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LETTER TO THE EDITOR

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Delayed cerebral fat embolism occurring after off-pump coronary artery bypass grafting

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Fat embolism syndrome (FES) is a serious clinical disorder occurring after trauma or orthopedic procedures. Only a small number of patients with fat embolism (FE) develop signs and symptoms due to multisystem dysfunction, mainly involving the lungs, brain and skin [1, 2]. The diagnosis of FES is based mainly on these major clinical features, but it may often be complex because laboratory tests are non-specific and because the Gurd criteria [3] are not satisfied in all clinical instances. In fact, patients could be affected by incomplete types of FES [4], such as cerebral fat embolism (CFE) [5]. When CFE is specifically secondary to uncommon causes of FE, such as non-traumatic conditions [6], it can represent a great diagnostic challenge. A 71-year-old man, with peripheral arterial occlusive disease, was admitted to the indicated hospital with dyspnea and chest pain. An electrocardiogram showed signs of myocardial ischemia. Therefore, an intra-aortic balloon pump had been introduced for heart failure, and the patient underwent an off-pump coronary artery bypass grafting (OP-CABG) with clamping of the ascending aorta and proximal anastomosis. The patient did not have angiographic findings of calcifications in the ascending aorta as assessed by preoperative angio-computed tomography (CT) scan; therefore, the risk of embolization was a priori low. In the following days, the patient showed progressive improvement of hemodynamic function, but a distal ischemia of the left foot, which caused subcutaneous fat tissue, toe and metatarsus bone necrosis, occurred. Two weeks later, the patient showed a sudden loss of consciousness and right hemiplegia. With the clinical suspicion of an acute stroke, he underwent brain CT, which unexpectedly showed, in the subcortical white matter of the parietal, temporal and occipital lobes, even though prevailing in the left hemisphere (Fig. 1A, B), the presence of round, hypodense lesions within the range of fat (–40 HU) suggesting FE. Magnetic resonance imaging (MRI) showed, on diffusion-weighted imaging, the presence of non-specific hyperintense lesions in the same regions as those detected by the brain CT scan, related both to an increased and to a decreased diffusion signal on axial apparent diffusion coefficient maps, revealing both vasogenic and cytotoxic edema. The gold standard for FE diagnosis is MRI, with typical bright spots on a dark background (starfield pattern), and microbleeding; however, these findings were not present in the current case. Nevertheless, the diagnosis of CFE can only be made by brain-CT, without MRI confirmation. A considered opinion, with careful examination of brain-CT findings, the topography and density measurements of round lesions, was enough to confirm the clinical suspicion of CFE.

The following day the patient was still comatose and presented a tonic-clonic seizure. An electroencephalogram (EEG) (Fig. 1C, D) performed on the same day showed the presence of a focal non-convulsive status epilepticus (NCSE) related to ictal EEG activity, characterized by recruiting sharp activity followed by lateral periodic discharges in the right occipital region, hence, intravenous valproate was started (loading dose

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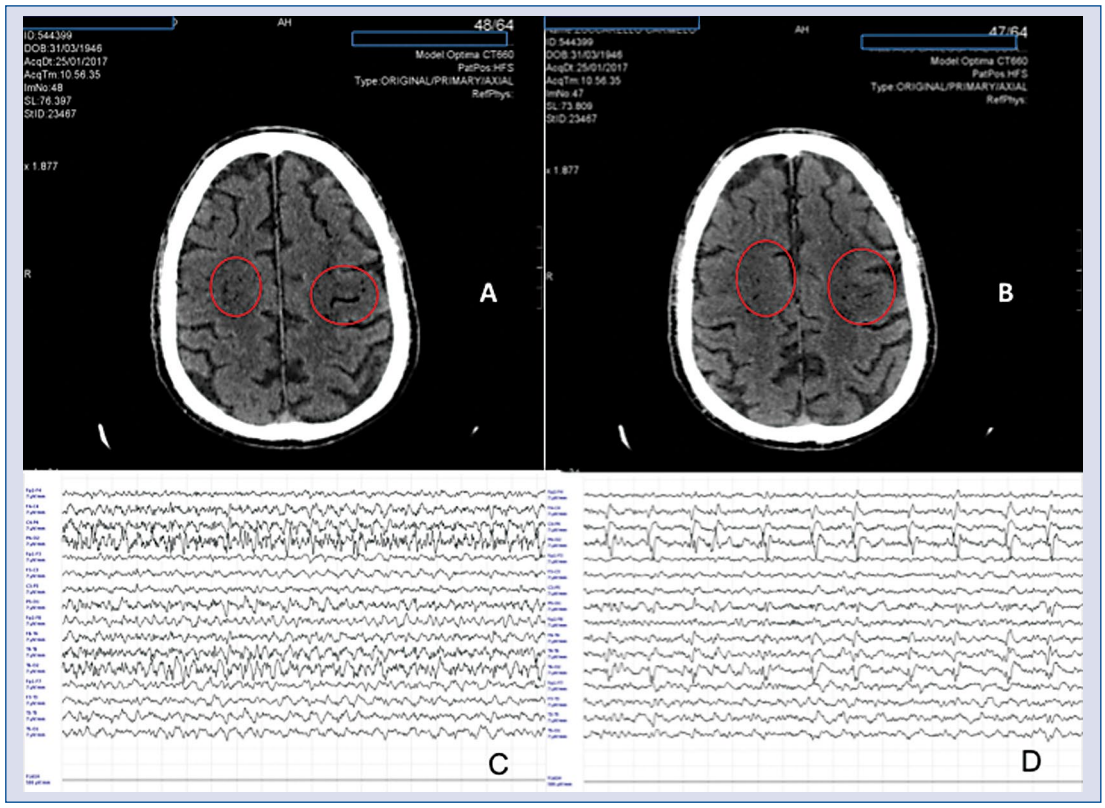

