

Cerebral Fat Embolism After Video-Assisted Thoracic Surgery



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Cerebral fat embolism (CFE) is an uncommon disease occurring mainly after traumatic lower limb long bone fractures. A 64-year-old woman with pneumonia and bilateral pleural effusion underwent video-assisted thoracic surgery (VATS). After 3 days, the patient had an acute decrease in consciousness level followed by the onset of seizures and right hemiparesis. Brain computed tomography and magnetic resonance imaging showed findings suggestive of CFE. CFE occurring after an uncommon nontraumatic cause of fat embolism, such as VATS, is a rare clinical event whose diagnosis could be challenging. Neuroimaging can hasten diagnosis and prevent other unnecessary investigations and treatments.

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The diagnosis of fat embolism (FE) syndrome is based on clinical features and exclusion of other pathologic conditions with similar symptoms. However, no universal diagnostic criteria have been defined [1]. Indeed, the widely accepted Gurd criteria [2] are not satisfied in all clinical instances. Cerebral fat embolism (CFE), an incomplete type of FE syndrome [3], could be one of these clinical eventualities. Especially when it is secondary to uncommon causes of FE, such as nontraumatic conditions [4], CFE may not be suspected clinically because neurologic signs and symptoms are variable and nonspecific, and this may lead to diagnostic delay. We report a case of nontraumatic CFE that developed after video-assisted thoracic surgery (VATS) treatment of pleural empyema that, not satisfying Gurd criteria, resulted in a challenging diagnosis.

A 64-year-old woman was admitted to our hospital (Azienda Ospedaliero-Universitaria Careggi, Florence, Italy) for chest pain, fever, and dyspnea. A chest computed tomography (CT) scan showed bilateral pulmonary consolidations. Despite prompt initiation of

antibiotic therapy, bilateral pulmonary empyema developed. The empyema was drained at first by thoracentesis, and the patient was then treated with VATS using general anesthesia. Three days later, the patient complained of sudden headache followed by loss of consciousness with tonic-clonic seizures and right hemiparesis. With the clinical suspicion of acute stroke, she underwent brain CT that unexpectedly showed hypodense, round lesions in the subcortical white matter of left parietal and occipital lobes (Fig 1A). The CT density of the brain lesions was within the range of fat, a finding suggesting CFE. Magnetic resonance imaging (MRI) confirmed the diagnostic hypothesis of CFE, by showing, in susceptibility-weighted imaging sequences, punctuate foci of low signal intensity compatible with microbleeding in the subcortical white matter of left parietal lobe (Fig 1B). Given the presence of seizures, the patient underwent an electroencephalogram (EEG), which showed the presence of continuous sharp and slow wave activity at 2 to 2.5 Hz on the right hemisphere and of paroxysmal activity, consisting of lateral periodic discharges on the left centroparietal leads, associated with decreased voltage of the background activity (Fig 2A). This pattern on the EEG was in keeping with a postictal state, so intravenous valproate (2.5 mg/kg per hour) was started. The next day, she underwent transesophageal echocardiography, showing the presence of a 5-mm patent foramen ovale (PFO) that was characterized by left-to-right shunt at Doppler echocardiography and by the evidence of right-to-left shunt during the Valsalva maneuver. The presence of right-to-left shunt was further confirmed by echocardiography with agitated saline contrast.

Two days after the onset of neurologic symptoms, another brain MRI scan showed, on fluid attenuated inversion recovery (FLAIR) sequences, the presence of hyperintense lesions (Fig 1C) related to an increased diffusion signal on axial apparent diffusion coefficient maps (Fig 1D). The imaging revealing vasogenic edema at the level of thalamus and on the left parietal cortex. Moreover, no further embolization was detected. At the same time, the patient showed significant improvement in consciousness level and an almost complete recovery from right hemiparesis. A follow-up EEG showed reappearance of medium-posterior background activity and remission of epileptiform discharges (Fig 2B). One week later, she underwent another brain CT scan, which showed the disappearance of the findings previously described by the first CT scan and absence of ischemic lesions. This evolution of the CT findings was compatible with vasogenic edema.

