

Early Diagnosis of Cerebral Fat Embolism Syndrome by Diffusion-Weighted MRI (Starfield Pattern)

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Background—Cerebral fat embolism syndrome is a rare, but potentially lethal, complication of long bone fractures. Neurological symptoms are variable, and the clinical diagnosis is difficult. The purpose of this case study is to demonstrate the value of diffusion-weighted MRI of the brain for early diagnosis of fat embolism syndrome.

Case Description—A non-head-injured 18-year-old woman suffered acute mental status changes 21 hours after an uncomplicated fracture of the left tibia. MRI of the brain was performed 48 hours after injury. T2-weighted images showed multiple nonconfluent areas of high signal intensity, which, on the diffusion-weighted scans, were revealed as bright spots on a dark background (“starfield” pattern). We suggest that this indicates areas of restricted diffusion that are due to cytotoxic edema, resulting from multiple microemboli.

Conclusions—High-intensity lesions in the brain on diffusion-weighted images may serve as an early-appearing and more sensitive indicator of the diagnosis of fat embolism in the clinical context of long bone injury without head trauma. (*Stroke*. 2001;32:2942-2944.)

Key Words: brain ■ cerebral ischemia ■ embolism, fat ■ magnetic resonance imaging, diffusion-weighted ■ stroke, cardioembolic

We report the value of diffusion-weighted MRI (DW-MRI) in the early diagnosis of cerebral fat embolism syndrome.

Case Report

A previously healthy 18-year-old woman was injured in a car crash. She was admitted to the hospital with an uncomplicated closed fracture of the left tibia. The left leg was immobilized. Further clinical inspection revealed an oblique linear ecchymosis over the left jugular region, presumably secondary to a seat-belt injury. There was no evidence of head injury. The patient was fully conscious. Respiratory and hemodynamic status was stable.

Next morning, 21 hours after the accident, the patient's consciousness suddenly deteriorated. She became restless, confused, and unresponsive to verbal stimuli. She suffered emotional outbursts, alternately crying and uttering incomprehensible speech, with tachypnea and tachycardia. No focal neurologic deficit was observed. There was no previous history of hysteria. Toxicology screening revealed no evidence of drug abuse. The Glasgow Coma Scale was 11 of 15 (eye opening, 4; motor response, 2; and verbal response, 5). Blood gas analysis showed mild hypoxemia, with a PaO_2 of 78 mm Hg and a Paco_2 of 33 mm Hg, with 21% oxygen

respiration. She was transferred to the Intensive Care Unit, where supportive treatment was given, and minimal hemoglobin and platelet levels were monitored. Duplex Doppler ultrasonography of the neck vessels was performed to exclude traumatic dissection but was found to be normal. A noncontrast CT scan of the brain showed no abnormalities.

Forty-eight hours after the motor vehicle crash, MRI of the brain was obtained (Figure 1). T1-weighted images were normal. Magnetic resonance angiography showed no evidence of dissection or thrombosis. However, on the T2-weighted images, multiple nodular or punctate foci of high signal intensity were found within the brain parenchyma. The lesions were located in the white matter (subcortical white matter and centrum semiovale) as well as in the gray matter (basal ganglia and thalami). There were no signal abnormalities in the posterior fossa. On the DW-MRI trace images, with a b value of 1000 mm^2/s , these lesions were seen as high signal intensity dots on a dark background. This resulted in a “starfield” appearance of the cerebral hemispheres. The diagnosis of cerebral fat emboli was proposed. Bronchoalveolar lavage was not performed because of the invasive nature of this procedure in a nonintubated patient.

On day 3 after admission, a petechial rash was noted on the anterior chest wall and in the axillary regions. At that time,

