

Cerebral fat embolism syndrome after long bone fracture due to gunshot injury

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Abstract

Cerebral fat embolism syndrome is a lethal complication of long-bone fractures and clinically manifested with respiratory distress, altered mental status, and petechial rash. We presented a 20-year-old male admitted with gun-shot wounds to his left leg. Twenty-four hours after the event, he had generalized tonic clonic seizures, decorticate posture and a Glasgow Coma Scale of seven with localization of painful stimuli. Subsequent magnetic resonance imaging of the brain showed a star-field pattern defining multiple lesions of restricted diffusion. On a 4-week follow-up, he had returned to normal neurological function. Despite the severity of the neurological condition upon initial presentation, the case cerebral fat embolism illustrates that, cerebral dysfunction associated with cerebral fat embolism illustrates reversible.

Keywords: Cerebral fat embolism, diffusion weighted magnetic resonance imaging, starfield pattern

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Introduction

Fat embolism syndrome (FES) is a rare and potentially lethal complication of long bone fractures.^[1] Many cases occur as subclinical events and remain undiagnosed. The incidence of clinically significant FES occurs only in 0.9-2.2% of long-bone fractures.^[1,2] Hypoxia or respiratory distress, deteriorating mental status, and petechial hemorrhage are the main diagnostic criteria; secondary diagnostic signs include tachycardia, fever, anemia, and thrombocytopenia typically seen in the context of long-bone fracture.^[2,3] Neurologic symptoms can be transient and widely varies from a diffuse encephalopathy to focal deficits.^[3] Fat emboli can pass through the pulmonary vasculature, resulting in systemic embolization, most commonly in the brain and kidneys. In this article, we aimed to highlight the diagnosis and treatment of cerebral fat embolism through an illustrative a case developing change of consciousness, respiratory

failure and epileptic seizures after long-bone fracture caused by gunshot injury.

Case Report

A 20-year-old male patient was admitted to the emergency department with sudden change in consciousness and muscle contractions after 1 day follow-up in a hospital for long bone fractures of femur and tibia due to gun-shot injury. On admission, he had generalised tonic clonic seizures and his Glasgow Coma Scale was seven with localization of painful stimuli, incomprehensible muttering, and no eye opening. In the physical examination, blood pressure was 130/80 mmHg, pulse was 128/min, breath rate was 22/min and body temperature was 38°C. His pupils were isocoric and bilaterally responsive to light. He had decorticated posture in response to noxious stimulus. No lateralising motor deficit was found. There was a long bone splint on the left lower extremity. Examination of other systems were normal. The patient was intubated and respiration was supported by mechanical ventilation.

The results of hematologic and biochemical parameters of the patient with the range of normal values of our laboratory in the parenthesis were as follows at first admission: Hemoglobine: 11.1 gr/dl (13,6-17,2),

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platelet count: 75000/ μ l (156000-373000), glucose: 189 mg/dl (70-110), aspartate aminotransferase (AST): 143 IU/L (0-46), alanine aminotransferase (ALT): 53 IU/L (0-46), and creatine phosphokinase (CPK): 7866 IU/L (35-195). Other biochemical findings and blood gas analysis were normal. There were no abnormalities on a postero-anterior chest X-ray cervical and thoracic computed tomography (CT). An initial CT of the head was unremarkable for intracranial abnormalities. Magnetic resonance imaging (MRI) was performed diffusion weighted MRI trace images with a 2 mm slice gap, 5 mm slice thickness, Echoplanar imaging (EPI) sequence type, b value of 1000 mm/s², signal intensity dots on a dark background. This resulted in a "starfield" appearance of the cerebral hemispheres [Figures 1a and 2a]. Diffusion coefficient map slice of the

patient revealed the hypointense punctate to lesions in centrum semiovale and periventricular regions [Figures 1b and 2b].

An electroencephalogram was performed and diffuse bioelectrical delay on the right hemisphere was found. With the above findings, the diagnosis of the case was cerebral diffuse FES. Antiepileptic therapy with phenytoin (2 \times 150 mg) and low molecular weighted heparin therapy with enoxaparin sodium (2 \times 4000 U) were administered.

