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Case Report

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Fat Embolism with no Specific Clinical Presentation: Multimodal MRI is the Solution

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1. Abstract

Fat embolism syndrome (FES) is a rare syndrom which leads to systemic inflammatory cascade affecting multiple organ systems, with high morbidity and mortality rate. It is caused by embolization of fat particles into multiple organs including the brain. No symptom or investigation is completely specific and cerebral multimodal MRI may be the solution.

We report the case of a previously healthy 19-year old male with closed midshaft femoral fracture after an unrestrained head-on motor vehicle collision. He was on admission and during 12 hours, fully awake, and hemodynamically stable and within biological abnormalities.

Eighteen hours later, the patient developed acute respiratory failure with tachypnoea, desaturation and tachycardia and developed neurological manifestation with a Glasgow Coma Scale (GCS) score at 3, requiring intubation and ventilation.

The diagnosis of Fat embolism syndrome was suspected and was confirmed by an MRI. Fluid-Attenuated Inversion Recovery (FLAIR) sequence MRI showed confluent hyperintenselesions in the white matter. The lesions were distributing dmainly in the bilateral border-zone areas, corpus callosum; bilateral thalami, central semi-oval and grey-white matter junctions. All these lesions are of restricted diffusion, pleading in favor of a cytotoxic edema. MR spectroscopy showed a lipid and a lactate peak, particularly in the short TE images.

Based on clinical features and initial MRI, the diagnosis of cerebral FES was admitted, and supportive care therapies were started.

MR spectroscopy can represent a help-tool in accurate diagnosis by identifying the extra cerebral fat in these corresponding lesions.

2. Introduction

Fat embolism syndrome (FES) is a rare syndrome caused by embolization of fat particles into multiple organs, typically within the first three days, after a fracture of the long bones.

The pathophysiology of FES remains unclear with no specific clinical presentation.

Cerebral Multimodal MRI may be the solution to recognize this pathology and to start symptomatic treatment.

3. Case Report

A previously healthy 19-year old male was brought to emergency department (ED) after an unrestrained head-on motor vehicle collision. The patient's injuries included closed midshaft femoral fractures which had been temporarily fixed with a plaster cast.

There were no thoracic, abdominal, or pelvic abnormalities upon initial examination. Additionally, the patient had a normal neurologic status with an initial GCS at 15. Initial vitals exam showed a heart rate at 72/mn, a respiratory rate at 18/mn, with oxygen saturation at 99% under 2L of oxygen. The initial hemoglobin level was at 14.8l g/d, and platelet count was within normal limits (192000/mm3). The head computed tomography (CT) scan was normal. On admission and during 12 hours, he was fully awake, and hemodynamically stable.

4. Outcome and Follow Up

Eighteen hours later, the patient developed acute respiratory failure with tachypnoea, desaturation and tachycardia. Secondary, the patient developed neurological manifestation with irritability,

confusion and became progressively unconscious with a Glasgow Coma Scale (GCS) score at 3, requiring intubation and ventilation. Clinical examination showed Petechial rash on the upper chest, neck and conjunctiva. Biological parameters showed a fall of hemoglobin (7.8g/dl) with evident thrombopenia (78000/mm3) .The fundus examination showed a papillary edema. The diagnosis of Fat embolism syndrome was suspected and was confirmed by an MRI. Fluid-Attenuated Inversion Recovery (FLAIR) sequence MRI showed confluent hyperintense lesions in the white matter (Figure A, B). The lesions were distributing mainly in the bilateral border-zone areas, corpus callosum; bilateral thalami, central semi-oval and grey-white matter junctions and they are type 2A patterns. All these lesions are of restricted diffusion, pleading in favor of a cytotoxic edema.

MR spectroscopy showed a lipid and a lactate peak, particularly in the short TE images (Figure C, D).Based on clinical features and initial MRI, the diagnosis of cerebral FES was admitted, and supportive care therapies were started.

Under symptomatic treatment, the evolution was marked by improvement of neurological, respiratory and hematological parameters, 10 days after ICU admission. He was discharged from ICU 15 days later with a good neurological status.