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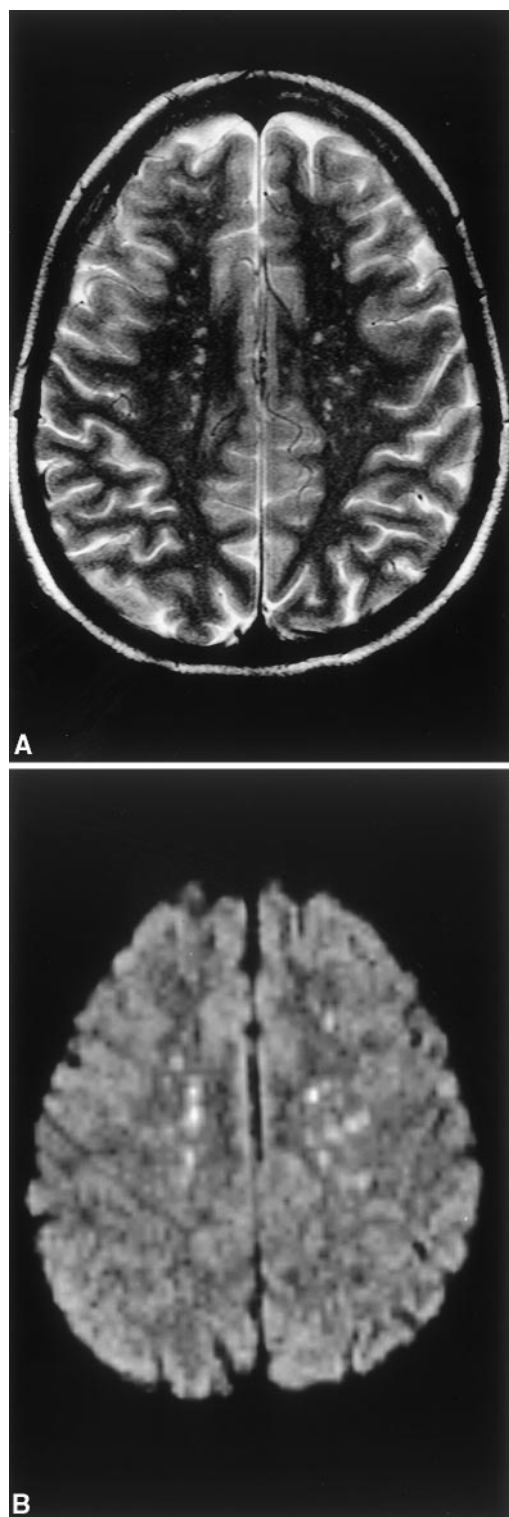


Figure 1. Axial T2-weighted MRI (A) and DW-MRI (B) scans through the centrum semiovale, obtained 48 hours after injury. The T2-weighted image (A) shows multiple punctiform hyperintense lesions in the white matter of both cerebral hemispheres. The diffusion-weighted sequence (B) reveals that several of these lesions are of high signal intensity, indicating areas of restricted diffusion due to cytotoxic edema.

the patient developed a partial right facial nerve paralysis. Fundoscopy was normal. Fat globules were not seen in either the urine or the sputum.

The patient's Glasgow Coma Scale started to improve gradually on day 4 after admission, and by day 7, she had regained full consciousness. Clinical neurologic examination was back to normal, and the facial nerve paralysis had completely resolved. She was discharged from the hospital on day 8 in excellent clinical condition.

A follow-up MRI examination of the brain was obtained after 4 weeks (Figure 2). All signal abnormalities in the basal ganglia had disappeared. The number of white matter lesions in the centrum semiovale had significantly decreased. Most important, there were no signal abnormalities on the DW-MRI.

Discussion

The term fat embolism syndrome refers to a clinical entity that consists of pulmonary, central nervous system, and cutaneous manifestations.^{1,2} It is an uncommon, but potentially life-threatening, complication of long bone fractures.^{1,3–6} Hypoxia, deteriorating mental status, and petechiae are the main diagnostic criteria; secondary diagnostic signs include tachycardia, fever, anemia, and thrombocytopenia.⁷ The incidence of fat embolism syndrome after bone fractures is in the range of 0.9% to 2.2%.⁸ The pathogenesis remains controversial, and several theories have been proposed.⁸ Fat emboli can pass through the pulmonary vasculature, resulting in systemic embolization, most commonly in the brain and kidneys.⁹ Cerebral manifestations of fat embolism syndrome are highly variable and nonspecific: headache, lethargy, irritability, delirium, stupor, convulsions, or coma.² Many cases occur as subclinical events and remain undiagnosed.^{3,9}

Clinical diagnosis of cerebral fat embolism syndrome can be aided by noting the presence of respiratory failure, hypoxemia, and cutaneous petechiae.⁶ Neurological dysfunction varies from confusion to encephalopathy with coma and seizures.¹⁰ Blunt carotid artery injury and vertebrobasilar artery thrombosis should also be considered in the differential diagnosis of acute mental status change in non-head-injured patients.¹¹

The neuroradiological diagnosis of cerebral fat embolism is equally challenging. Cerebral CT scans are usually negative.^{5,12,13} MRI is more sensitive and consistently shows multiple small, scattered, nonconfluent hyperintense intracerebral lesions on T2-weighted scans.^{3,4,12–14} Signal abnormalities occur in both gray and white matter.^{13,15} Their number correlates with the Glasgow Coma Scale.⁴ Lesions gradually disappear within a few weeks to a few months.^{4,13} However, T2-weighted MRI scans are of limited help in the (hyper-)acute phase because the abnormalities may take several days to develop, and the findings remain highly nonspecific.¹² Differential diagnosis of disseminated hyperintense lesions on T2-weighted scans includes diffuse axonal injury, areas of vasogenic edema associated with microinfarcts, foci of gliosis, dilated perivascular Virchow-Robin spaces, and demyelinating disease.¹² Our case demonstrates that the addition of DW-MRI may enhance the sensitivity and specificity of the

