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CASE REPORT

Fatal cerebral fat embolism associated with a patent foramen ovale

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ABSTRACT
Fat embolism syndrome (FES) has been recognized for more than 100 years, but controversy remains with regard to the mechanism of systemic fat embolism. We report a case of healthy 30-years-old men who developed respiratory failure and brain death within 48 h of bilateral tibial fractures. Autopsy showed fat embolism in the lung and brain, and a large patent oval foramen that may have contributed to massive cerebral fat embolism.
Keywords: Cerebral fat embolism; Fat embolism syndrome; Patent foramen ovale.

Introduction

Fat embolism syndrome (FES) remains a rare, but potentially life threatening complication of long bone fractures. The true incidence of this syndrome cannot be accurately assessed as many subclinical forms remain unrecognised. It varies from 0.5 % to 30 % of fractured patient [1]. Cerebral involvement has been frequently reported and seems to aggravate the prognosis of FES [2].

Observation

A 30 year old man, victim of bilateral opened tibia fractures, without head injury, by a traffic accident, was admitted at emergency wall. During admission, the patient was alert, oriented, normotensive, and eupnoeic. Neurological examination showed no abnormalities. Two hours later, external fixation of his fractures was performed under general anesthesia. After complete recovery from anesthesia, he was transferred extubated to the ICU; he was haemodynamically stable and had a normal respiratory pattern. At the postoperative twelfth hour, he developed signs of FES including fever (more than 38° C), hypoxemia, oliguria. He was unconscious, responding only to painful stimuli. Neurological examination showed a Glasgow coma score of 7 (eye opening 2, motor response 3, verbal response 2), while pupils were equal in size and reactive to light. An immediate endotracheal intubation and mechanical ventilation were performed because of unconsciousness and respiratory insufficiency. Chest radiography showed diffuse pulmonary infiltrations. Electrocardiogram showed sinus tachycardia and diffuse nonspecific ST-T wave segment abnormalities. Cerebral computed tomography, performed shortly after mental status deterioration, revealed diffuse brain edema. Laboratory analysis revealed anemia. Arterial blood gas analysis showed a partial oxygen tension of 60 mmHg and partial carbon dioxide tension of 30 mmHg. Retinal examination demonstrated characteristic cotton wool spots across the vascular beds, which are indicative of fat embolism. The patient remained under mechanical ventilation, general supportive care in the ICU, and regular neurosurgical evaluation.

Forty eight hours after hospital admission, the pupils suddenly became fixed and dilated. A repeated computed tomographic scan of the head showed loss of the cerebral sulci and basal cisterns consistent with cerebral edema, but no focal abnormalities. Numerous petechiae appeared on the upper part of the trunk (Photo N°1) and conjunctivae (Photo N°2). The criteria for brain death were fulfilled and the patient died.

Autopsy showed acute lung congestion and edema with numerous fat emboli in the small arteries and capillaries. There was a patent foramen ovale (PFO) with a diameter of 1 cm. In the brain, numerous fat emboli were surrounded by areas of acute infarction. There was no gross or microscopic evidence of traumatic brain injury. Edema arising from the infarctions caused raised intracranial pressure, uncal herniation, cerebellar tonsil herniation, and death.

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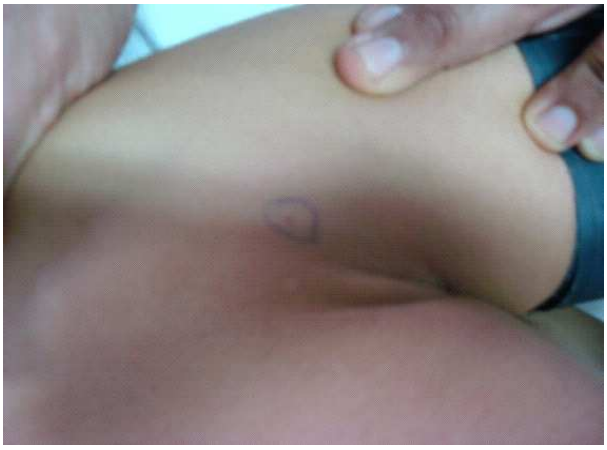