The patient had hypoxemia and the arterial blood gas analysis revealed PaO₂ as 65 mmHg, and saturation of arterial oxygen as 82% requiring mechanical ventilation, disturbance of consciousness and sudden drop in hemoglobin and platelet count. Hemoglobin

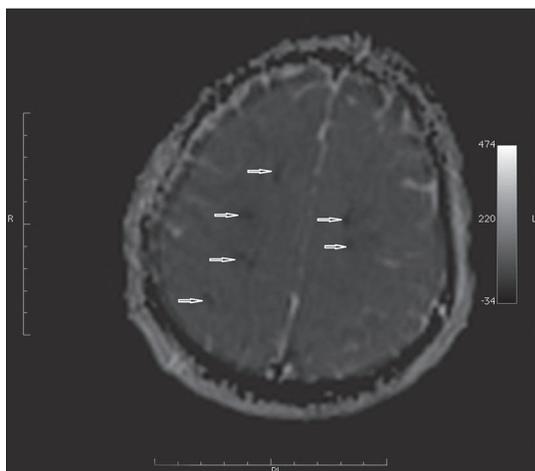


Figure 1a: Diffusion weighted cranial magnetic resonance image reveals starfield pattern; bilaterally localised multiple hyperintense punctate lesions in centrum semiovale regions (arrows). The slice thickness of the images was 5 mm, Echoplanar imaging was used and the b value of the slices was 500

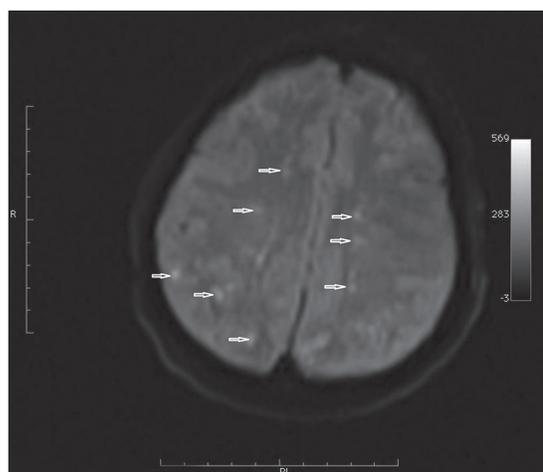


Figure 1b: Diffusion coefficient map slice of the patient reveals the hypointense punctate to lesions (arrows) in centrum semiovale region. The slice thickness of the images was 5 mm, Echoplanar imaging was used and the b value of the slices was 500

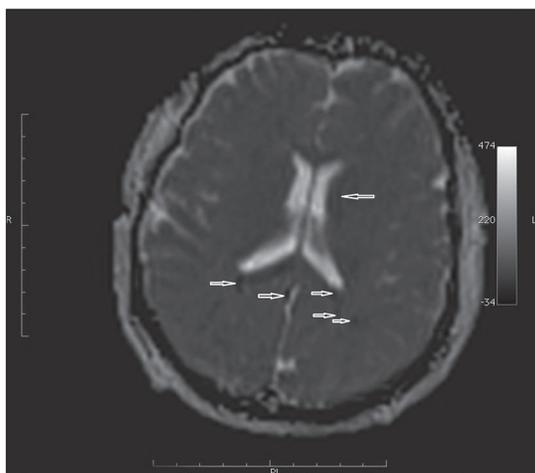


Figure 2a: Diffusion weighted magnetic resonance image reveals starfield pattern; multiple millimetric sized hyperintense lesions (arrows) due to restricted diffusion areas in periventricular regions. Echoplanar imagings were taken with slice thickness of 5 mm and b value of 1000

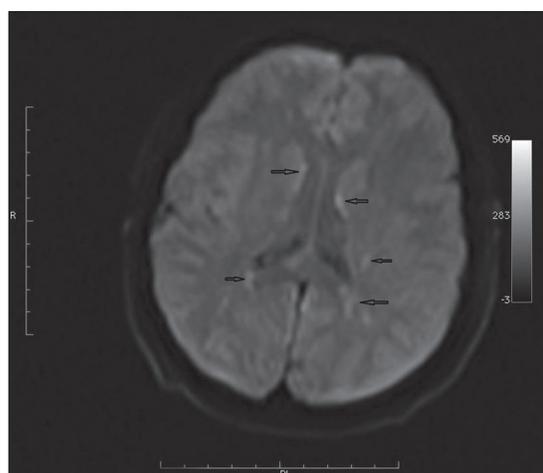


Figure 2b: Diffusion coefficient map slices of the patient reveals the hypointense punctate lesions (arrows) in periventricular region. The slice thickness was 5 mm, Echoplanar imaging with b value of 500 was used

was 7.1 gm/dl and platelet count had decreased to 53000/ μ l at that time. The patient was rehydrated and administered with sulbactam/ampicillin (4 \times 1 g i.v.) for fever and a possible infection due to open injuries. Three units of erythrocyte suspension and 3 units of fresh frozen plasma were given to patient to replace blood loss. On the 5th day of hospitalization, the level of consciousness of the patient improved and mechanical ventilatory support could be discontinued.

