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BRIEF REPORT

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# Magnetic resonance imaging of cerebral fat embolism: a case report

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**Abstract** Fat embolism syndrome (FES) is one of the most important causes of morbidity and mortality following multiple fractures. Neurological involvement (cerebral fat embolism) has been reported frequently. A case of cerebral fat embolism is reported. While CT scan revealed no abnormalities, MRI, performed in this patient 8 days after trauma, showed relative low-intensity areas on T1-weighted images and high intensity areas on T2-weighted images involving cerebral white matter, corpus callosum and basal ganglia. MRI follow-up (1 and 3 months post-trauma) showed nearly complete resolution of the abnormal signal. MRI seems to be a useful diagnostic tool for detecting and quantifying lesions in fat embolism syndrome.

**Key words** Fat embolism · Magnetic resonance imaging

## Introduction

Fat embolism syndrome (FES) is one of the most important causes of morbidity and mortality following multiple fractures. Although fat embolism is at least subclinically present after all the fractures of long bones, it's clinical incidence has been reported at 0.5–11% of fractured patients [1] with an associated 10% mortality [2]. The full clinical picture, first described by Von Bergman in 1873, is characterised by: pulmonary insufficiency with hypoxemia and tachypnea, neurological dysfunction, pyrexia, tachycardia and petechiae occurring 12 to 48 h after trauma. Neurological disorders have been reported in 84% of the diagnosed cases, usually preceding the pulmonary dysfunction by 6 to 12 h [3]. While CT scan performed on the second day from clinical onset did not reveal focal brain lesions but only cerebral oedema, MRI, obtained 8 days after trauma, showed diffuse lesions seen as areas of hyperintensity on T2-weighted images and areas of relative hypointensity on T1-weighted images. MRI follow-up examination (1 and 3 months post-trauma) showed a marked reduction of signal alterations. MRI seem to be an useful diagnostic tool for detecting brain lesions in fat embolism syndrome.

## Case report

A 17-year-old white male fractured his femur and humerus in a motor-bike accident. Upon admission in a Regional Hospital, the patient was alert, well oriented, normotense and eupnoeic; there was no direct blow to his head. After transcheletric traction positioning, the patient was hospitalised in an Orthopaedic division. At 12 h post trauma, the patient was suddenly agitated, confused and hypotense. Glasgow coma score was 4 (E1, V1, M2); pupils were isocoric, isocyclic and reactive. Mass expansion, initially plasma expanders and successively blood and plasma, was immediately established and the patient was intubated. The blood gas analysis after intubation (Pressure support ventilation, $FIO_2$ 0.4, PEEP 5 $cmH_2O$) was $PO_2$ 112 mmHg, $PCO_2$ 30.1 mmHg, pH 7.33, BE −7.5 mmol/l. Chest X-ray was unremarkable. Brain CT scan performed 2 h later failed to reveal any abnormality: no visible lesions, normal ventricular system, perimesencephalic cisterns not compressed. On the second day, Glasgow coma score was 5 (E2, V1, M2). The patient underwent closed reduction of his fractures. Before and during surgery he was transfused with 4 units of PRBC and 2 units of FFP. Another CT scan performed before surgery revealed diffuse brain oedema; antioedema therapy was instituted.

The blood gas analysis on the seond day (Pressure support ventilation) at $FIO_2$ 0.3 was $PO_2$ 69 mmHg, $PCO_2$ 40.1 mmHg; at $FIO_2$ 0.4 $PO_2$ went to 140 mmHg. Pulmonary compliance was 66 ml/cm$H_2O$. Platelets dropped from 160000/ml (1st day) to 59000/ml (day 3); no petechiae were noted. In the following days the patient was clinically stable. Auditory and somatosensorial evoked potentials were normal and electroencephalogram showed diffuse electrical alterations. Cerebral angiography was performed and it didn't reveal any vascular abnormality.

On day 8 the patient was transferred to our Hospital. GCS upon arrival was 7 (E 4, V 1 T, M 2); the patient showed hypertone and pyramidal signs of hyperreflexia. He was intubated and ventilated (PS 7 cm$H_2O$ above PEEP, $FIO_2$ 0.4, PEEP 5 cm$H_2O$); arterial blood gas analysis was $PO_2$ 176 mmHg, $PCO_2$ 38.3 mmHg. A MRI study (Siemens Magnetom 1.5 Tesla) was executed. During the MRI studies, the patient was monitored with EKG and Pulse-Oxymetry (Nellcor 100); arterial pressure was measured with Dinamap and we ventilated him with an amagnetic volumetric ventilator (Monaghan 225 SIM V).

MRI demonstrated multiple hyperintense abnormalities on the T2-weighted images in the cerebral white matter (Fig. 1), basal ganglia, corpus callosum and cerebellar hemispheres corresponding to multiple hypointense areas on T1-weighted images. MRI provided a confirmation of clinical suspicion of cerebral FES.

Ophthalmoscopy revealed cottonwool spots along the vascular arcades bilaterally. Electroencephalogram (slow asymmetric waves) and auditory and somatosensorial evoked potential (normal) were performed. A transesophageal echocardiography was successively performed demonstrating a mild tricuspid insufficiency and no interatrial defects.

The patient was supported in CPAP until day 10 and extubated on 12th day. GCS after extubation was 8 (E 4, V 1, M 3). The patient obeyed commands 1 month after trauma. MRI follow-up performed at 1 and 3 months showed a reduction of signal alterations: only areas of subtle hyperintensity were still evident in cerebral white matter in T2-weighted sequences (Fig. 2). Glasgow outcome score at 3 months was "Moderate disability".

