Fat embolism syndrome: Case report of a clinical conundrum

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Abstract

Fat embolism syndrome is a rare clinical condition associated with trauma, particularly of long bones. FES after fracture of neck of femur or head of humerus is uncommon. We report a case of FES following fracture in neck of femur and head of humerus in a man with history of mitral valve replacement, on long-term oral anticoagulant therapy, with an alleged history of convulsions. Our dilemma in clinical diagnosis is discussed.

Key words: Clinical criteria, fat embolism syndrome, head of humerus fracture, neck of femur fracture

Introduction

Fat embolism syndrome is a unique clinical condition in which circulating fat droplets lead to multisystem dysfunction. Diagnosis often follows a process of elimination of other causes for organ dysfunction. Absence of long bone fracture in our patient, along with presence of a cardiac condition, chronic oral anticoagulant therapy, and alleged history of convulsions prior to fall, lessened clinical suspicion for FES. Development of FES after fractured femoral neck and humeral head is extremely rare. Such a clinical situation has been previously rarely reported.

Case Report

A 38-year-old man fell from his bed, following sudden convulsive movements, and sustained injury to his right shoulder and right hip. He had undergone mitral valve replacement 11 years ago for rheumatic heart disease with mitral valve regurgitation, thus was on penicillin prophylaxis

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and oral anticoagulant (acenocoumarol 2 mg/day). In a local hospital, he developed shortness of breath and giddiness, and was referred to our hospital. In the casualty, 13 h after his fall, the patient was febrile (38.3°C) with a pulse rate of 128 beats min-1 (bpm), and was breathing at 32 bpm. SpO₂ was 93% on room air. On auscultation, lungs were clear and he was hemodynamically stable. There was no loss of consciousness. Radiographs confirmed comminuted fractures of the head of right humerus and neck of right femur. Computed tomography (CT) brain was normal and two-dimensional (2D) echocardiography revealed a wellfunctioning prosthetic mitral valve. The patient was shifted to critical care unit (CCU) for further management, where petechiae were noticed in the axillae and upper trunk. Initially, on bilevel positive airway pressure, patient was intubated on the 2nd day in CCU (about 40 h after trauma) and ventilated in view of increasing respiratory insufficiency and deterioration of sensorium. Crepitations on auscultation, frothy secretion in the endotracheal tube, and serial chest X-rays [Figure 1] showing progressive bilateral infiltrations suggested pulmonary edema.

Oxygenation improved with pressure controlled ventilation with assist control. Supportive therapy included diuretics, antiepileptics, corticosteroids, antibiotics, nutrition, hydration, and calcium supplementation. Ventilatory support was weaned over next 6 days and trachea was extubated. During this period, PaO₂/FiO₂ ratio improved from 85 to 436. The patient was shifted from the CCU to ward on the 7th day. He subsequently underwent open reduction and internal fixation for fractured head of right humerus and bipolar hemiarthroplasty for fractured neck of right femur. Operative procedures and recovery were

uneventful. He was discharged from hospital on the 4th postoperative day.

Discussion

Although the first clinical description of FES dates back to 1873,[1] its diagnosis remains a challenge for clinicians. Asymptomatic embolism of fat can occur with all long bone fractures after intramedullary nailing. After an asymptomatic period of 12-72 h, few patients develop a triad of lung, brain, and skin involvement known as FES.[2] Features of multisystem dysfunction due to either (1) mechanical obstruction of capillaries by fat emboli, or (2) production of toxic free fatty acids by hydrolysis of fat occurs predominantly (95%) after major trauma. Atypical presentations of FES have been reported. [3] Only 1-3% of patients with a single long bone fracture may develop the syndrome, while it may be seen in up to a third of patients with bilateral femoral fractures. [4,5] FES following fracture of femoral neck or humeral head, as is described in our case report, is highly uncommon. Incidence of 0.06% of FES following unilateral fracture of neck of femur was reported, while the incidence was too low to calculate with isolated fractures of the pelvis, ribs, humerus, radius, or ulna. [6]

Our differential diagnoses were heart failure, pulmonary thrombembolism, neurogenic pulmonary edema, alveolar hemorrhage, and FES. Our patient had undergone mitral valve replacement and was on long-term anticoagulant therapy. Our initial efforts were biased toward evaluating the patient's cardiac status. 2D echo helped to rule out complications of the preexisting heart condition. Alleged history of convulsions prompted thorough neurological investigation, including electroencephalography, but could not highlight a cause of the alleged convulsions prior to fall. Intracranial bleed or alveolar hemorrhage, as a complication of chronic anticoagulant therapy, was considered.

