and 1C. Diffusion-weighted MR images at two different levels, showing multiple dot-shaped areas with diffusion restriction in both hemispheres

Diagnosis

Isolated cerebral fat embolism after hip surgery in the presence of a patent foramen ovale

Fat embolism syndrome

Patients with fat embolism syndrome typically present with a triad of petechial rash, pulmonary distress and neurological dysfunction 24-72 hours after the initial trauma.[1] The presumed

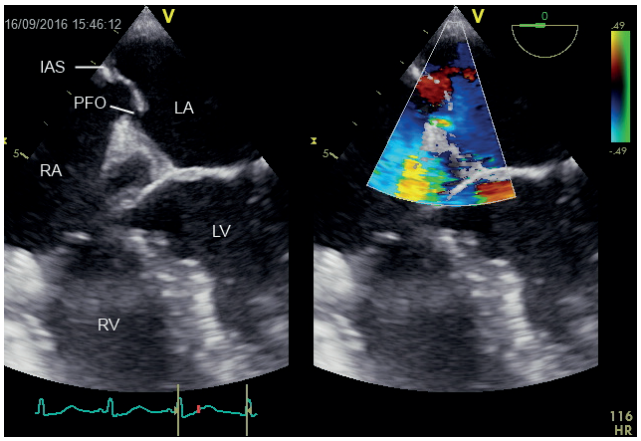

Figure 2. Still of a transesophageal echocardiography, demonstrating a patent foramen ovale (PFO) with intermitting flow (right to left shunt)
RA = right atrium; LA = left atrium; RV = right ventricle; LV= left ventricle; IAS = interatrial septum.

pathway is the formation of fat emboli in the affected limb, which are transported via the vena cava to the heart. Thereafter, the emboli enter the pulmonary circulation and obstruct the blood flow. The most prominent symptoms in patients are often of pulmonary origin, although an isolated form of cerebral fat embolism syndrome has been described.[2] Although the only way to bypass this pulmonary circulation would be a patent foramen ovale, there is also evidence that microemboli can pass through the pulmonary capillaries or the opening of recruitable pulmonary vessels.[3] The diffusion restriction demonstrated on cerebral MRI reflects the immediate cytotoxic oedema secondary to ischaemic occlusion of the cerebral arterioles. A susceptibility-weighted imaging sequence of MRI would have been more specific and sensitive than diffusion-weighted imaging for the detection of fat embolism syndrome.[4,5] However, this sequence was not yet available in our hospital at the time the patient presented. A T2* sequence showed a subtle hypointense region in the subependymal white matter of the left lateral ventricle, at the same location as a hyperintense lesion on diffusion-weighted imaging was shown. The treatment of fat embolism syndrome mainly consists of supportive care, after which full recovery is possible.[6]

Disclosures

All authors declare no conflict of interest. No funding or financial support was received.

References

1. Buskens CJ, Gratama JW, Hogervorst M, van Leeuwen RB, Rommes JH, Spronk PE. Encephalopathy and MRI abnormalities in fat embolism syndrome: a case report. Med Sci Monit. 2008;14:CS125-9.
2. DeFroda SF, Klinge SA. Fat Embolism Syndrome With Cerebral Fat Embolism Associated With Long-Bone Fracture. Am J Orthop (Belle Mead NJ). 2016;45:E515-21
3. Sulek CA1, Davies LK, Enneking FK, Gearen PA, Lobato EB. Cerebral microembolism diagnosed by transcranial Doppler during total knee arthroplasty: correlation with transesophageal echocardiography. Anesthesiology. 1999;91:672-6.
4. Zaitsu Y, Terae S, Kudo K, et al. Susceptibility-weighted imaging of cerebral fat embolism. J Comput Assist Tomography. 2010;34:107-12.
5. Yeap P, Kanodia AK, Main G, Yong A. Role of susceptibility-weighted imaging in demonstration of cerebral fat embolism. BMJ Case Rep. 2015 Jan 8.
6. Shaikh N. Emergency management of fat embolism syndrome. J Emerg Trauma Shock. 2009;2:29-33.