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## Fat embolism Syndrome: Is it common in smokers

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### Abstract

**Introduction:** Diagnosis of Fat Embolism Syndrome (FES) depends mainly on clinical features and there are no diagnostic laboratory tests available. In spite of so many advancements in the management of long bone fractures, management of FES is mainly supportive. Patients who are prone to develop FES are to be carefully screened as a part of pre-operative work up in order to prevent the complications later. There are very few studies which establish the risk factors associated with fat embolism.

**Materials and methods:** 26 Orthopaedic trauma patients who were diagnosed with fat embolism syndrome according to Gurd and Wilson criteria and Schonfeld's FES index, and as per inclusion-exclusion criteria of the study were included. All demographic data, bone involved, type of fracture, timing and type of surgery was noted and data regarding their smoking status was taken into consideration.

**Observation:** Out of 26 patients of FES, 25 were male and 1 was female. Mean age was 27.1 years. 11 were fractures of Femur and 15 were fractures of Tibia, 4 patients had multiple fractures. There were 5 cases of open fractures. Among the 26 patients who developed FES, 80.7% of them were cigarette smokers and all of them were male patients.

**Conclusion:** It is our observation that among the patients who developed FES, 80.7% were cigarette smokers. Though this study does not establish a direct causal relationship between smoking and FES which requires further detailed analytical studies, there appears to be an association between FES and cigarette smoking. Hence, we conclude that smokers are prone to develop FES.

**Keywords:** Fat embolism syndrome, cigarette smoking, long bone fractures

### Introduction

Fat embolism can be defined as the presence of fat droplets within the peripheral and lung micro circulation with or without clinical sequelae. Fat embolism syndrome (FES) is a clinical syndrome that is characterized by clear signs and symptoms [1]. FES is a serious manifestation of fat embolism that involves dermatological, neurological and respiratory systems usually occurring 24 to 72 hours of injury. However, not all fat emboli progress to FES [2, 3]. Incidence of fat embolism syndrome in long bone fractures ranges from 0.9% to as high as 33% in various studies. Whereas fat embolism is seen in upto 82% of trauma cases [3-6].

There are several causes that can be attributed to fat embolism. Majority of the times it is trauma related [7]. The traumatic causes include long bone fractures, pelvic fractures, burns, Orthopaedics procedures and liposuction. Some of the non traumatic causes associated with fat embolism include pancreatitis, diabetes mellitus, alcoholic (fatty) liver disease and lipid infusion [8]. The diagnosis of FES can be done using Gurd and Wilson criteria [9] and Schonfeld [10] criteria.

Theories explaining the pathogenesis of fat embolism syndrome are mechanical theory proposed by Gauss [11] and the biochemical theory proposed by Lehman [12]. According to mechanical theory, an increase in intramedullary pressure either due to the injury itself or iatrogenic factors, causes the marrow and fat to enter the systemic circulation via open venous sinusoids. These particles causes obstruction of small pulmonary and possibly of systemic vessels due to embolization [11].

According to biochemical theory proposed by Gauss, the release of free fatty acids and other mediators exert their biochemical and toxic effects causing vasculitis, pneumonitis and local inflammatory reaction leading to injury to lung endothelium and pneumocytes.

All these ultimately leads to respiratory failure [12]. Smoking causes IHD, malignancies, respiratory illness, small and medium vessel disease [13]. Smoking is known to cause pro oxidant/antioxidant imbalance [14]. Biochemical changes due to cigarette smoking are increased inflammatory mediators, increased lipid peroxidation, leucocyte and platelet activation, increased prothrombotic factors leading to endothelial damage, thrombus formation vasomotor dysfunction [15]. There are several risk factors associated with fat embolism syndrome (Table 1) [8]

**Table 1:** Risk factors for fat embolism syndrome

Young age
Closed fractures
Multiple fractures
Conservative therapy for long-bone fractures
After intramedullary nailing
Prosthesis cementation
Associated chest injuries

The timing of definitive fixation has been a subject of debate for patients with polytrauma. It is recommended to categorize patients based on the physiological status into stable, unstable, borderline and in extremis at the time of presentation. Patients who are stable and are physiologically fit to undergo the procedure should be operated within 12 hours. However, once the patient becomes symptomatic, it is better to postpone intramedullary instrumentation. Hence ruling out FES should be part of preoperative workup [16].

Reaming the intramedullary canal is hypothesized to increase the incidence of fat embolism syndrome according to the mechanical theory. However the decision to ream or not has to be based on several other factors such as fracture pattern, concomitant injuries and physiological status [17, 18].

As there are no diagnostic lab tests available and as diagnosis mainly depends upon clinical features [8, 18, 19] there is a need to know about the predisposing/ risk factors which may lead to fat embolism syndrome. Knowledge about predisposing factors will help the treating team anticipate the prognosis and complications.

