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Case report

# Pathogenesis, diagnosis and treatment of cerebral fat embolism

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ABSTRACT

In this study, we analyzed two cases of pure cerebral fat embolism and reviewed related literatures to explore the pathogenesis, clinical manifestations, diagnosis and treatment of cerebral fat embolism, improve the treatment efficiency and reduce the misdiagnosis rate. In our cases, patients fully returned to consciousness at the different times with good prognosis, normal vital signs and without obvious sequelae. For patients with the limb fractures, who developed coma without chest distress, dyspnea or other pulmonary symptoms 12 or 24 h post injury, cerebral fat embolism should be highly suspected, except for those with intracranial lesions, such as delayed traumatic intracerebral hemorrhage, etc. The early diagnosis and comprehensive treatment can improve prognosis.


## 1. Introduction

Fat embolism is caused by the lipid droplets in the blood circulation which block the small vessels, commonly seen in long bone fractures. In adipose tissue with severe contusion or fatty liver with crush injury, the lipid droplets are freely educed from the ruptured fat cells and enter the blood stream through the broken small vein and cause fat embolism. The severity of fat embolism depends on the size and quantity of lipid droplets, and the level of systemic involvement. Fat embolism mainly affects the lungs and nervous system. In 1970 Gurd[1] proposed the clinical diagnostic criteria for fat embolism: 1) The main criteria: subcutaneous hemorrhage; respiratory symptoms and lung X-ray manifestations; neurologic symptoms without craniocerebral trauma; 2) The secondary criteria: arterial partial pressure of oxygen lower than 8.0 kpa (60 mmHg); decreased hemoglobin (<100 g/L); 3) Reference criteria: fever; pulse greater than 120 beats/minute; breathing greater than 30 beats/minute; erythrocyte sedimentation rate greater than 70 mm/h; the decreased platelets; lipid droplets in the urine. The diagnosis of fat embolism can be confirmed by two main criteria above, or one main criterion plus four secondary and reference criteria. Sevitt divided the fat embolism into three types: eruption type, complete and incomplete types, in which the incomplete type includes pure cerebral type, pure pulmonary type and mixed type. Diagnosis of pulmonary and mixed types is easier, based on the pulmonary symptoms such as dyspnea, hemoptysis and lung wet rales, patchy shadows in the lung scan, hypoxemia, etc. But the cerebral fat embolism mainly manifests as disturbance of cerebral circulation, cerebral ischemia and consciousness and loss of nerve function; there are no manifestations of chest distress, dyspnea or other pulmonary manifestations, therefore it is easily misdiagnosed.

In this study, two cases of cerebral fat embolism were analyzed and the related literature was reviewed, to explore the pathogenesis and the effective treatment.

## 2. Case report

### 2.1. Case 1

A 25-year-old male was admitted to the department of ophthalmology of our hospital in August 25, 2012, who complained about left eye pain, vision loss, and right lower limb movement disorder for 13 h after a traffic accident. Physical examination showed normal mental status, irregular left pupil with disappeared light reflex, right pupil of 2.5 mm in diameter with fast pupillary light reflex. The examination of heart, lungs and abdomen showed no abnormalities. Right lower limb was fixed by external fixation with restrained movement. The results were negative in neurological examination. The X-ray performed in a local hospital the day

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before showed right tibial and fibular fractures. The brain CT in both our hospital and the local hospital showed no abnormalities. Left corneal debridement was performed at 10:00 am on August 26. All the operations were successful, and then the patient returned to the ward. At 13:50 pm of the same day, the patient presented dysphoria and unconsciousness. CT scans of head, neck and chest all were negative. The next day unconsciousness aggravated and patient was transferred to our department. Head MRI showed long $T_1$ and $T_2$ signals with diffuse punctuates, and high signal intensity on diffusion-weighted imaging (DWI) throughout bilateral cerebral cortex, white matter, basal ganglion, thalamus and brainstem (Fig. 1).

### 2.2. Case 2

A 25-year-old male suffered coma with intermittent convulsive movements of limb for 9 days after trauma and was admitted to our hospital on May 25, 2014. The patient was conscious at first after trauma and the CT performed in local hospital found no obvious signs of cerebral hemorrhage. However during the treatment, consciousness disturbance appeared and even developed deep coma, with frequent convulsion. Physical examination revealed deep coma, bilateral isocoria with about 3 mm in diameter, and fast pupillary light reflex. There was coarse sound in bilateral lungs and no distinct dry or wet rale. Both lower limbs were fixed by plaster cast. There was swelling and deformity in the right thigh. The X-rays from the local hospital showed right femoral comminuted fracture and bilateral tibial comminuted fracture; head MRI showed diffuse abnormal signals in bilateral cerebral hemisphere, callus, cerebellum and brainstem, which may reveal the hematogenous embolization (Fig. 2). After admission, symptomatic and supportive treatments were given to lower the intracranial pressure, protect brain, and prevent convulsion. Finally convulsion was stopped and consciousness recovered.