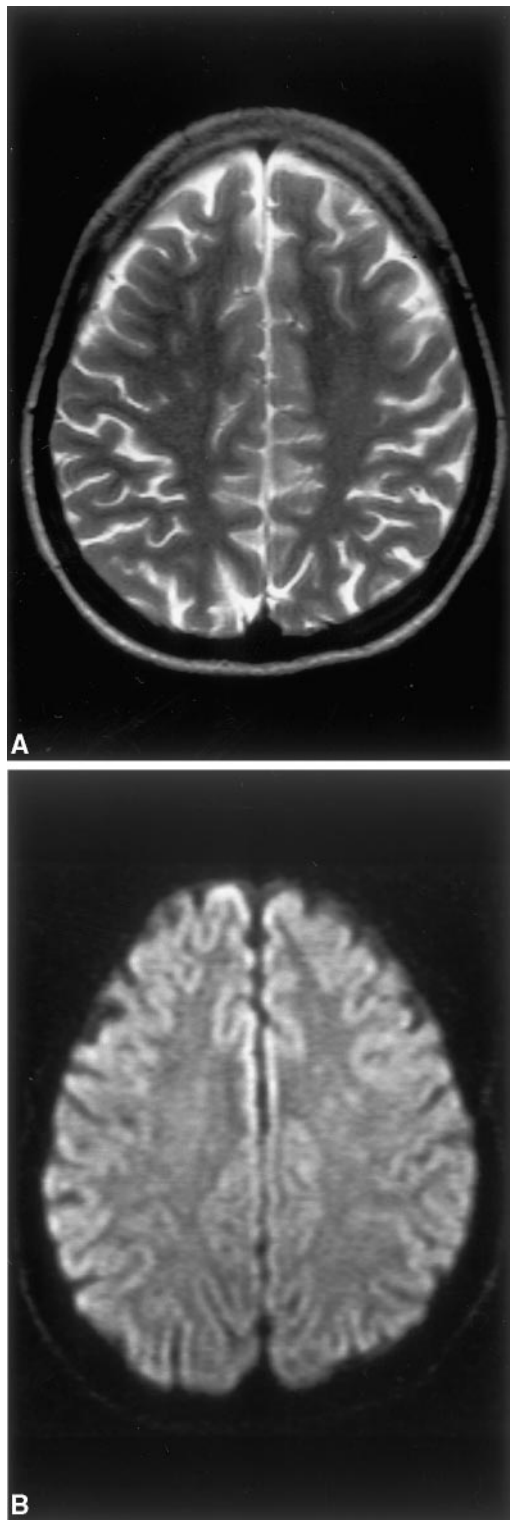


Figure 2. Follow-up axial T2-weighted MRI (A) and DW-MRI (B) scans obtained 4 weeks after the accident. On the T2-weighted image (A), only a few hyperintense lesions remain, presumably reflecting gliotic scars. The diffusion-weighted image (B) is normal.

neuroradiological diagnosis by the presence of the starfield pattern of scattered bright spots on a dark background.

Alternative diagnostic methods for revealing decreased cerebral blood flow in the acute stage of cerebral emboli are

[^{99m}Tc]hexamethylpropylenamine oxime single-photon emission CT or transcranial Doppler sonography.^{5,16} However, the degree of confidence in pattern recognition and the spatial resolution provided by these methods are significantly lower than they are for DW-MRI.

In conclusion, any non-head-injured trauma patient who is initially lucid and subsequently develops an acute mental status deterioration should undergo immediate evaluation for possible cerebral fat embolism or neck vessel injury.¹¹ We advocate that DW-MRI of the brain should become the first step in the diagnostic algorithm to rule out cerebral fat embolism. We consider the starfield pattern of scattered bright spots to be pathognomonic of acute cerebral microinfarcts; the signal intensity abnormalities presumably reflect foci of cytotoxic edema. T2-weighted images may also show high signal abnormalities in gray and white matter, but the lesions take longer to become evident. Areas of increased signal intensity on T2-weighted scans presumably reflect vasogenic edema, which develops at a later stage, whereas DW-MRI reveals the cytotoxic edema, which develops immediately.

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