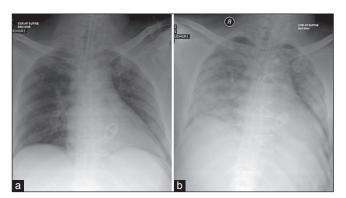


Figure 1: Chest X-ray on (a) admission and (b) 2^{nd} day in critical care unit (CCU; bilateral infiltrations); prosthetic mitral valve (arrow)

Gurd and Wilson^[7] published the most widely accepted guideline for the diagnosis of FES in 1974. It has been adapted many times since then. Modified Gurd and Wilson's criteria^[2,8] requires at least one major and at least four minor signs for diagnosis [Table 1]. Schonfeld *et al.*,^[9] incorporated assessment of oxygenation with ABG in their scoring system [Table 2]. A cumulative score >5 is required for diagnosis. These criteria, however, are not the gold standard for diagnosis.

Reddish-brown nonpalpable petechiae were noticed in bilateral axillae and upper trunk of the patient. These petechiae, due to embolization of small dermal capillaries leading to extravasation of erythrocytes, occur in only 20-50% of patients and resolve quickly. They are virtually diagnostic in the right clinical setting. [10]

As per modified Gurd and Wilson's criteria [Table 1], three major (petechiae, respiratory insufficiency, and cerebral involvement) and four minor criteria (tachycardia, fever, >20% fall in hemoglobin (Hb) level from 12.2 to 9.2 g dl⁻¹, and increase in erythrocyte sedimentation rate (ESR) from 21 to 74 mm h⁻¹) were satisfied in our patient. All criteria in Schonfeld's scoring system^[9] [Table 2] were fulfilled. A diagnosis of FES was made.

Table 1: Modified Gurd and Wilson's criteria

Major criteria

Respiratory insufficiency (hypoxemia ${\rm PaO_2} < 60~{\rm mmHg}, {\rm FiO_2} = 0.4$) Central nervous system (CNS) depression disproportionate to hypoxemia

Pulmonary edema

Axillary or subconjunctival petechiae

Minor criteria

Tachycardia >110 min⁻¹

Pyrexia >38.5°C

Emboli present in the retina on fundoscopy

Fat globules present in urine

Jaundice

Oligoanuria

Sudden drop in hemoglobin (Hb) >20%

Sudden thrombocytopenia >50%

Increasing erythrocyte sedimentation rate (ESR) >71 mm h⁻¹

Fat globules present in the sputum

Fat macroglobinema

Tachypnea (>30 bpm)

Table 2: Schonfeld's criteria		
Clinical sign	Score	
Petechiae	5	
Chest X-ray changes (diffuse alveolar infiltrates)	4	
Hypoxemia (PaO ₂ <70 mmHg)	3	
Fever (>38°C)	1	
Tachycardia (>120 bpm)	1	

Prevention of FES includes early stabilization of long bone fractures and prophylactic corticosteroids. Treatment of FES is mainly supportive. Use of corticosteroids and heparin remains controversial. Possible beneficial effects of corticosteroids include stabilization of the pulmonary capillary membrane, thus reducing interstitial edema, blunting the inflammatory response, stabilizing complement system activation, and retarding platelet aggregation. [2] Heparin is known to clear lipemic serum by stimulating lipase activity. [2]

Without specific tests and validated clinical criteria, diagnosis of FES is challenging. One should uphold a high degree of suspicion in spite of lesser fractures not usually associated with FES. Although most patients recover fully, a mortality of 5-15% has been reported. [2,4] Early diagnosis and treatment of symptoms is of paramount importance for a successful outcome.

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