CASE REPORT

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Cerebral Fat Embolism Syndrome – Pathogenesis and Treatment

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Abstract

We describe a case of cerebral fat embolism – which developed 3 days after roadside accident, in a young boy who suffered limb fracture of femur and tibia. In cerebral fat embolism that manifests chiefly as disturbances of cerebral circulation and ischaemia, chest dyspnoea and pulmonary manifestations are strikingly lacking leading to misdiagnosis or delay in its diagnosis. The present case chiefly developed cerebral fat embolism syndrome. Early recognition and comprehensive management is warranted for a better outcome. Aetiopathogenesis, presentation, diagnosis and treatment of cerebral fat embolism is briefly described and discussed.

Key words: Cerebrum, fat embolism, limb fracture.

Introduction

Fat embolism is defined by the presence of lipid droplets in the blood circulation blocking the small vessels. The term fat embolism syndrome refers to the clinical scenario following an insult that releases fat droplets in the circulation with resultant pulmonary and systemic involvement. The severity of fat embolism depends on the size and quality of the lipid droplets, and the level of systemic involvement – lung, brain. or both. The diagnosis of pulmonary or mixed type (pulmonary and brain) is often easier as pulmonic features like dyspnoea, haemoptysis, wet rales in the lungs, patchy shadows on imaging studies, hypoxaemia, etc., are recognised easily; while central fat embolism manifests as disturbances of cerebral circulation that typically manifests in isolation from development of acute confusional state/ altered level of consciousness to seizures and focal defects[1,2]. In cerebral fat embolism, that manifests chiefly as disturbances of cerebral circulation and ischaemia, chest distress, dyspnoea and pulmonary manifestations are strikingly lacking, leading to misdiagnosis or delay in its diagnosis.

Case report

A 24-year-old male was hospitalised at the department of orthopaedics following a traffic accident. History of concussion, seizures, vomiting were denied. Physical examination revealed normal mental status with normal pupillary and corneal reflexes. He was found to have comminuted fractures of the right limb – femur neck and tibial shaft. His vitals were maintained. Lungs, CVS and per abdomen examinations were unrevealing. SpO_2 was normal. The detailed neurologic examination was within normal limits. The right lower limb was fixed by external fixation with restrained movements. After 24 hrs, the patient's consciousness deteriorated, X-ray chest done was normal, SpO_2 was 88%. Patient was transferred to medical ICU, brought in a semi-comatose condition, and was responding to questions with delay. Pupillary and corneal reflexes were normal. Systemic examination, including neurologic, were normal. X-ray chest and HRCT done were within normal limits. Blood gas analysis reports were within normal limits. MRI brain showed long T1 T2 signals with diffuse punctuates and high signal intensity as diffusion weighted imaging DW1, throughout bilateral cerebral cortex, white matter, basal ganglion and thalamus (Fig. 1). Patient was provided with symptomatic and supportive treatment with nasal oxygen, fluids and methylprednisolone 500 mg 8 hrly x 5 days, LMWH in dose of 0.6 cc BD x 5 days. Repeat counts and blood gas analysis were normal on day 5 of hospitalisation and subsequently within normal limits. Repeat MRI after 2 weeks revealed significant regressions of diffuse punctuate and high signaled intensities on DW1. Patient was discharged after 2 weeks of hospitalisation with a diagnosis of cerebral fat embolism and long bone fractures with normal vitals and systemic examination with advice to seek an orthopaedic consultation.

Discussion

Almost all cases of fat embolism syndrome (FES) are due to fractures of long bones and/or pelvic bones. However, certain cases are non-orthopaedic and/or non-trauma