The bone fractures were multi-fragmental in left tibia with articular defects. The fragments were internally fixed. He subsequently had uncomplicated recovery and was discharged home after 27 days of hospitalization.

Discussion

FES was first described in 1862 after an autopsy identified fat in the pulmonary vasculature following a crush injury.^[1] Pinney *et al.*,^[4] reported a FES rate of only 4% in a study of 274 consecutive patients with isolated femoral shaft fractures. Fat globules generated within the systemic circulation may cause pulmonary dysfunction, neurological changes, dermal symptoms, and dysfunction of several other organs. The incidence of symptomatic fat embolism was found much lower (0.9%) than the incidence of fat globules within the circulation (22.0%).^[5,6] Long bone fractures are known as recognized to be important risk-factors for FES, especially femur fracture, followed by tibia fracture. The present patient had tibia fracture due to gunshot injury. Most of the FES were reported 24-72 h after long bone fractures.^[6] In the present case, the clinical symptoms and seizure appeared 24 h after the event. Neurological symptoms can be transient and widely varied from a diffuse encephalopathy to focal deficits.^[6] The most common clinical symptoms were reported as hypoxemia (PaO₂ < 70 mmHg), change of consciousness, infiltrates/infiltration findings on chest plain film, and sudden drop in hemoglobin and platelets.^[2] The present patient also had hypoxemia requiring mechanical ventilation, disturbance of consciousness and a sudden drop in platelet count.

Cranial CT does not reveal abnormalities in FES. However, multiple microembolic infarcts could be seen in diffusion-weighted MRI.^[2,3] Gregorakos *et al.*,^[7] described two teenage males suffering from prolonged coma secondary to FES due to lower extremity fractures from a motor vehicle collision. MRI showed multiple areas of increased signal intensity in the cerebral white

matter in the first patient and diffuse high signal intensity in periventricular and subcortical white matter in the second one. In a patient with FES, Chang *et al.*,^[1] reported multifocal high signal intensity changes in bilateral cerebral hemispheres and cerebellum in diffusion weighted magnetic resonance images. The majority of case reports on FES found that cerebral dysfunction associated with FES to be reversible.^[2-4] Parizel *et al.*,^[8] described a teenage female who fractured her left tibia secondary to motor vehicle injury. Diffusion weighted MRI showed punctate foci of high signal intensity in subcortical white and gray matter and the patient had full neurological recovery by 1 week and repeated magnetic resonance images 4 weeks after the accident revealed disappearance of signal abnormalities.^[2] The outcome of patients with FES who receive supportive care is generally complete resolution of pulmonary, neurological, and dermatological lesions with a mortality of less than 10%.

Conclusion

Cerebral fat embolism is extremely rare but it should be suspected in trauma patients with long-bone fractures accompanied by unexplained neurological or respiratory deterioration.

References

1. Chang RN, Kim JH, Lee H, Baik HJ, Chung RK, Kim CH, *et al.* Cerebral fat embolism after bilateral total knee replacement arthroplasty: A case report. *Korean J Anesthesiol* 2010;59:S207-10.
2. Tsai IT, Hsu CJ, Chen YH, Fong YC, Hsu HC, Tsai CH. Fat embolism syndrome in long bone fracture: Clinical experience in a tertiary referral center in Taiwan. *J Chin Med Assoc* 2010;73:407-10.
3. Habashi NM, Andrews PL, Seale TM. Therapeutic aspects of fat embolism syndrome. *Injury* 2006;37:S68-73.
4. Pinney SJ, Keating JF, Meek RN. Fat embolism syndrome in isolated femoral fractures: Does timing of nailing influence incidence? *Injury* 1998;29:131-3.
5. Mueller F, Pfeifer C, Kinner B, Englert C, Nerlich M, Neumann C. Post-traumatic fulminant paradoxical fat embolism syndrome in conjunction with asymptomatic atrial septal defect: A case report and review of the literature. *J Med Case Rep* 2011;5:142.
6. Aravapalli A, Fox J, Lazaridis C. Cerebral fat embolism and the "starfield" pattern: A case report. *Cases J* 2009;2:212.
7. Gregorakos L, Sakayanni K, Hroni D, Harizopoulou V, Markou N, Georgiadou F, *et al.* Prolonged coma due to cerebral fat embolism: Report of two cases. *J Accid Emerg Med* 2000;17:144-6.
8. Parizel PM, Demey HE, Veeckmans G, Verstreken F, Cras P, Jorens PG, *et al.* Early diagnosis of cerebral fat embolism syndrome by diffusion-weighted MRI (starfield pattern). *Stroke* 2001;32:2942-4.

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