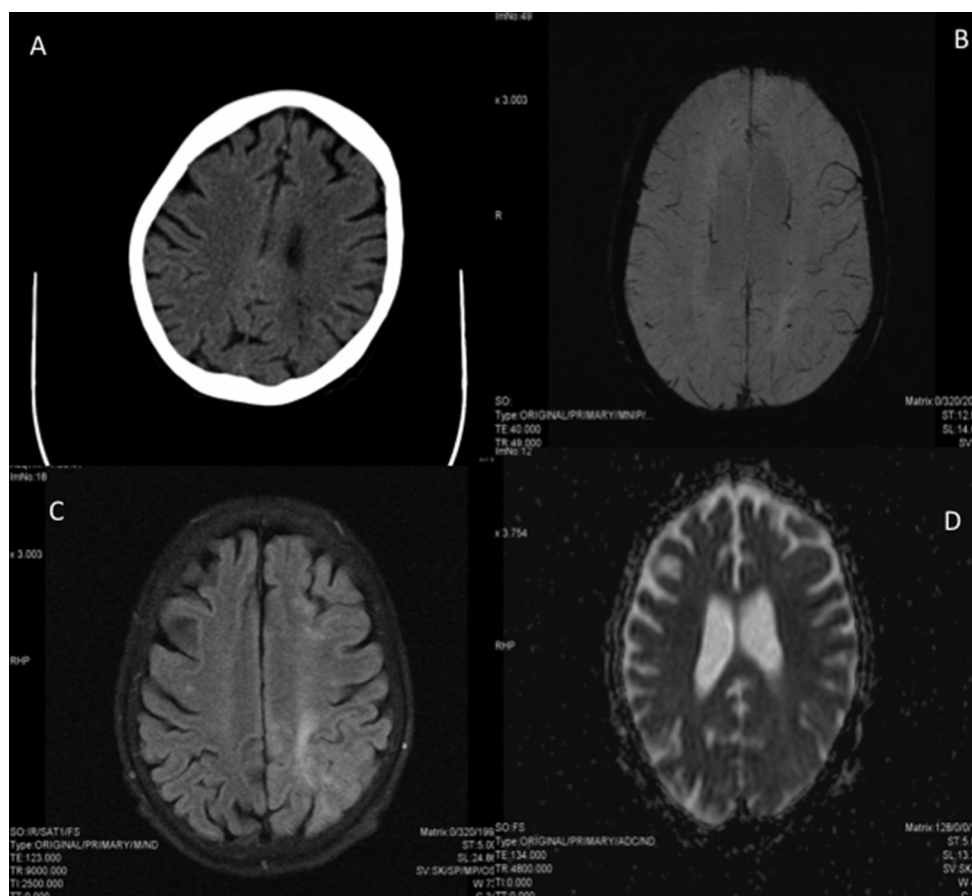
Comment

Previously, Yilmaz and colleagues [5] reported one case of CFE secondary to empyema, but this patient was treated with pleural irrigation and not with VATS. In that case the diagnosis of CFE was made by brain CT, without confirmation by MRI (the gold standard for the diagnosis of these incomplete cases of FE). CFE, especially with an

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Fig 1. Neuroimaging. (A) Computed tomography: small left parietal sulcal fat hypodensity. (B) Susceptibility weighted imaging: punctuate subcortical left parietal hypointensity. (C) Fluid attenuated inversion recovery: left parietal subcortical hyperintensity. (D) Apparent diffusion coefficient maps: increased left parietal subcortical diffusion.



uncommon, nontraumatic cause, may be a great diagnostic challenge. Indeed, in our patient the presence of nonspecific neurologic signs and symptoms led to the clinical suspicion of acute stroke. CFE was suspected from brain CT, but only MRI allowed confirmation of the diagnosis. In the early stages of CFE, as shown in this case, diffusion-weighted imaging and susceptibility-weighted imaging are more sensitive than FLAIR and T2-weighted sequences to detect lesions and can hasten the establishment of the diagnosis. Anyway, in our patient, the size and distribution of the brain lesions did not correlate with the extent of clinical neurologic impairment. The prompt and almost complete resolution of the neurologic symptoms, associated with improvement of activity on the EEG but with the persistence of brain lesions detected by MRI, is compatible with our assumption that an epileptic status before, and a postictal status afterward, could have contributed to the decreased level of consciousness and the motor symptoms (Todd paralysis).