In spite of so many advancements in the management of long bone fractures and its complications, etiopathogenesis of FES, male preponderance, the reason for predominant pulmonary and neurological involvement and why all long bone fractures do not end up in FES are not clearly understood. FES still remains to be a clinical diagnosis based on certain criteria [19]. Management of FES is mainly supportive and included administration of oxygen, steroids and heparin [18, 19]

Patients who are prone to develop fat embolism are to be carefully screened as a part of pre-operative work up in order to prevent complications later on. There are very few studies which establish the risk factors associated with fat embolism. Cigarette smoking as a predisposing factor for development of FES has not been studied. Based on our clinical experience, we retrospectively collected the data of patients who developed FES and this study was done to see if cigarette smokers were prone to develop FES.

## Materials and Methods

This was a retrospective study of 10 years duration. All patients presenting to MVJ Medical College and Research Hospital with long bone fractures and who developed tachycardia, tachypnoea and drop in saturation, were included in the study. The smoking habits of all patients was documented. The patients were

considered in our study based on the following including/exclusion criteria.

The study group included patients who were aged between 15 to 60 years, acute long bone fractures, multiple fractures, open fractures, preoperative FES and postoperative FES. Patients who fulfilled the criteria according to Gurd and Wilson (Table 2) and Schonfeld's fat embolism index (Table 3) were included. Schonfeld's fat embolism index was also included as chest X rays was done for all our patients.

**Table 2:** Gurd and Wilson

Major criteria	Minor criteria
Respiratory insufficiency	Pyrexia
Cerebral involvement	Tachycardia
Petechial rash	Retinal changes
	Jaundice
	Renal changes (anuria or oliguria)
	Thrombocytopenia (a drop of >50% of the admission thrombocyte value)
	High erythrocyte sedimentation rate
	Fat macroglobulinemia

2major / 1 major + 3 minor criteria for diagnosis

**Table 3:** Schonfield FES index

SIGN	SCORE
Petechial rash	5
Diffuse alveolar infiltrate	4
Hypoxaemia PaO< 70 Hg, FiO2> 100	3
Confusion	1
Fever	1
Heart Rate	1
Respiratory rate	1

Score of 5 or more diagnostic of FES

Patients who had head injury, polytrauma, sepsis and spinal cord trauma were excluded from the study. Patients who had prior history of IHD, CVA, respiratory illness also were excluded from the study.

According to our hospital protocol all patients who got admitted, initial assessment and splinting was done. Detailed examination to assess other injuries was done. Pre operatively all routine investigations like X rays, blood investigations and pre anesthetic checkup were carried out. There were cases of early and delayed presentation. Only haemodynamically stable cases were operated within 24 hours. There were cases of delayed presentation and delayed treatment due to multiple reasons who were operated after 24 hours.

In all patients who developed FES/ suspected FES, investigations were done including chest X ray, ECG, fundoscopy, arterial blood gas analysis and other investigations as required to rule out previous lung disease or lung injury was done. The diagnosis of FES was made based on Gurd and Wilson and Schonfield criteria. CT of brain and chest was done based on suggestion from neurologist and intensivist. Immobilization and splintage of the fractures were checked and confirmed routinely. Medical, Ophthalmology and neurological consultations were obtained. Patients were monitored in the ICU. All patients were managed as per hospital protocol. All patients received oxygen to improve their saturation, IV fluid for hydration, steroids and heparin. Surgery was delayed in patients who had FES as per anesthetist advice. Patients were monitored for tachycardia, tachypnoea, urine output, oxygen saturation, distal neurovascular deficits compartment syndrome preoperatively and postoperatively. Standard protocol for

surgical fixation of fracture was followed either using intramedullary nail or external fixator.

All demographic data, bone involved, type of fracture, associated fractures, timing and type of surgery was noted. The data regarding their cigarette smoking status was taken into consideration.

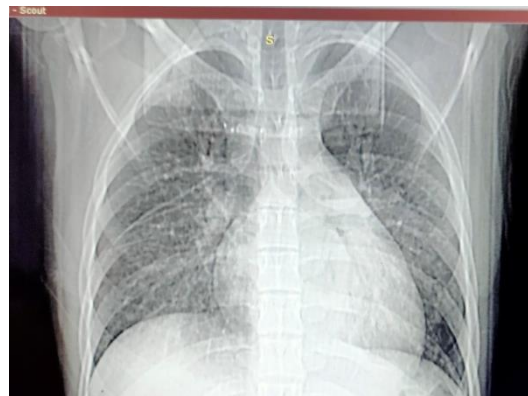
### Observation

Out of the 26 patients who were diagnosed to have fat embolism, 25(96.1%) were males and 1(3.9%) was female. Mean age was 27.1 (range 16-57) years. Of the long bone fractures 11 (42.3%) were femur and 15(57.7%) were tibia. There were 5 (19.2%) cases of open fractures out of which 2 were type 1, 1 was type 2 and 2 were type 3 open fractures

