

# Fat Embolism Syndrome: A Case Series from a Single Tertiary Care Hospital

Bhaskar Borgohain<sup>1</sup>, A S Naveen<sup>1</sup>, Tashi G Khonglah<sup>1</sup>

## Learning Point of the Article:

Hypoxia and elevated serum IL-6 level appear to be the earliest detectable indicators of evolving FES, preceding the full clinical syndrome.

## Abstract

**Introduction:** Fat embolism syndrome (FES) is a rare but potentially fatal complication predominantly affecting young adults following long bone fractures. No definitive diagnostic test exists; diagnosis remains one of exclusion based on Gurd's criteria. Early recognition before the syndrome becomes fully established is critical to reduce preventable mortality.

**Materials and Methods:** We report a case series of six patients diagnosed with FES between 2020 and 2025, examining their clinical and laboratory parameters, including injury severity score, number of fractures, interleukin-6 (IL-6), C-reactive protein (CRP), hemoglobin (Hb), platelet count, and neurological status in relation to FES development. We charted the key parameters to depict the natural history of FES.

**Results:** All six patients were young adults (age 21–32 years) who sustained long bone fractures following road traffic accidents. All developed hypoxia and neurological deterioration (Glasgow coma scale drop) within days. Serum IL-6 and CRP were elevated in all patients for whom these were measured. All patients showed a fall in Hb and thrombocytopenia. Petechiae were present in 4/6 cases (67%). Two patients died (33%); both had high injury burden and markedly elevated IL-6.

**Conclusion:** Hypoxia and elevated IL-6 appear to be the earliest detectable indicators of evolving FES, preceding the full clinical syndrome. Acute anemia, thrombocytopenia, hypoalbuminemia, and oliguria were common. Only after the disease is clinically advanced Gurd's criteria were fulfilled. Rapid fracture stabilisation, continuous SpO<sub>2</sub> monitoring, serial IL-6 measurement, and universal team awareness are essential strategies for its potential prevention.

**Keywords:** Long bone fractures, pulse oximetry, fat embolism syndrome, Gurd's criteria, interleukin-6, early diagnosis.

## Introduction

Fat embolism syndrome (FES) is a rare but life-threatening systemic condition arising from the introduction of fat globules into the microcirculation, most often following long bone fractures or orthopedic surgery. First described by Zenker in 1863 and clinically characterised by Von Bergmann in 1873, FES has continued to challenge clinicians for over a century [1,2].

While fat embolism is the mere presence of fat particles in the systemic circulation and occurs in most long bone fractures, the full clinical syndrome of FES is substantially rarer (the reported incidence 0.9–11%), depending on the criteria applied and the study population [3,4].

FES has a particular predilection for young adults; typically, under 40 years of age after sustaining high-energy trauma, most

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## Author's Photo Gallery



Dr. Bhaskar Borgohain



Dr. A S Naveen



Dr. Tashi G Khonglah

<sup>1</sup>Department of Orthopaedics and Trauma, North Eastern Indira Gandhi Regional Institute of Health and Medical Sciences, Shillong, Meghalaya, India.

### Address of Correspondence:

Dr. Bhaskar Borgohain,  
Department of Orthopaedics and Trauma, North Eastern Indira Gandhi Regional Institute of Health and Medical Sciences, Shillong - 793018, Meghalaya, India.  
E-mail: bhaskarprofessor@gmail.com.

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**Table 1: Summary of the key variables of interest (number of fractures, ISS, IL-6, and CRP)**

Patient	Age/Sex	No. of Fractures	ISS (/75)	IL-6 (pg/mL)	CRP (mg/L)
1	25/M	3	41	155	85
2	23/F	1	34	NA	36
3	32/M	2	31	37	90
4	30/M	2	46	112	78
5	23/M	2	29	356.6	NA
6	21/M	4	36	117	5.9–9.27

**ISS: Injury severity score, IL-6: Interleukin-6, CRP: C-reactive protein, NA: Not available**

often from motor vehicle collisions (MVC). Multiple or bilateral long bone fractures are established risk factors. The syndrome characteristically manifests 24–72 h after injury with a triad of respiratory distress, neurological impairment, and petechial rash, though this complete triad is present in a minority of patients [5,6].

The pathophysiology of FES involves two principal theories; the mechanical theory emphasizes the role of direct vascular obstruction by fat emboli released from fractured bone marrow. The biochemical theory highlights that hydrolysis of neutral fat to free fatty acids triggers a systemic inflammatory cascade mediated by cytokines, including interleukin-6 (IL-6), tumor necrosis factor- $\alpha$ , and IL-1 $\beta$  [5,7].

Laboratory markers, including elevated C-reactive protein (CRP), elevated IL-6, falling hemoglobin (Hb), and thrombocytopenia, have been observed in association with FES. FES is often preceded by a period of inapparent hypoxia detectable by continuous pulse oximetry (CPOM), which may precede the full clinical syndrome by many hours and represents a critical early warning window [8]. Elevated serum IL-6 at 12 h post-trauma, with a proposed cut-off value of 131 pg/mL, has been specifically correlated with the subsequent development of FES in prospective clinical studies [9,10].

Overall, FES mortality has been reported between 5% and 20%,

with higher rates in older adults and those with greater injury severity [11,12]. In this case series, we describe six patients with FES between 2020 and 2025, with a focus on early clinical and inflammatory markers that may facilitate pre-emptive recognition before the syndrome becomes fully established and potentially irreversible.