Concerning the pathogenesis of this condition, the patient had no clearly evident risk factors that could have facilitated FE (ie, she was not obese, and she was not parenterally fed). According to the literature, two distinct theories, one mechanical and the other biochemical, have been postulated [4]. In our patient, both mechanisms could have had a pathogenic role. Taking into account the nontraumatic origin of CFE and the unusually long

period of time elapsed between VATS and the onset of symptoms (approximately 72 hours instead of the usual debut within 48 hours), we hypothesize that a biochemical mechanism is the most probable. In fact, an alteration in lipid metabolism that could facilitate FE may occur in distressed conditions such those of our patient, who had pneumonia complicated by empyema requiring surgical management with general anesthesia [1]. Moreover, the presence of PFO could have facilitated the transition of embolic particles from the venous circulation to the systemic circulation, thus resulting in CFE. Indeed, investigators have reported that the presence of PFO is a risk factor for more severe manifestations of paradoxical FE, particularly in the absence of lung involvement [6, 7]. Although less likely, a mechanical origin may have contributed to the genesis of CFE. The destruction of fat cells and venous sinusoids of soft tissues during VATS could have allowed the access of fat in the venous circulation. However, the 72-hour delay after VATS makes this mechanism quite unlikely.

CFE may be difficult to differentiate from other neurologic conditions, and its diagnosis can be particularly challenging, mainly when it is secondary to uncommon nontraumatic causes of FE, such as in this case. CT and MRI (to confirm the diagnosis) should be the standard diagnostic tests. If such imaging is obtained early in the course of the condition, it can hasten the

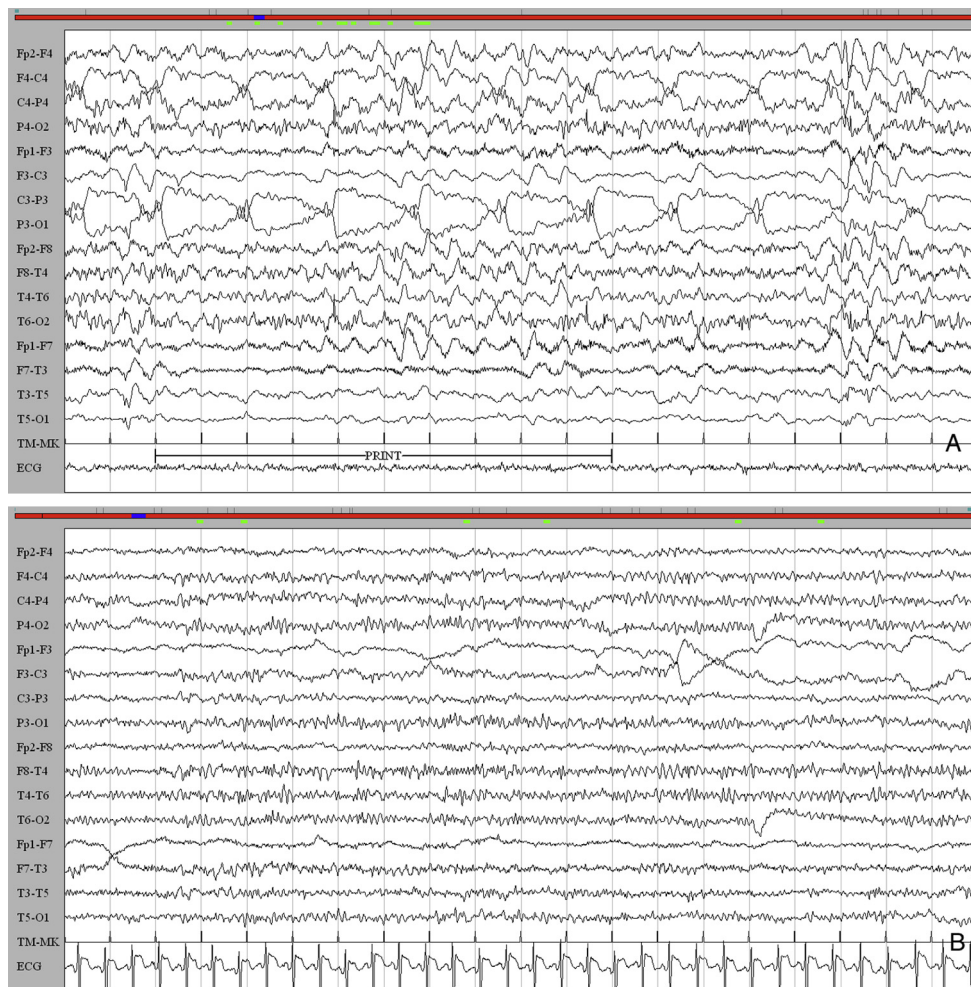


Fig 2. Electroencephalogram. (A) Sharp and slow wave activity on the right hemisphere and lateralized periodic discharges with background attenuation on the left hemisphere. (B) Reappearance of posterior alpha rhythm and remission of epileptiform discharges.

diagnosis and can be used to avoid other unnecessary investigations and treatments.

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