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Brief Communication

Super-refractory nonconvulsive status epilepticus secondary to fat embolism: A clinical, electrophysiological, and pathological study

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ABSTRACT

Background: Fat embolism syndrome (FES) is a rare complication of long-bone fractures and joint reconstruction surgery. To the best of our knowledge, we describe the clinical, electrophysiological, neuroimaging, and neuropathological features of the first case of super-refractory nonconvulsive status epilepticus (sr-NCSE) secondary to fat embolism.
Clinical case: An 82-year-old woman was transferred to our intensive care unit because of a sudden decrease of consciousness level, right hemiparesis, and acute respiratory failure in the early postoperative period of knee prosthesis surgery. Brain computed tomography (TC) including angio-CT and CT perfusion was normal. An urgent video-electroencephalography (v-EEG) evaluation showed continuous sharp-and slow-wave at 2.0–2.5 Hz in keeping with the diagnosis of generalized NCSE. Epileptiform discharges ceased after the administration of 5 mg of intravenous diazepam, and background activity constituted by diffuse theta waves was observed without clinical improvement. Treatment with levetiracetam (1000 mg/day) and sedation with propofol and midazolam were initiated. Moreover, continuous v-EEG monitoring was also started. Despite antiepileptic therapy, epileptiform activity recurred after the interruption of profound sedation, and valproate and lacosamide were added during the ensuing days. Magnetic resonance imaging (MRI) disclosed small scattered foci of acute ischemic infarcts and diffuse petechiae involving the basal ganglia and pons and centrum semiovale in keeping with fat embolism. Super-refractory nonconvulsive status epilepticus remained without control for 2 weeks. Finally, the patient died. The clinical autopsy revealed a bilateral lung fat embolism associated with a hemorrhagic infarction in the left lower lobe. Fatty lesions were also seen in the intestine and pancreas. Scattered microscopic cerebral infarcts associated with fat emboli in the capillaries were noticed, affecting both supra- and infratentorial structures. In addition, occasional focal areas of ischemic injury showing filiform neurons with reactive astrocytic gliosis background consistent with acute lesions were observed in CA3.
Conclusions: Fat embolism should be considered a potential cause of sr-NCSE.

This article is part of a Special Issue entitled "Status Epilepticus".

1. Introduction

Nonconvulsive status epilepticus (NCSE) is a pleomorphic epileptic condition in which behavior disturbance and the level of consciousness may range from minimal to coma [1,2]. Although more commonly recognized as an etiology of mental status changes in a patient who has suffered trauma with long-bone fractures, fat embolism syndrome (FES) is a rare but potential mechanism of encephalopathy or even coma in patients who previously underwent orthopedic procedures or cardiac surgery. In addition to the central nervous system manifestations, this syndrome is also characterized by pulmonary and cutaneous symptoms.

To the best of our knowledge, we describe the clinical, electrophysiological, neuroimaging, and neuropathological features of the first case of super-refractory nonconvulsive status epilepticus (sr-NCSE) secondary to fat embolism.

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2. Clinical case

An 82-year-old woman with a history of mild cognitive impairment, hypothyroidism, severe osteoporosis, and osteoarthritis was transferred to our intensive care unit (ICU) because of a sudden decrease of consciousness level, right hemiparesis, and acute respiratory failure in the early postoperative period of knee prosthesis surgery. Surgical or anesthetic incidents were not mentioned during the intervention. Once the sedation had been withdrawn, the patient awoke and was extubated. However, 2 h after, she suddenly experienced right hemiparesis, an abrupt decrease in the level of consciousness (Glasgow Coma Scale 7), and hypoxemic respiratory failure. Focal motor or generalized tonic–clonic seizures were not observed. On ICU admission, the patient did not show cutaneous lesions. Laboratory abnormalities included a leukocyte count of 23,000/ml, hemoglobin of 10.6 g/dl, and procalcitonine of 1.38 ng/ml. The clinical course during the first hours was characterized by shock and severe hypoxemic respiratory failure, with the need of intensive fluid resuscitation and vasoactive drugs. Transthoracic echocardiography was performed, showing findings compatible with pulmonary embolism, but a patent foramen ovale was not observed. A chest computed tomography (CT) angiography test ruled out pulmonary embolism and disclosed a right lower lobe pneumonia without abnormalities in the aortic arch. Supportive treatment was maintained, and prophylactic treatment with broad spectrum antibiotics was instituted. Brain CT angiography and CT perfusion did not reveal anomalies. Subsequently, hemodynamic and respiratory improvement allowed the withdrawal of sedation on day 2 after admission. However, the patient remained in deep coma (Glasgow