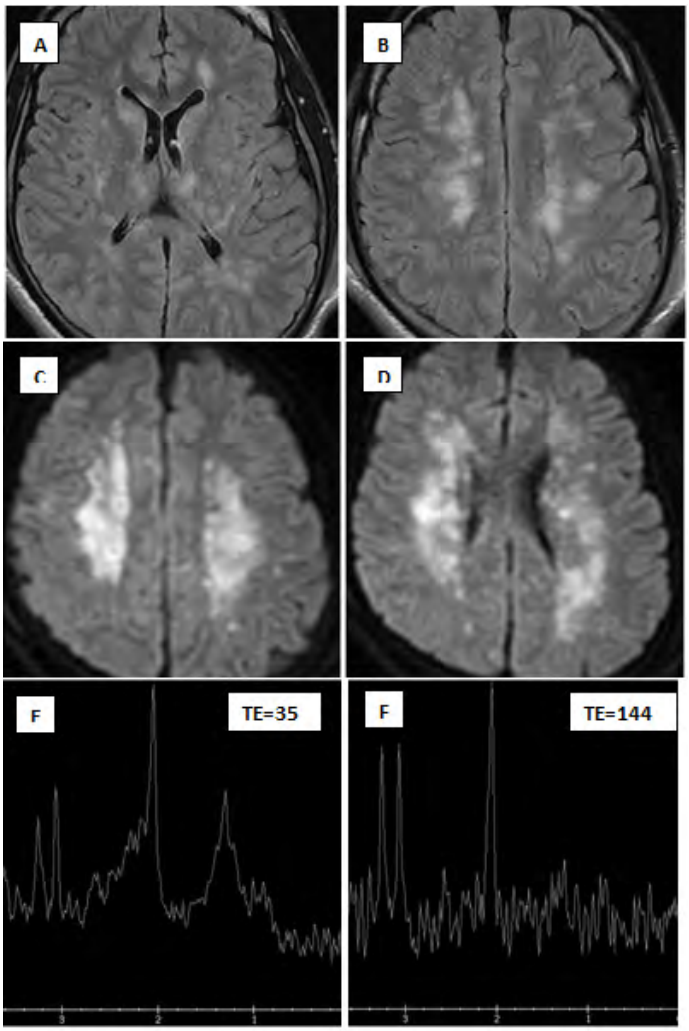

Figure 1: Magnetic resonance (MR) imaging: Fluid-attenuated inversion recovery (FLAIR) [A, B] sequences showing multiple focal area of increased signal and of bilateral and symmetrical distributionin the periventricular white-matter of the frontal and parietal lobes, in corpus callosum, in the semi-oval center and in the central gray nuclei. The fact that all these lesions are of restricted diffusion [C, D] pleads in favor of a cytotoxic edema. **MR spectroscopy:** elevated lipid peak (1.3 ppm) in the short TE, without elevated lactate peak [E, F].

5. Discussion

Our observation confirms that MR spectroscopy can represent a help-tool in accurate diagnosis by identifying the extra cerebral fat in these corresponding lesions. The lipid peak, particularly in the short TE images, confirms the presence of fat in the circulation and the lactate peak suggests the anaerobic metabolism due to blockage of circulation by fat globules [1].

Fat embolism syndrome (FES) is a rare syndrome (0.5% to 12.5%) caused by embolization of fat particles into multiple organs including the brain. This leads to systemic inflammatory cascade affecting multiple organ systems, with high morbidity and mortality rate. No symptom or investigation is completely specific in this multi-system disorder. It is a serious multi-system pathology.

As a consequence, early diagnosis and treatment improve outcome in this specific condition. MR imaging is the diagnostic modality of choice in evaluations of patients with CFE.

This syndrom was firstly diagnosed in 1861 by Zencker who described fat droplets in the lung capillaries of a rail road worker after thoraco-abdominal trauma. FES was first described following fracture of long bones by Bergmannin 1873, as a triad of confusion, dyspnoea, and petachiae [1]. It is usually associated with fracture of the long bones, and is most often seen within the first three days, after the injury [2].

It typically manifests with petechial rash, deteriorating mental status, and progressive respiratory insufficiency, usually occurring within 24-48 h of trauma with long-bone fractures or an orthopedic surgery [2].The pathophysiology of FES remains unclear. FES mechanisms include the histotoxic effects of free fatty acids, from the bone marrow or produced during inflammation, which altering capillary permeability and causing cytotoxic edema and ischemic changes [2].

Typical neurological complaints include headache, acute-onset confusion, disorientation, weakness, convulsions and even coma. Most neurological issues are transient and often completely reversible.

Cerebral CT is often the first imaging performed in acute neurological dysfunction, being fast and easily accessible. CT in the acute phase may occasionally identify low attenuation areas of focal or generalized edema and high attenuation focal areas of hemorrhage. However, up to this day, MR imaging represents the choice diagnostic modality for CFE. Reported findings include patchy or confluent edema changes, sometimes with restricted diffusion and enhancement on post contrast evaluation. The lesions usually involve the watershed zone, deep grey matter and white matter; less commonly, there is involvement of the corpus callosum, cerebral peduncles and posterior internal capsule. There may also be petechia hemorrhages in the white matter distribution [1]. The lesion's number is correlated with the Glasgow Coma Scale. And they gradually disappear within a few weeks to a few months [1]. However, T2-weighted MRI scans are of limited help in the (hyper) acute phase because this abnormalities may takes several days to be developed, and the findings remain highly non specific [2]. Moreover, the previously described changes are not specific to fat emboli and may be seen after systemic embolization, disseminated infections, areas of vasogenic edema hypoxic leukoencephalopathy, toxic leukoencephalopathy, hypoglycemic encephalopathy and diffuse axonal injuries [1]. As consequence, MR spectroscopy may be help in accurate diagnosis by identifying the extra cerebral fat in these corresponding lesions. The lipid peak, particularly in the short TE images, confirms the presence of fat in the circulation and the lactate peak suggests the anaerobic metabolism due to blockage of circulation by fat globules [1]. These findings were also seen in our case.

We must mention that the diagnosis of cerebral FES should be suspected in all patients with orthopedic trauma without head injury and who develop neurological manifestations few hours after the trauma. Early recognition of cerebral FES is important for appropriate medical management and to avoid adverse outcomes and improve prognosis. Immobilization of the fracture site, maintenance of intravascular volume, hemodynamic, and ventilatory support are the main elements of treatment [3].

6. Conclusion

Cerebral involvement of FES is a severe complication of bone-trauma. Patients suffering from FES require recognition and supportive management in an appropriate setting. MR spectroscopy can represent a help-tool in accurate diagnosis by identifying the extra cerebral fat in these corresponding lesions. The lipid peak, particularly in the short TE images, confirms the presence of fat in the circulation and the lactate peak suggests the anaerobic metabolism due to blockage of circulation by fat globules.

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