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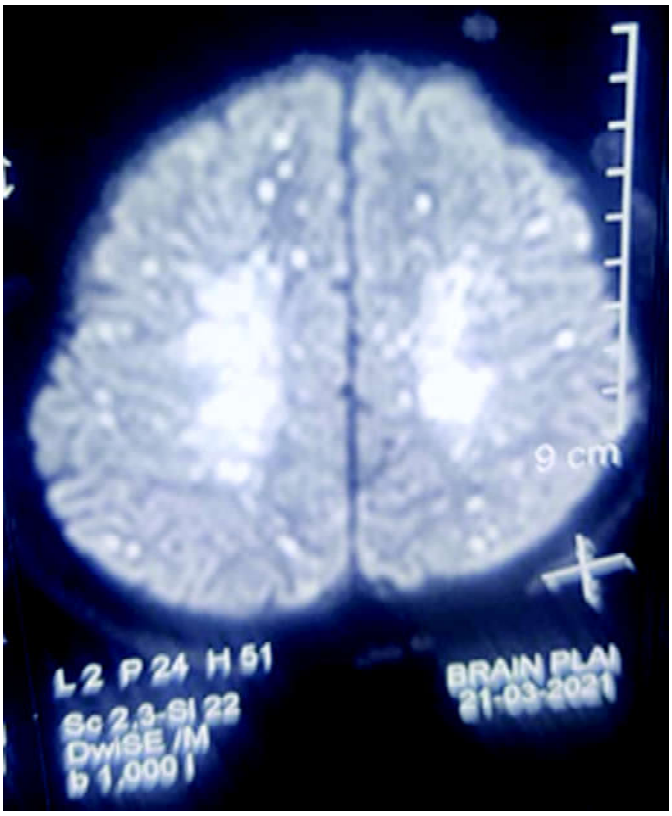

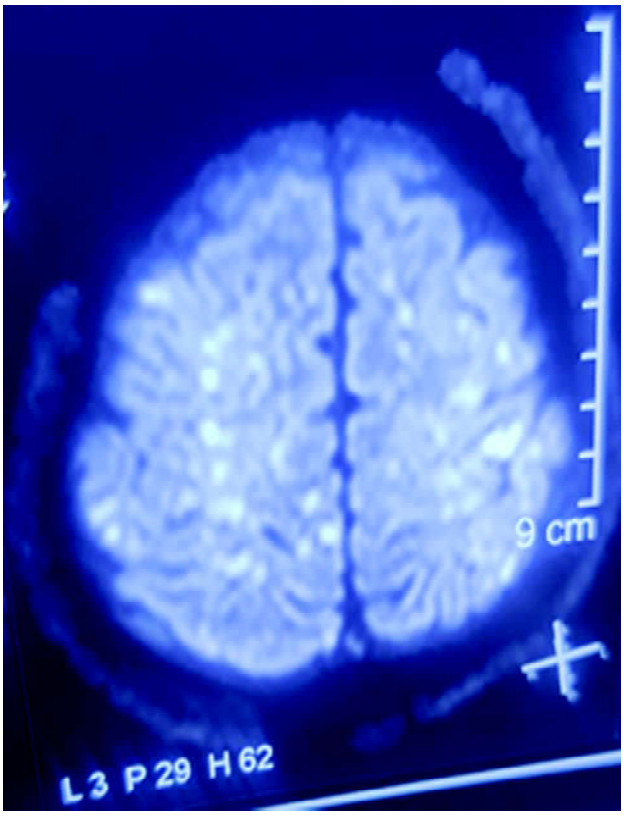

Fig. 1: *Shows T1 and T2 signals with diffuse punctuates and high signal intensities on DW1 throughout bilateral cerebral cortex, white matter, basal ganglion and thalamus.*

related. Rib fractures also have been responsible for FES. Surgical trauma occurring during surgical procedures such as hip/knee arthoplasty where excess bone marrow needs to be handled, interosseous access, infusion of lipid-based material, contrast agents, intramuscular injections of oil for cosmetic purposes, fatty liver disease are relatively uncommon causes of this syndrome[3]. In one autopsy data, in non-traumatic population, 63% cases had FES, representing the entity to be subclinical[4].