## Discussion

The classic fat embolism syndrome consists of long bone fractures, a 12–48 h symptom free period followed by respiratory insufficiency, petechial rash, neurological involvement [4]. Additional features are pyrexia, tachycardia, RBC reduction, coagulopathy, ESR raise, retinal cotton-wool spots. However, the frequent partial syndrome makes the diagnosis difficult in many cases.

Neurological involvement in fat embolism syndrome is very frequent (84% of diagnosed cases; [3]). It takes form of confusion or encephalopathy progressing to coma and/or focal signs.

Classic histological lesions of cerebral fat embolism are described as ball, ring and perivascular haemorrhages [9]. Characteristically, these minute haemorrhages are most abundant in the centrum semiovale, digitate white matter, internal capsule and cerebellar white matter. Also anaemic infarcts were seen. Fat was demonstrated within some haemorrhagic and anaemic infarcts and in small vessels.

When a posttraumatic neurological deterioration occurs without head injury, it is necessary to consider the possibility of cerebral fat embolism. CT scan is a very useful tool in excluding traumatic cerebral involvement and in differentiating this more frequent condition from cerebral fat embolism. Cases of cerebral fat embolism studied by CT scan have been reported normal or showing diffuse oedema [2, 5, 6]. More rarely low density areas in the white matter with haemorrhages were described. In this case the first CT scan, performed 2 h after the initial clinical signs, failed to reveal any abnormality. The sec-

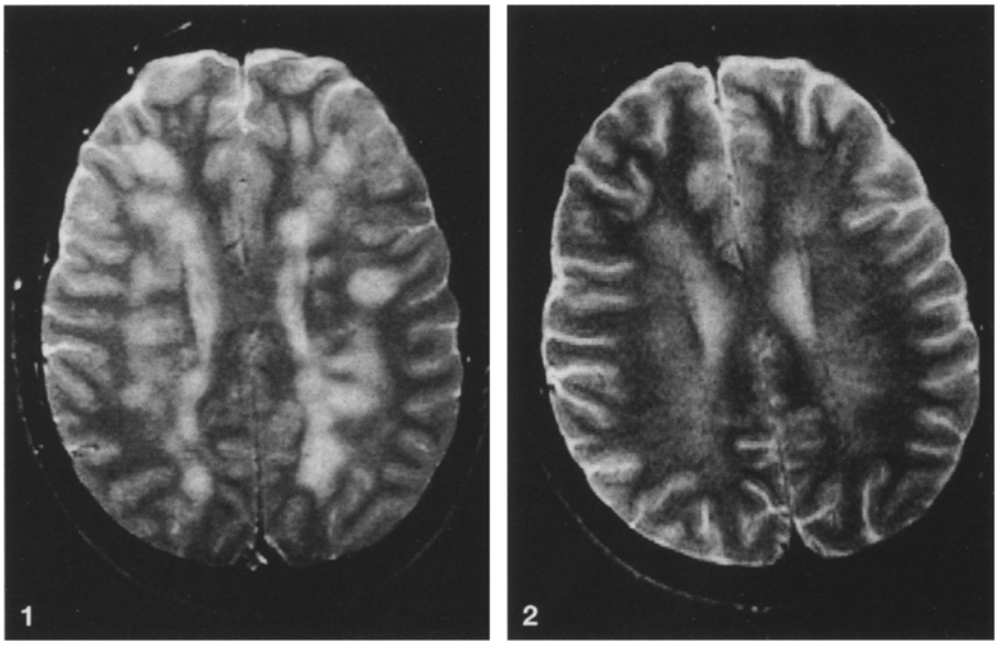


**Fig. 1** MR performed on day eight from trauma. Axial T2-weighted image. Multiple hyperintense areas of various size are evident in the hemispheric deep and subcortical white matter

**Fig. 2** Axial T2-weighted image. MR performed 3 months after fat embolism showed a marked reduction of signal alterations in cerebral white matter

ond CT, performed 18 h after, revealed diffuse brain oedema.

MRI has been shown to be more sensitive in detecting degenerative and vascular injuries and non haemorrhagic contusion. However, the usefulness of MRI as a diagnostic tool in the evaluation of cerebral fat embolism has not been fully explored. To our knowledge, a very small number of case reports utilising MRI have been published [7, 8] although MR studies were not performed in the acute phase. The MR findings of cerebral fat embolism are described as slightly hypointense signal alterations on T1-weighted images and hyperintense signal alterations on T2-weighted sequences. Petechial haemorrhages were not seen because they might have been too small [7, 8]. Some authors believe that the hyperintense areas on T1-weighted images may represent haemorrhagic infarcts while those seen in T2-weighted images may represent anaemic infarcts [8]. It is probable that fat globules in both situations (infarcts or occluded vessels) are too small to be detected on T1-weighted images.

In this case, the characteristics (hypointense signal alterations on T1-weighted images) and the localisation of lesions (cerebral deep white matter, basal ganglia, corpus callosum and cerebellar hemispheres) are in complete accordance with previous reports [7, 8].

At the 3 months MR follow-up, we found an almost complete resolution of previously demonstrated lesions. Kawano et al. [7] reported complete resolution of a milder form of cerebral fat embolism 8 weeks post-trauma explaining that reversible ischemia and oedema were of a sufficient degree to make the lesions visible at the acute stage and that the sequelae were too small to be seen later. This explanation seem to be reasonable also in this case.

MRI seems to be a very useful diagnostic tool in the acute case and during recovery for detecting and quantifying lesions in cerebral fat embolism syndrome.

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