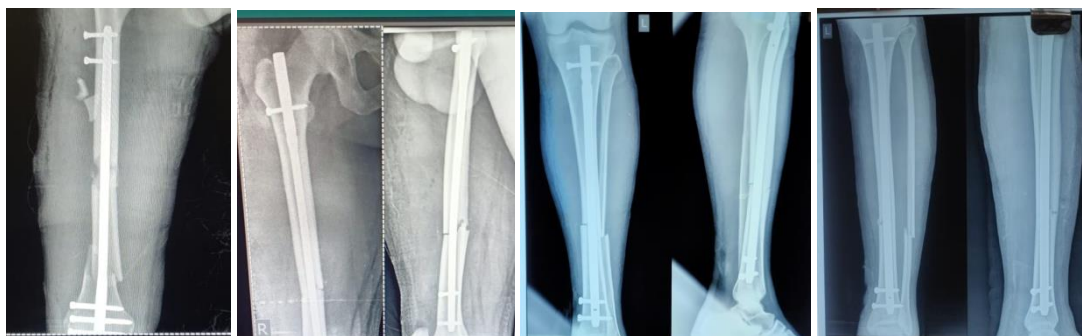
according to Gustilo Anderson classification. 22 (84.6%) patients developed FES preoperatively and 4 (15.4%) patients developed postoperative FES. All patients with femur fracture (11) (42.3%) were operated with intramedullary nailing. 3 (11.5%) patients with open tibia fractures were treated with external fixator. 4 (15.4%) patients had multiple fractures. Reaming was done in all the patients who underwent intramedullary nailing. 1 patient required ventilator support. 4 patients had to be referred outside due to the severity of disease upon the request of the patient party or physician. 1 patient was lost for follow up. Of the remaining 25 patients there was no mortality. Among the 26 patients who developed FES, 21(80.7%) of them were cigarette smokers and all of them were male.



Clinical photo and X ray of patient with femur fracture and FES



Chest X ray in patient FES



X rays of few patients who developed FES

### Discussion

Although pulmonary fat embolization occurs in almost all patients with long-bone fractures [1, 4, 6], only 1% to 30% of patients develop the full clinical fat embolism syndrome that

consists of petechial rash, diffuse pulmonary infiltrates, hypoxemia, confusion, pyrexia, tachycardia, and tachypnea, 24 to 48 hours after trauma [3, 5, 7].

Fat embolism syndrome can be due to trauma as well as non



trauma related causes.

FES genesis is a complex phenomenon and theories like Gauss mechanical theory and Lehmann biochemical theory have been proposed to explain its pathogenesis [8].

Diagnosis is made by using criteria like Gurd and Wilson, and Schonfield. There is no single laboratory test which is diagnostic of FES. Hence this requires early diagnosis and treatment, there is importance in knowing the risk factors [20].

Risk factors for fat embolism are male patient, young patient, closed fracture, intramedullary nailing and conservative management of long bone fractures [8, 21].

According to a study by Gopinathan *et al.* [20], the predictors of fat embolism were NISS score, serum lactate level at time of admission and hypoxaemia. However this study has included patients only with polytrauma. Though prevention is discussed by White *et al.* [18], there is more emphasis on identifying the risk factors as there is no definitive management. Risk factors include level of initial trauma, timing of the surgery, genetic predisposition and IL6 levels.

In our study, 25 were males and 1 was female as compared to a study done by Parvaiz *et al.* [22] who reported all male patients. The median age in their study was 28 years and in our study mean age was 27.1 years. Mortality in their study was 8%. There was no mortality in our study except for one patient who was referred to another centre and was lost for follow up. Whereas older literature has reported mortality of 3-16%, newer studies have shown decreasing mortality in fat embolism probably due to early diagnosis and treatment, and also early fixation of long bone fractures [23].

In our study, 71.8% of closed fractures and 19.2% of open fractures had developed FES compared to a study done by Thanigaimani *et al.* who reported 67% and 33% respectively [24]. In our study majority of the long bone fractures involved was tibia in contrast to other studies where it was predominantly femur. This was because of more number of total tibia cases compared to femur. The total number of tibia cases who did not develop FES was not taken into consideration as it is beyond the scope of this study. Among the patients who had multiple fractures, two patients sustained pelvic fractures associated with femur fracture. One was ilium and other was pubic ramus fracture. Another patient had a humerus fracture with tibia fracture and the other patient had patella fracture with femur fracture.

Among the 26 patients who developed FES, 21 (80.7%) were cigarette smokers. Though smoking is known to be associated with conditions like ischemic heart disease, malignancies, deep vein thrombosis and respiratory illness, no study was found showing association between fat embolism and smoking. The biochemical changes in smoking like lipid peroxidation, increased inflammatory mediators and increased free radicals causes exaggeration of inflammation. Whether these changes alter the pathophysiology of FES or exaggerate FES are to be studied further.

As FES is a diagnosis based on clinical criteria and complication occurring after 24 hours of injury, clinicians need to be aware of the patients who are at risk to develop FES. This would ensure early diagnosis and treatment.

### Conclusion

It is our observation that among the patients who developed FES, 80.7% were cigarette smokers. Though this study does not establish a direct causal relationship between smoking and FES which requires further detailed analytical studies, there appears to be an association between FES and smoking. Hence we

conclude that cigarette smokers are prone to develop FES.

### Limitation

Patients who were smokers but not developing FES were not included in the study.

Details of smoking and quantitative analysis was not done. As cigarette smoking tends to be more common in males, this may be a confounding factor in the study. Its only an observational study. More studies are required to prove a definitive association between smoking and FES with statistical analysis.

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