Figure 1. Neuroimaging findings: brain computed tomography (CT) axial image showing hypodense, round lesions (inside ovals) in the cortical and subcortical white matter of the frontal, temporal and occipital lobes, prevaling in the left hemisphere at the brain CT (**A**). The CT density of the round lesions was within the range of fat (**B**). Electroencephalogram findings: Ictal epileptiform activity of recruiting spikes on right parieto-occipital leads (**C**) associated with periodic lateralized epileptiform discharges in the same regions (**D**).

25 mg/kg, followed by infusion of 2 mg/kg/h). On the same day, he also underwent transesophageal echocardiography, failing to demonstrate the presence of a patent foramen ovale (PFO). Two days later the patient presented with open eyes, but was unresponsive to external stimuli. Another EEG showed the resolution of NCSE, even though slowed background activity had persisted. Almost 15 days after the onset of the symptomatology, the patient underwent another MRI that on fluid attenuated inversion recovery sequences showed the presence of important ischemic areas in the same regions as those detected by previous neuroimaging. The day after, the patient was discharged from the intensive care unit showing spontaneous movements of the limbs and eye opening in response to voices, but was still not responding to simple instructions. CFE, especially when secondary to an uncommon, non-traumatic cause, may result in a great diagnostic challenge and could lead to diagnostic delay because neurologic signs and symptoms are variable and non-specific, and because they did not satisfy the Gurd criteria [3]. In the present case, the neurologic symptomatology was characterized by a sudden loss of consciousness and right hemiplegia, followed by tonic-clonic seizure. In the following days, the resolution of the right focal NCSE, as detected by the EEG, which was not associated with an improvement in consciousness, gave rise to the suspicion that patient symptomatology was mainly due to the extent of the brain lesions rather than to the presence of ictal activity. FES pathophysiology may be due to mechanical and/or biochemical causes [6]. In this case, the origin of FE remained quite uncertain and could be multifactorial. Taking into account the ischemia of the left foot, a mechanical mechanism can be hypothesized, similar to what happens in traumatic conditions. In fact, the necrosis due to ischemia could have caused damage not only to the bones and subcutaneous fat tissue, but also to the intramedullary veins, as it occurred after a trauma, allowing marrow fat to intravasate and embolize first to the lungs and then to the brain. Indeed, FE

has been reported not only after long-bone injuries, but also after rib or tarsal bone involvement [7]. On the other hand, a biochemical mechanism might be responsible as well. In fact, according to this theory, there is an alteration in lipid metabolism that could facilitate FE in distressed conditions, such as those of the present patient who underwent cardiac surgery under general anesthesia some days earlier, and showed an unstable hemodynamic condition. Concerning the absence of a right-to-left heart defect as demonstrated at the transesophageal echocardiography, it is well established in the literature that CFE can also occur in the absence of a PFO [8]. In fact, such emboli are believed to reach the systemic circulation either through pulmonary precapillary shunts or directly across the pulmonary capillary bed, even in the absence of lung symptoms. However, in the absence of a PFO, this physiopathological mechanism could be considered rather speculative. Finally, because the diagnosis of FE remains a difficult task as there are no universal diagnostic criteria [1], and because incomplete forms, such as CFE, are uncommon, it is important to perform neuroimaging at an early stage, keeping this clinical suspicion in mind at the same time. In the present case brain CT findings (round hypodense lesions, –40 HU) were suggestive for CFE, whereas the typical MRI findings of starfield patterns were not observed. Then, in some cases, only brain CT can confirm the diagnosis. Keeping CFE suspicion in mind enables an avoidance of unnecessary treatments, as in the presented case, in which the stroke team had been alerted in anticipation of performing thrombolysis, suspecting a brain infarction in the first instance. CFE is an incomplete form of FE occurring mainly after traumatic lower-limb long-bone fractures. When it is secondary to an uncommon non-traumatic cause, diagnoses can be challenging. Neuroimaging hastens diagnosis, thus preventing unnecessary investigations and treatments. Finally, since this patient had other comorbidities more frequently associated with FE, such as foot necrosis, the delayed occurrence of the embolism 2 weeks after the off-pump surgery precluded a direct causal relationship with this procedure.

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