## 3. Discussion

### 3.1. The pathogenesis of cerebral fat embolism

Besides their ages, the two patients have several things in common: both suffered trauma, combined with lower extremity long bone fractures; both were conscious within 24 h after injury, then consciousness disturbance appeared and it developed into deep coma 24 h post injury; multiple head CT results were negative. In the hospital, both patients' respiration was stable without obvious dyspnea, chest distress or other pulmonary manifestations. Multiple blood gas analysis was normal. Considering that patients suffered trauma, obnubilation appeared and no evidence of intracranial hemorrhage was seen in multiple CT, diffuse axonal injury was suggested. The main characteristics of diffuse axonal injury are: after traumatic shearing forces that occur when the head is rapidly accelerated or decelerated, patients became coma or restless immediately after injury and the state will last a long time and recover slowly. A small number of the patients may have lucid intervals, and no clear signs of focal lesion in the nervous system. These lesions often cannot be found in head CT. While in head MRI they mainly manifest as diffuse edema of bilateral cerebral white matter, brain swelling, ambiguous boundary of gray matter, the narrowed or disappeared ventricle, cerebral pool, cerebral sulcus and subarachnoid space, absence of midline shift, diffuse brain swelling associated with diffuse punctuates throughout white matter and splinter hemorrhage; but no hematoma or space-occupying effect was found and thin layer of subdural hemorrhage may be accompanied with.

Our patients have a noticeable history of trauma, but without obvious shear stress, and they were conscious after injury, then progressed into coma during the treatment, 24 h after trauma. CT was negative before and after coma and the main clinical manifestations were confusion and unstable breathing. The results of head MRI were similar: long $T_1$ and $T_2$ signals with diffuse punctuates, and high signal intensity on DWI throughout bilateral cerebral cortex, white matter, basal ganglion, thalamus and brainstem. Because these clinical manifestations do not fully comply with the diffuse axonal injury, we considered that maybe some other diseases existed. Considering the patients' age and the history of trauma, hypertension-induced infarction, other chronic diseases, and intracranial infection (especially disseminated encephalitis) pose a small possibility. Due to a history of long bone fracture caused by trauma, a cerebral fat embolism should be suspected. Fat embolism often occurs in the lungs. A pure cerebral fat embolism is rare, and its pathogenesis is not fully understood.

Most literatures reveal that a fat embolism mostly occurs after a long bone fracture, especially after a femoral fracture. Gleich et al[2] reported that a 42-year-old healthy male patient suffered fractures

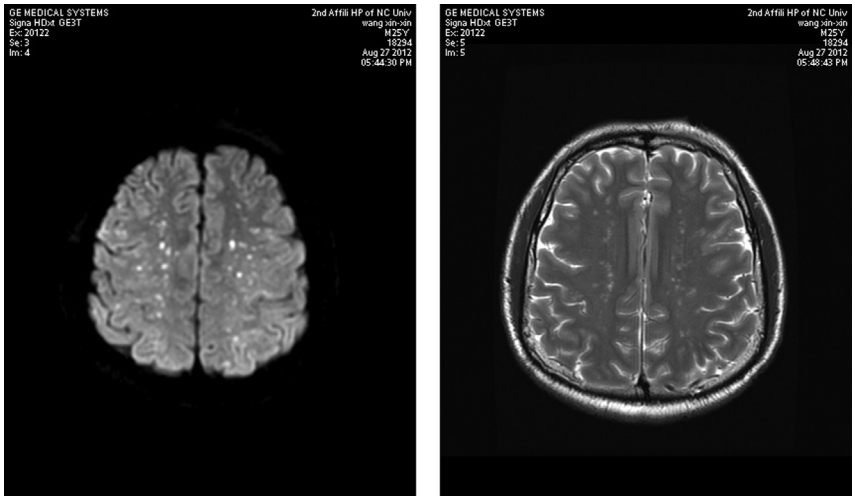


**Fig. 1.** Head MRI showed long $T_1$ and $T_2$ signals with diffuse punctuates, and high signal intensity on DWI throughout bilateral cerebral cortex, white matter, basal ganglion, thalamus and brainstem.