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DIFFUSE ALVEOLAR HAEMORRHAGE, A RARE PRESENTATION OF FAT EMBOLISM SYNDROME

Anaesthesiology

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ABSTRACT

Fat embolism syndrome (FES) is a rare clinical condition in which circulating fat emboli lead to multisystem dysfunction. It is commonly associated with long bone fractures and their surgeries. The clinical manifestations are varied, most commonly involving the respiratory system, central nervous system and skin. However, respiratory failure accompanying diffuse alveolar haemorrhage (DAH) in FES is rare. This case report describes a rare case of FES presenting as DAH in a 22-year-old male patient who suffered a fracture of femur and tibia after a road traffic accident.

KEYWORDS

Fat embolism, Fat embolism syndrome, Diffuse alveolar haemorrhage

INTRODUCTION

Fat embolism is a rare clinical condition, commonly associated with long bone fractures. The diagnosis of Fat embolism syndrome (FES) is made on clinical ground, as it may present with an atypical symptom such as diffuse alveolar haemorrhage (DAH).

CASE REPORT

A 22-year-old male healthy patient presented to orthopaedic emergency department of our hospital following a road traffic accident. There was no history of loss of consciousness, nausea, vomiting, seizures, or ear bleeding. The patient was hemodynamically stable. Physical examination revealed swelling and deformity of the right thigh and leg with no open wound. There was no distal neurovascular deficit. X-ray scan of right thigh and leg revealed closed sub trochanteric fracture femur, fracture both bone leg and medial malleolus. Chest X-ray was unremarkable. The patient was awaiting surgical management of his fractures.

Seventy hours after trauma, the patient developed dyspnoea with an oxygen saturation between 86-88 % on room air and tachycardia with a heart rate above 130 beats per minute. Twice, he coughed out blood, 15 ml per episode. On auscultation, crepitations were present in bilateral lung fields. He remained afebrile with no skin rash or petechia on the buccal mucosa. He was conscious with a Glasgow coma scale of 15. Suspecting pulmonary embolism, oxygen therapy was instituted using a face mask and a subcutaneous injection of low molecular weight heparin was immediately given. He was shifted to the intensive care unit of our hospital. Arterial blood gas analysis showed a PaO2/FiO2 ratio of 180 and normal lactate levels. Bed Side echocardiography showed an ejection fraction of 65 % with the normal-sized right ventricle. ECG was also normal. A computed tomography pulmonary angiogram was performed which ruled out pulmonary embolism. But it showed crazy paving patterns in bilateral lung fields with multiple consolidations, interlobular septal thickening, and ground glass opacities with pulmonary nodules, suggestive of DAH. (Fig 1)

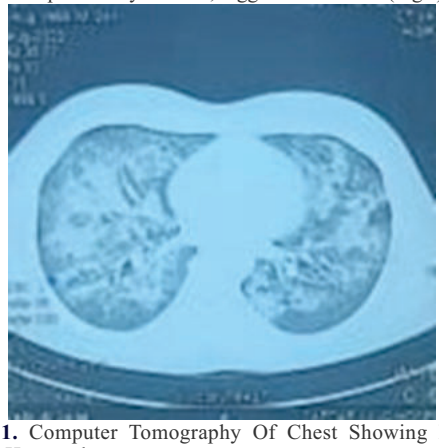

Figure 1. Computer Tomography Of Chest Showing Bilateral Alveolar Haemorrhage

Laboratory tests showed an acute drop in haemoglobin (from 11 mg/dl to 8.6 mg/dl)The platelet count was normal, D- dimer (1323 ng/ml against normal range of < 500 ng/ml), and ESR was raised (32 mm/hr against normal range of 0-20 mm/hr). Urine examination for fat globules was positive, though fundoscopy did not reveal fat globules in the retina. X-ray showed diffuse alveolar opacities. (Fig 2) The patient underwent flexible bronchoscopy with the bronchoalveolar lavage (BAL) which showed numerous debris-laden alveolar macrophages. BAL's complete microbiological workup was negative.

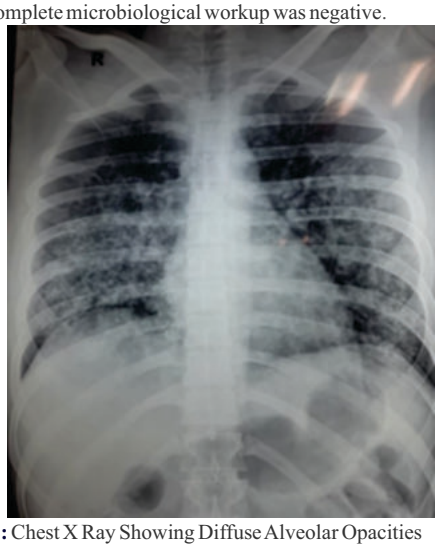

Figure 2: Chest X Ray Showing Diffuse Alveolar Opacities

Other investigations like COVID-19, sputum AFB, and gene expert for mycobacterium tuberculosis, Anti - nuclear antibody, and anti-neutrophilic cytoplasmic antibody were all negative.