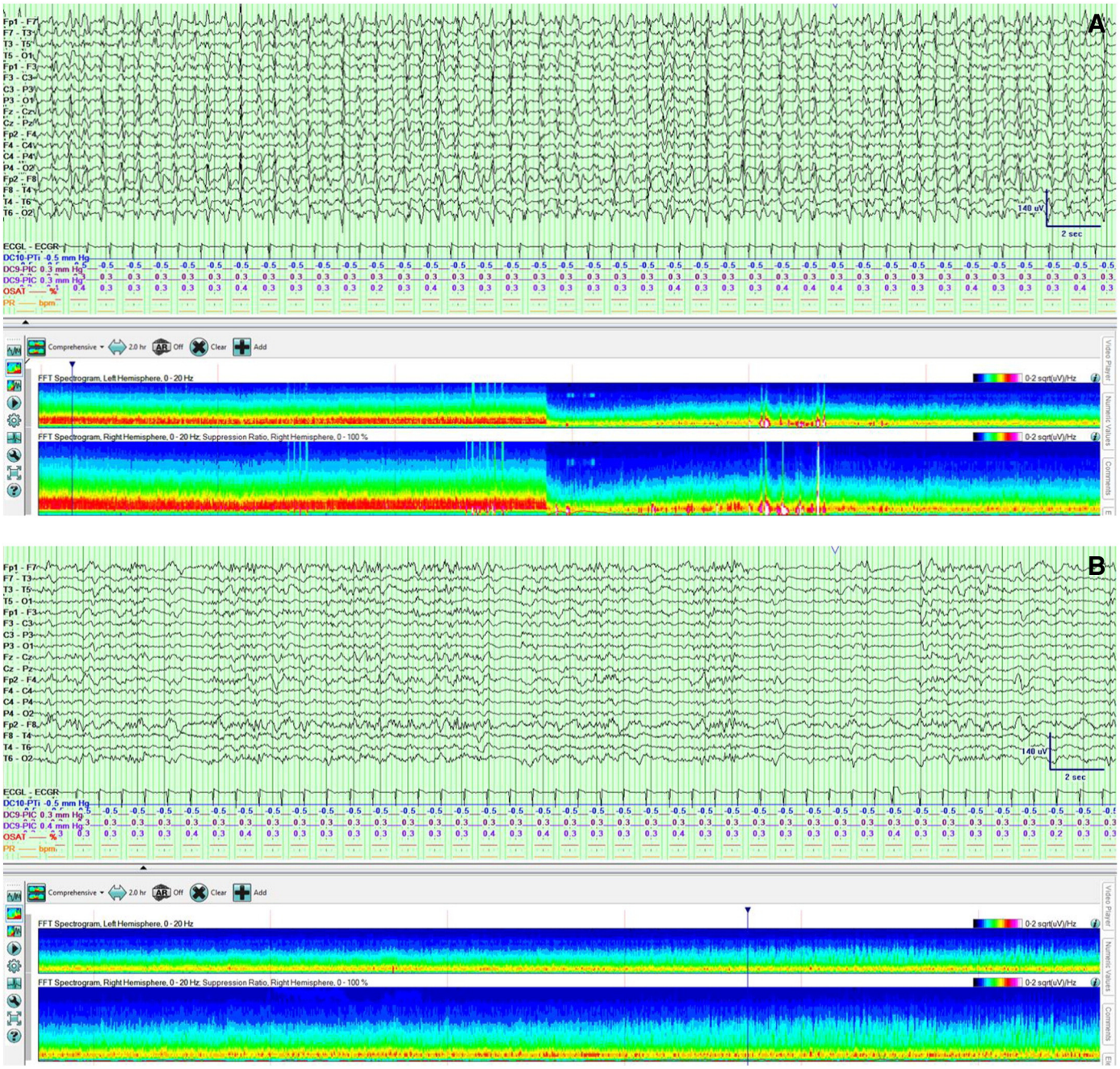

Fig. 1. Continuous videoelectroencephalography. A) Observe the presence of generalized periodic discharges (GPDs) at 2.5 Hz in keeping with generalized NCSE in a comatose patient. B) EEG after the infusion of propofol and midazolam. Notice the presence of abundant diffuse fast rhythms and periods of generalized decrement of the background activity and abolition of GPDs. Note the change of the spectrogram during NCSE and after the abolition of the epileptiform discharges. LF: 0.53 Hz, HF: 70 Hz, and NF: 50 Hz; vertical bar: 140 μV; horizontal bar: 2 s.

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