Favorable Outcome of Cerebral Fat Embolism Syndrome with a Glasgow Coma Scale of 3: a Case Report and Review of the Literature

Keng-Yi Lin · Kai-Chen Wang · Yao-Li Chen ·
Ping-Yi Lin · Kuo-Hua Lin

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Abstract Prognosis of deep coma caused by cerebral fat embolism syndrome (CFES) is rarely reported. We present a case of fulminant CFES which was induced by long bone fracture, with a Glasgow Coma Scale (GCS) of 3/15. The brain magnetic resonance imaging (MRI) revealed abnormal spotty lesions scattered over both cerebral hemispheres and the posterior fossa. Thirty days later, the patient regained consciousness with a GCS of 15/15.

Keywords Cerebral fat embolism syndrome · Fat embolism · GCS

Introduction

Deep coma caused by cerebral fat embolism syndrome in traumatic long bone fracture is rare [1]. Reitboeck et al. reported the only patient of CFES with a GCS of 3/15 [2]. Here, we present another case of deep coma induced by fulminant CFES, with only minor hypoxia. The patient regained consciousness and became fully oriented 30 days later; however, he still suffered severe upper and lower limb weakness.

Case Report

A previously healthy 28-year-old man was brought to the emergency department because of loss of consciousness and multiple skeletal injuries following a motorcycle accident. Initial examination revealed a GCS score of

15/15, minor left-sided pneumothorax, fracture of the right distal radius, and open fracture of the left femoral shaft (Fig. 1). The first brain computed tomographic (CT) scan showed normal findings. Because of the risk of delayed intracranial hemorrhage, the left femoral shaft was initially reduced by skeletal traction rather than immediate open reduction. Approximately 12 h after admission, the patient suddenly became unresponsive. His GCS was 8/15, oxygen saturation was 94 %, and oxygen partial pressure was 76 mmHg. Brain CT scan revealed multiple hypo-dense lesions in the brain parenchyma. Result of laboratory test revealed a hemoglobin level of 9.4 g/dL and a platelet count of 60,000/ μ L. An endotracheal tube was inserted for airway protection, and a left chest tube was placed to prevent tension pneumothorax. Approximately 56 h after admission, brain MRI revealed an extensive starfield appearance characterized by abnormal spotty lesions scattered over both cerebral hemispheres and the posterior fossa. Both gray and white matters were involved, a finding consistent with acute cerebral fat emboli (Fig. 2). Follow-up chest radiograph and arterial blood gas values were normal. Echocardiogram did not demonstrate a patent foramen ovale or other congenital defects. Open reduction and internal fixation of the right distal radial and left femur fractures were performed on the 4th day. Postoperative vital signs were relatively stable; however, the patient remained comatose with a GCS of 3/15. He was transferred to another hospital 7 days after admission at the request of his family.

At that hospital, intracranial pressure (ICP) monitoring was performed. Approximately 30 days after initial injury, the patient regained consciousness and his GCS score gradually returned to 15. However, the patient remains bedridden because of upper and lower limb weakness.

K.-Y. Lin · K.-C. Wang · Y.-L. Chen · P.-Y. Lin · K.-H. Lin (✉)
Department of Surgery, Changhua Christian Hospital, No. 135,
Nanxiao St, Changhua City, Changhua County, Taiwan
e-mail: 120380@cch.org.tw

Fig. 1 Fracture of left femoral shaft; fracture of right distal radius

Discussion

The incidence of fat embolism syndrome (FES) in patients with long bone fracture ranges from 0.9~2.2 % [3]. Gurd criteria defines FES with three major symptoms: pulmonary distress ($\text{PaO}_2 < 60 \text{ mmHg}$), CNS depression, and petechiae. Hypoxia is the most common manifestation (96 %), followed by CNS depression (59 %) [4]. CNS depression was hypothesized caused by fat emboli. Based on our review of the literature, only one case with a GCS of 3/15 has been reported [2].

There were two hypotheses of the pathophysiology of CFES [5]. One theory is that small (7–10 μm in diameter) fat globules escape to the systemic circulation through an intracardiac shunt or because of a pulmonary arteriovenous malformation, causing ischemic “starfield” brain injury. Another theory is that freely circulating fatty acid produced by lipase during inflammation can aggregate fat globules and release inflammatory mediators, which changes capillary permeability, causing cytotoxic edema and ischemic change. In

our patient, there was no echocardiographic evidence of intra-cardiac shunting or arteriovenous malformation, which leads us to believe that the latter theory is more likely.

Definitive diagnosis of CFES depends on image findings. The classical starfield appearance on diffusion-weighted MRI imaging represents cytotoxic edema due to fat emboli. A recent systemic review revealed that scattered cytotoxic edema is the most common image finding of acute CFES, present in 84.6 % patients [6].

It is paramount that patients of CFES with long bone fracture receive fluid resuscitation, airway ventilation, and early fixation within 24 h. ICP monitoring is also beneficial [7]. Albumin binding to oleic acid can decrease the cytotoxic effect of fatty acid. However, treatments with corticosteroids, heparin, alcohol, aspirin, and dextran have failed to reduce morbidity or mortality [8].

Few studies have reported on the recovery of patients after deep coma caused by CFES. Reitboeck et al. reported on a patient with CFES with a GCS of 3/15, who recovered to GCS

Fig. 2 “Starfield” appearance on diffusion-weighted MRI imaging: multiple hyperintensity lesions represent cytotoxic edema due to fat emboli

of 15/15 with disoriented in place [2]. Scopa et al. reported on a patient with CFES and serious neurologic deficit who recovered without neurologic sequelae [9]. Our patient also regained consciousness 30 days after insult and has made a partial recovery. Large-scale studies on the long-term outcome of patients with CFES-induced coma are needed.

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