The patient was haemodynamically stable but, continued to have 4-5 episodes of haemoptysis per day (10-15 ml per episode) and required 30 % oxygen via nasal prongs for maintaining 94% oxygen saturation. Supportive care using supplemental oxygen was continued and gradually tapered off in the next 5 days. His daily serial haemoglobin monitoring did not show any further fall. LMWH was never repeated. He was kept under medical surveillance until symptoms disappeared. He was discharged on the 5th day of an episode from ICU. He was operated on a day later and was discharged on 3rd postoperative day.

DISCUSSION:

FES is a rare clinical condition in which circulating fat emboli lead to multisystem dysfunction. The actual incidence of FES is very high but literature ranges its incidence from < 1 to 29% as mild cases go unnoticed.[1] FES is most commonly associated with high-velocity orthopaedic trauma involving long bones.[2] The risk factors for the development of FES are young age (10-40 years), male, closed fractures, multiple fractures, and conservative therapy for long-bone fractures.[3] Rarely, atraumatic causes such as bone marrow

transplantation, pancreatitis, sickle cell disease, liposuction, decompression sickness, lipid infusions, etc can also cause FES. [3] Our patient was young male suffering from a closed fracture of the femur and tibia due to a road traffic accident and was waiting for surgical fixation of his fractures.

Though the exact mechanism of FES is still not known, two leading theories for the formation of fat embolism are mechanical obstruction and biochemical injury.

Mechanical theory suggests that following orthopaedic trauma or during the surgical intervention of long bones, intramedullary pressure increases, leading to the direct release of bone marrow or fat globules into the venous system through open venous sinusoids. These fat globules get mechanically trapped in the pulmonary capillary bed leading to an acute rise in pulmonary arterial pressure. If emboli are massive, it can lead to circulatory collapse and death. Smaller size fat droplets can pass through pulmonary capillaries to systemic circulation or via patent foramen ovale in the heart.[4]

The biochemical theory suggests that regardless of the mechanism of initiating fat embolism, it leads to an intense inflammatory response. Fat globules in circulation get broken down by tissue lipases, resulting in high concentrations of glycerol and toxic free fatty acids. The elevated free fatty acids and other pro-inflammatory cytokines damage the endothelial lining in the lung, brain, skin, and other organs. In the lungs, damage to alveolocapillary endothelium produces interstitial edema and haemorrhage leading to acute lung injury, acute respiratory distress syndrome, or diffuse alveolar haemorrhage (DAH).[4,5] This also explains FES in nontraumatic settings as well as the delay of symptoms from hours to days from the traumatic insult as time is required for build up these toxic metabolites.

The clinical manifestations usually develop 24–72 h after trauma. The respiratory system is the one most commonly involved (95%), followed by the central nervous system and skin.[5] Pulmonary manifestations include dyspnoea, tachypnoea, hypoxemia, and respiratory failure. Neurological abnormalities include confusion, lethargy, restlessness, focal deficits, and coma. The petechial rash is classically located in nondependent regions like conjunctivae, head, neck, anterior thorax, or axillae.[6] Apart from this classical triad, FES can account for multiple clinical presentations. In absence of any specific clinical feature or gold standard test, different authors have proposed clinical diagnostic criteria, the most common being Gurd′s criteria.[7] (Major Criteria include petechial rash, respiratory insufficiency, and cerebral involvement in non-head injury patients. Minor criteria include fever greater than 38.5 C, heart rate greater than 110 beats per minute, retinal involvement, renal signs, anaemia, thrombocytopenia, high ESR, and fat globules in sputum) Diagnosis of FES requires 1 major and 4 minor criteria. Severe cases of FES can be complicated by disseminated intravascular coagulation, right ventricular failure, biventricular failure, acute respiratory distress syndrome, shock, and death. DAH has been reported as a rare manifestation of fat embolism.[8,9,10] It presents as haemoptysis, bilateral diffuse alveolar opacities on Chest X-ray, haemoglobin fall, and evidence of haemorrhagic BAL. Our patient presented with all the features of DAH.

Considering, the age of our patient, precipitating factors and clinical syndrome [respiratory distress,(Major criterion) tachycardia, fall of haemoglobin, high ESR, fat globules in urine, (Minor criterion) diffuse alveolar opacities on Chest x-ray and HRCT findings], diagnosis of fat embolism presenting as diffuse alveolar haemorrhage seems appropriate.

There is no specific treatment for FES. Treatment of FES consists of ensuring good arterial oxygenation and maintenance of intravascular volume. Systemic anticoagulation by LMWH is not recommended as it stimulates lipase activity, which will increase free fatty acids and can exacerbate the underlying proinflammatory physiology. Furthermore, anticoagulation in the setting of trauma and pre-existing hematologic abnormalities may prove harmful.[3] Corticosteroid therapy has been proposed as a potential therapy as it can limit free fatty acid levels, stabilizes membranes, and inhibit complement-mediated leukocyte aggregation. But studies have refuted this claim.[11] Some clinicians still prefer to give steroids and methylprednisolone in the dose range from 6 to 90 mg/kg. We had not given heparin or steroid to our patient.

FES is well-recognized morbidity of orthopaedic trauma and its surgical intervention. It may rarely present as DAH for which clinical suspicion is important and management is supportive. Prognosis is usually good with complete resolution of all symptoms. Early operative fixation of long-bone fractures has reduced the incidence of FES.

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