



## "DOES NEUROLOGICAL MANIFESTATIONS IN A PATIENT DIAGNOSED WITH FAT EMBOLISM SYNDROME AN INDICATION TO RULE OUT CARDIAC ABNORMALITIES ? - A CASE REPORT".

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### ABSTRACT

Fat embolism occurs when bony or soft tissue trauma has caused fat to enter the circulation, or in atraumatic disorders where circulating fat particles have coalesced abnormally within the circulation. The fat particles deposit in the pulmonary and systemic circulations, although only 1 - 2% develop a clinical disorder with respiratory, cerebral and dermal manifestations known as the fat embolism syndrome<sup>1</sup>. Management of FES requires early immobilization of fracture, symptomatic management of respiratory, cardiovascular, and neurological abnormalities<sup>2</sup>. Here we discuss about a 32 year old male with marfanoid like features who came with alleged history of road traffic accident, diagnosed with both bone fracture right leg and comminuted inter trochanteric fracture left hip. Patient underwent PFNA2 fixation of left hip and post operatively developed decreased response, desaturation and low urine output. This case discusses the increased chances of neurological symptoms in those having congenital heart abnormalities like a patent foramen ovale and the need to do a further cardiac evaluation including imaging like trans esophageal echocardiography.

**KEYWORDS :** FES, patent foramen ovale, trans-esophageal echo

### CASE REPORT

A 32 year old male with alleged history of road traffic accident was diagnosed with both bone fracture right leg and comminuted inter trochanteric fracture of the left hip. Underwent proximal femoral nailing and fixation left hip under lumbar subarachnoid block. Post operatively he developed desaturation, decreased response and low urine output.

Patient was stuporous. Patient was febrile. Patient had tachycardia and was tachypnoeic. GCS was E2V1M3 and pupils were bilateral 3mm with sluggish response. Bilateral air entry was present with no added sounds on chest auscultation. Patient was anaemic with oliguria. ECG showed sinus tachycardia with RBBB, right axis deviation and S1Q3T3 pattern. Bedside echo showed features of right ventricular strain, severe PAH and cardiomegaly. Ultrasound abdomen did not show any evidence of bleed or collections. Fundoscopy showed macular area showing, retinal edema with cherry red spot which is suggestive of bilateral retinal artery occlusion. Patient had no previous history of any heart disease, cerebral vascular accident or renal diseases. Patient had congenital mutilated fingers with marfanoid features like arachnodactyly, high arched palate and long limbs. Similar history present in family members. Lab Investigations were Haemoglobin - 7.8 g% , Total leucocyte count - 7,900 cells/c mm ,Platelet - 1,52,000 lakhs/Cumm, Urea - 26 mg/dl, Creatinine - 0.7 mg/dl, Sodium - 137 mmol/L, Potassium - 3.8 mmol/L, D Dimer - 5359 ng/ml, Trop I - 37.2 ng/ml Peripheral smear showed dimorphic anaemia.

CT brain was found to be within normal limits. CT Pulmonary Angiogram was taken in view of suspected pulmonary embolism but reports showed no evidence of acute or chronic pulmonary thromboembolism. CT Pulmonary Angiogram showed patchy consolidation in the postero basal segments and ground glass opacities in lateral basal segments of bilateral lower lobes suggestive of Aspiration Pneumonia. MRI Brain was done which showed features of white matter changes in fronto-parieto-temporo-occipital areas suggestive of Cerebral Fat Embolism. A provisional diagnosis of fat embolism syndrome was made using Gurd's criteria and MRI brain findings.

Patient was electively intubated and Invasive lines were placed in the emergency department . Patient underwent an emergency closed reduction and internal fixation with tibia

nailing under general anaesthesia. Patient was initiated on anti-coagulation, IV antibiotics and fluid resuscitation. Patient's neurological status gradually improved and was extubated. Patient was haemodynamically stable and subsequently discharged. On follow up 2 D ECHO was within normal limit. Due to his strange presentation of predominant neurological manifestations it was decided to further investigate his heart. Trans-esophageal echo showed a patent foramen ovale (Figure-1), good biventricular function and no evidence of VSD.

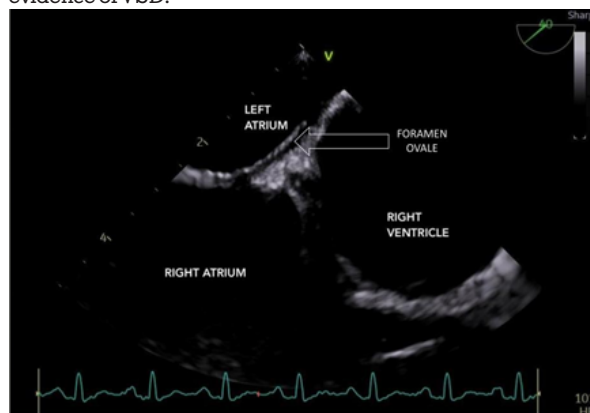


Figure 1 - Trans esophageal echocardiography image showing foramen ovale

### DISCUSSION

Generally, CT scan brain is normal in patients with isolated fat embolism. Magnetic resonance imaging is more sensitive and demonstrates multiple small hyperintense, intracerebral lesions<sup>2</sup>. In our patient CT Brain was within normal limits but MRI Brain showed features of Fat Embolism Syndrome (FES).

Neurological manifestations of FES initially manifests as agitated delirium but may progress to stupor, seizures, or coma.<sup>3</sup> In our case, the patient started having neurological features within 24 hours of trauma and immediately after the initial surgery. He became initially agitated, restless and then developed period of reduced level of consciousness. There was hypoxemia, but petechial rashes were absent. Neurological manifestations were more predominant than pulmonary symptoms.

A curious aspect of FES is the involvement of systemic embolisation without pulmonary features. One suggestion is that this can occur via a patent foramen ovale, which has a

prevalence of around 35% in the general population, and systemic embolisation via this route has been reported.<sup>4</sup> In our case the neurological symptoms were the most predominant symptoms. Our patient had marfanoid like features which made us suspect an underlying heart disease. The pre-operative 2D echocardiogram showed concentric LVH, moderate PAH and good biventricular function. It was subsequently noted via a trans esophageal echocardiogram that our patient had a patent foramen ovale which may be the reason why our patient developed neurological manifestations.

## CONCLUSION

This case shows that patients with a congenital heart disease have more chances of developing neurological manifestations rather than the more common pulmonary manifestations seen in a patient with Fat Embolism Syndrome. A patient who has FES with predominant neurological manifestations should get a trans-esophageal echo done in order to rule out any cardiac abnormalities. The role of MRI Brain is significant in FES as it cannot be picked up in CT Brain. The early identification of FES and fixation of the fracture plays an important role in the recovery of the patient.

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