

ABSTRACT:

Fat embolism syndrome (FES) is a life-threatening complication in patients with trauma, especially in long bone fractures. The diagnosis of fat embolism is mainly clinical. The significance of the FES is that it has no specific laboratory findings nor specific treatment except supportive care, although it can be prevented by early fixation of the fractured bone.

Here we report a case which occurred immediately after the surgical fixation is done. We report a case of twenty days old fracture neck of femur which was diagnosed as FES on the basis of clinical findings as per GURD and WILSON criteria. The patient received supportive management and a short course of intravenous methylprednisolone.

INTRODUCTION:**FAT EMBOLISM SYNDROME :**

The incidence of fat embolism can approach up to 90% in patients with sustained injuries. If it affects, it can affect multiple organ system with its systemic inflammatory cascade resulting in high morbidities and mortalities. So early diagnosis and management is important. The diagnosis of FES is mainly clinical, depends upon clinical signs like dyspnoea, petechia, cognitive dysfunction in the first few days of trauma specially long bone fracture or intramedullary surgery. FES occurs 2-5% of patients who have long bone fracture. Studies says if diagnosed early and treated promptly, the mortality may go down to 10%. GURD and WILSON CRITERIA mentioned below is one of the important tool to diagnose FES.

GURD and WILSON CRITERIA(published in 1974):**Major criteria:**

1. Symptoms and radiological evidence of respiratory insufficiency(hypoxemia $\text{PaO}_2 < 60, \text{FiO}_2 = 0.4$)
2. Cerebral sequelae unrelated to head injury or CNS depression disproportionate to the level of hypoxia.
3. Petechial rashes

Minor criteria:

1. Tachycardia(>110/min)
2. Pyrexia(>38.5 degree C)
3. Retinal emboli on fundoscopy
4. Renal dysfunction(oligoanuria)
5. Jaundice
6. Acute drop in haemoglobin >20%
7. Sudden thrombocytopenia >50%
8. Elevated ESR(>70 mm 1 st hr)
9. Fat macroglobulinemia,fat globules in sputum.

Therefore,It was thought to be reported because it's a very rare entity and also because of its difficulty in diagnosis as well as if at all diagnosed early can lead to prevention of lot of complications and reduction in mortality.

CASE REPORT:

A patient 72 year male,a chronic ethanolic was admitted with the alleged history of fall followed by right trochanteric fracture .Initially after the fall,he was at home but later after 20 days ,he went to a local hospital where he was operated for the fracture.On the day of operation,he developed altered sensorium immediately after the surgery.He was shifted to an another local hospital with ICU facilities where he was transfused with 16 units of FFP and 2 Units of PRBCs for his deranged PT/INR and Low Hemoglobin level.Later when the condition of the patient further deteriorated,he was shifted to our tertiary care hospital for further management of the case.

On admission to our hospital,the patient was in altered sensorium,drowsiness(E2V2M5) and respiratory distress.Initially it was presumed to be a case of hepatic encephalopathy with ARDS,TRALI with AKI and deranged LFT. He was immediately put on invasive ventilation and shifted to Intensive Care Unit.

CXR ,ABG were done suggestive of ARDS.Bronchoscopy was done and lavage was taken and sent for analysis.Trop I was normal(0.098) with normal ECG and normal ECHO.Gradually the patient went into shock and inotropes were started.His platelet count came down to 75000,LDH was 570 and direct coombs test was positive.There was oozing from multiple iv puncture sites and petechial rashes spread all over his body.his urine output gradually decreased.Bronchoscopy lavage showed

pseudomonas and wound culture from his Bedsore showed acinetobacter growth(MDR).Antibiotics were revised as per hospital antibiogram .

Since there is neurological impairment,respiratory insufficiency with rashes all over the body along with acute drop in platelets count with haemoglobin and there was fat globules in urine ,it was thought to be a case of FAT EMBOLISM SYNDROME(FES) as per GURD and WILSON CRITERIA.

Injectable methylprednisolone was started with iv albumin.there was growth of yeast cells in the urine,so inj Anidulafungin was also started.Gradually the TC count of the patient came down,FiO2 decreased to 40%,inotropes support reduced and the patient was extubated on eighth day.His D- dimer and FDP came positive.Later the patient significantly improved and was shifted to the ward.

INVESTIGATIONS:

Hb:5.9,TLC:32.9,Plat:75000,PT(T):19.5,PT©:13.2,INR:1.56,Na:149,K:3.09,Creat:1.4,B UN:65,Bil:1.37, LDH:570, Albumin:2.5,Fibrinogen:Low,D-dimer positive,stool for occult blood positive,

UGIE:pangastritis,USG whole abdomen:liver parenchymal changes with bright kidneys,ascites.Urine for fat globulin positive,CT brain:normal except age related atrophy.Coomb,s test positive(direct).ECG:normal sinus rhythm,ECHO:normal,Tropl:Normal(0.098),CXR:B/L diffuse infiltrates.

Bronchoscopy lavage(BAL)positive for pseudomonas,Bedsore swab culture positive for acinetobacter.Urine positive for budding yeast cells(heavy).

DISCUSSION:

FES is a rare but potentially life threatening complication.A high level of suspicion should be taken when patients present with hypoxia,confusion or rash within 24-72 hrs following long bone fractures and/or postoperatively. Features of multisystem dysfunction occurs due to either(1)mechanical obstruction of capillaries by fat emboli or (2)production of toxic free fatty acids by hydrolysis of fat after trauma.

Close differential diagnosis include pulmonary embolism,acute respiratory distress syndrome,pulmonary edema or atypical infections.

The mainstay of treatment is always supportive; ensuring good arterial oxygenation, maintaining adequate intravascular volume. Prevention of FES includes early stabilisation of long bone fractures and prophylactic corticosteroids. Possible beneficial effects of steroids include stabilisation of the pulmonary capillary membrane, thus reducing interstitial edema, blunting the inflammatory response, stabilising complement activating system, and reducing platelet aggregation. Heparin is known to clear lipemic serum by stimulating lipase activity.

Without specific tests and validated criteria, diagnosis of FES is challenging. Although many patients recover fully if treated timely. Early diagnosis and treatment is of paramount importance for a successful outcome.

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