The exact pathogenesis is unknown. The present case had inflicted roadside trauma with long bone fractures of right lower extremity. He was conscious for 24 hrs post-injury and developed consciousness disturbances 30 hrs post-injury. Patient respiration was stable without obvious dyspnoea and pulmonary manifestations. SpO_2 was normal initially on admission but dropped to 88% after 24 hrs. Patient's X-ray chest and HRCT were normal. This patient's history was denied for any lucid interval, focal lesions. The MRI showed diffuse abnormal signals in the bilateral cerebral hemispheres, long T1 T2 signals with diffuse punctuates and high signal intensity on DW1 throughout bilateral cerebral cortex, white matter, basal ganglion, and thalamus. A possibility of cerebral fat embolism was strongly entertained. Though fat embolism often occurs in the lungs, an isolated brain fat embolism is however rare and its pathogenesis is not fully understood. Though SpO_2 done was 88% for a short period, subsequent X-ray chest and HRCT were within normal limits. Biochemical-induced hypoxaemia or shunt development was postulated as a probable mechanism for cerebral manifestations in the case. Echo was also done. It did not reveal evidence of patent foramina ovale or any cardiac shunt. Though in normal, PFO is open in 35% healthy individuals.

In cerebral fat embolism, normally the embolus from the venous system migrates to intracranial vessels only through pulmonary circulation. The ischaemia and hypoxaemia of pulmonary circulation manifests with spectrum of clinical features, but without the development of vital pulmonary features. Cerebral changes are noted in 86% of patients with FES. The present patient developed cerebral fat embolism bypassing the pulmonary manifestations and paradoxical embolisation. Two theories have been postulated under such circumstances:–

I. Microembolisation: which may occur when emboli are very small. Perhaps because of a few fat droplets with a small diameter < 7 - 20 µm which pass through the pulmonary capillaries into the systemic circulation

and lodge in the cerebral vessels[5]. This mechanism is supported by the findings of embolised material to systemic side in absence of cardiac shunts or PFO. The theory does not explain why the patient remains normal for a 24 - 48 hrs interval following acute insult of injury.

II. The other postulation is based on biochemical theory. The hypothesis is that the embolised fat degrades in two toxic intermediaries with pro-inflammatory effects and is supported by enhanced level of free fatty acids and cytokines including TNFα, IL-1, IL-6 and CRP. CRP is elevated in the present case suggestive of increased inflammation. The elevated level in the present case appears to be responsible for lipid agglutination obstructing blood flow in microcirculation.

The production of proinflammatory lipid mediators may explain the 24 - 48 hrs delay from the injury event to clinically apparent FES. This latent period explains the onset of symptoms that coincides with degradation and agglutination of fat and development of intermediaries. The clinical classic triad of hypoxaemia, neurologic abnormalities, and petechial rash are sometimes considered specific for FES. Rashes are red-brown, seen in 20 - 50% of cases, seen on non-dependent regions including head, neck, anterior chest, axilla and conjunctiva. Hypoxia sometimes may be part of ARDS syndrome, often noted in 96% of cases. ARDS develops in 50% of such individuals. Such cases may require assisted ventilation for severe hypoxaemia. None of these classic triad features, however, are specific for FES. Less commonly, patients of FES present with anaemia, thrombocytopenia, DIC, hypotension, shock[6].

The diagnostic evaluation requires assessment for severity of the disease of FES especially in the absence of pulmonary manifestations and bid for the need of supportive care in the case. Early intervention for the management of fracture may prevent development of FES. Though here, prompt supportive care remains the mainstay of therapy in clinically symptomatic brain FES. Management requires oxygen therapy, fluid resuscitation, steroids, low molecular weight heparin and NIV or invasive mechanical ventilation whenever applicable.

Use of corticosteroids, though controversial, but rationale for the use is based upon its anti-inflammatory effect targeting stabilisation of cell microsomal membranes, reduction of inflammatory response caused by FFA, capillary permeability and reducing tissue oedema. Methylprednisolone provided as 500 - 1,000 mg/day x 3 - 5 days or dexamethasone 20 - 30 mg/day or even 60 mg/day until brain oedema subsides.

5% alcohol glucose solution can inhibit formation of fat droplets. Low molecular weight heparin can reduce blood viscosity and reduce stress-induced chemical bio-metabolites and improve microcirculation. Low molecular weight dextran also is used in clinical practice[7].

Effectiveness of early hyperbaric oxygen therapy for cerebral fat embolism has been found useful as it enhances oxygen content, pressure as well diffusion in capillaries of brain micro-circulation[5]. In isolated cerebral fat embolism, chest distress, dyspnoea and pulmonary manifestations are strikingly lacking leading to misdiagnosis or delay in its diagnosis. Early recognition of features are warranted.

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