Photo N°1: Petechiae on the upper part of the trunk

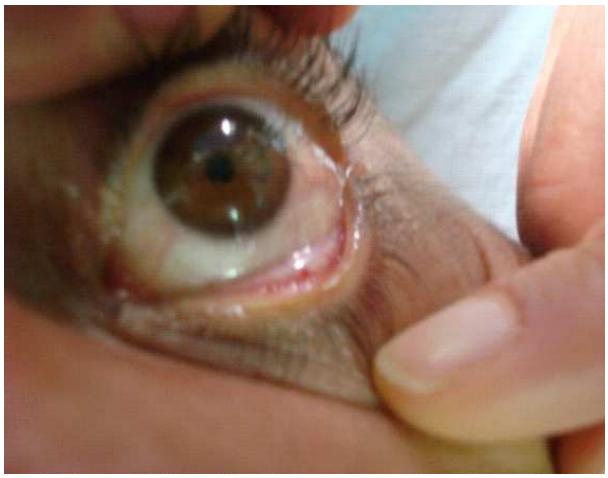

Photo N°2: Petechiae in the conjunctivae

Discussion

Clinical manifestations of cerebral fat embolism (CFE) are highly variable and nonspecific: headache, lethargy, delirium, confusion, convulsion, coma [3].

In our patient the diagnosis was established by Gurd's criteria [2], the characteristic fat globules on retinal examination, and the findings on brain computed tomography. The pathogenesis of CFE remains controversial, and is thought to be multietiologic. The cause of neurological dysfunction induced by fat embolism was previously thought to be secondary to hypoxia due to respiratory distress or intracranial hypertension. More recently, however, several theories have been proposed to explain the cause of neurological dysfunction. The first is mechanical: intraluminal fat globules smaller than 7 µm in diameter can pass through the pulmonary arteriolar network which enters directly into the brain causing a blockage of capillary blood vessels [4]. The second is toxic: fat globules activate chemical mediators that alter capillary permeability [5]. Microscopically, fat embolisms are observed mainly in the gray matter because of its abundant capillary network, and the pathologic changes are predominant in the white matter because of its poor collateral circulation and susceptibility to ischemia [6].

On postmortem examination, victims of the fulminant form of the syndrome present occlusion of small blood vessels by fat emboli, with areas of brain micro infarction and hemorrhage [4]. Due to cerebral fat emboli, the brain often appears edematous and shows an inflammatory reaction while numerous petechiae can cover the surface of the brain [5]. Endothelial damage results from toxic free fat and capillary obstruction by fat globules with associated platelet aggregation, release of vasoactives substances, and development of coagulopathy.

The presence or the reopening of a patent foramen ovale and a right to left shunt due to pulmonary hypertension is associated with an increased risk for systemic manifestations of FES [8].

Most patients with fat embolism syndrome recover fully. Mortality is usually cited as 5%–15%. Among several pharmacological treatments, only steroids have proved to be beneficial in the prophylaxis and treatment of FES, both in high and low doses [9]. Their mode of action has not been elucidated, but seems to be related to their anti-inflammatory and antiadhesive effects. Unfortunatly our patient did not received steroids because of its rapid death.

Many authors have suggested that early open reduction and surgical stabilisation of long bone fractures may reduce the incidence of FES [8]. Repeated manipulation of the fractured fragments may further stimulate the release of bone marrow fat into the circulation [8]. Similarly, the type of surgical fixation seems to influence the incidence of fat embolisation. External fixation and plate osteosynthesis have several advantages compared with intramedullary nailing techniques, which further increase intramedullary pressure and promote fat emboli release [8]. However, the syndrome is frequently observed despite prompt surgical intervention such as our case.

As physiopathology of the syndrome still remains obscure, attention should be paid to prevention and early recognition of this entity. Optimal immobilization, adequate fluid and blood replacement, meticulous monitoring, and use of steroids are principles commonly accepted in current treatment [8].

Conclusion

In summary, we reported a case of fatal cerebral fat embolism in a patient with a large PFO. We speculate that right to left shunting through the PFO may have been an important contributing factor to cerebral fat embolism.

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