**Materials and Methods**

This is a retrospective case series of six patients diagnosed with FES at between 2020 and 2025. Patient data were retrieved from medical records and the institutional mortality audit. The diagnosis of FES was established using Gurd’s

criteria – requiring at least two major criteria or one major and four minor criteria to be present.

Data collected included patient demographics (age, sex), date of injury, mechanism of injury, number of fractures, injury severity score (ISS), and sequential clinical parameters: Namely hypoxia, heart rate, respiratory rate, Glasgow coma scale (GCS) and any change therein, fever, petechiae, hypoalbuminemia and oliguria. Laboratory parameters of particular interest included serum IL-6, CRP, Hb, platelet count (Plt), and erythrocyte sedimentation rate (ESR). A focused sub-analysis table was constructed to examine the plausible link between ISS, number of fractures, IL-6, and CRP level with the subsequent development of FES.

**Table 2: Gurd’s criteria fulfillment – all six patients**

Criterion	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5†	Patient 6†
<b>Major criteria</b>						
Hypoxia/respiratory distress	+	+	+	+	+	+
Neurological deterioration (GCS drop)	+	+	+	+	+	+
Petechial rash	+	+	—	—	+	+
<b>Minor criteria</b>						
Tachycardia (>120 bpm)	+	—	—	—	+	—
Fever	—	—	—	—	+	—
Elevated ESR	+	+	+	+	NA	-
Thrombocytopenia/Low platelets	+	+	+	+	+	+
Anemia/Fall in Hb	+	+	+	+	+	+
Oliguria	+	—	+	+	—	—
Elevated CRP	+	+	+	+	NA	+
Elevated IL-6	+	NA	+	+	+	+
Reduced albumin	+	+	+	+	NA	NA
Gurd criteria satisfied	Yes 2 M+5 m	Yes 2 M+3 m	Yes 2 M+4 m	Yes 2 M+4 m	Yes 2 M+4 m	Yes 3 M+4 m

✓ present; – absent; NA: not available, D: Day, M: Major criterion, m: Minor criterion. †: Fatal outcome. IL-6: Interleukin-6, CRP: C-reactive protein, NA: Not available, GCS: Glasgow coma scale, ESR: Erythrocyte sedimentation rate, Hb: hemoglobin



## Case Series

### Case 1

A 25-year-old male presented following a MVC on February 12th, 2023. He sustained three long bone fractures with an ISS of 41/75, indicating severe trauma. On Day 1, he developed hypoxia with a heart rate of 125 bpm and a respiratory rate of 25 breaths/min. His initial GCS was 15/15. On Day 2, oliguria was noted, and by Day 3, petechiae appeared along with a decline in GCS, Hb was 8.5 g/dL, and Plt was 150,000/ $\mu$ L, both reduced from baseline, serum IL-6 was markedly elevated at 155 pg/mL, and CRP was 85 mg/L. ESR was 85 mm/h. albumin was reduced at 2.6 g/dL. Fever was absent throughout the hospital stay. Gurd's criteria were satisfied with hypoxia (major criterion), altered GCS, tachycardia, thrombocytopenia, and elevated ESR (minor criteria). He was admitted in intensive care unit (ICU) from Day 2, and all supportive care was given. Patient survived.

### Case 2

A 23-year-old female was admitted following an MVC on June 20th, 2020 with a single long bone fracture and an ISS of 34/75. Hypoxia was documented on Day 1, her heart rate was 104 bpm, and respiratory rate was 22 breaths/min. GCS on admission was 15/15, declining by Day 3. Petechiae were also observed on Day 3, Hb was 9.2 g/dL, and platelets were 156,000/ $\mu$ L. Serum IL-6 was not available for this patient; CRP was elevated at 36

mg/L, ESR was 56 mm/h, and albumin was 2.8 g/dL. No fever, jaundice, or oliguria was recorded. This case illustrates that FES can develop even with a single fracture; IL-6 data were not available. She was admitted in ICU from Day 2, and all supportive care was given. Patient survived.

### Case 3

A 32-year-old male presented following an MVC on June 14th, 2025, sustaining two long bone fractures. ISS was 31/75. Hypoxia developed on Day 2, heart rate was 112 bpm and respiratory rate was 22 breaths/min. GCS on Day 1 was 15/15, changing by Day 2. There were no petechiae. From Day 3, Hb was markedly low at 6.1 g/dL, and platelets were 142,000/ $\mu$ L. Serum IL-6 was 37 pg/mL, and CRP was elevated at 90 mg/L, ESR was 30 mm/h, and albumin was 2.8 g/dL. Oliguria was noted on Day 3. The delayed onset of hypoxia and the marked anemia underscore the heterogeneous clinical presentation of FES. He was admitted in ICU from Day 2, and all supportive care was given. Patient survived.

### Case 4

A 30-year-old male was admitted on September 13th, 2025 following an MVC with two long bone fractures and the highest ISS of the series at 46/75. Hypoxia developed on Day 1 with a heart rate of 116 bpm and respiratory rate of 24 breaths/min, and a GCS of 15/15. GCS declined on Day 2, and Plt was

156,000/ $\mu$ L, Hb was 7.8 g/dL, serum IL-6 was substantially elevated at 112 pg/mL, and CRP was 78 mg/L, ESR was 77 mm/h, and albumin was low at 2.4 g/dL. Oliguria was also documented on Day 2. No fever, and petechiae were documented. This case, with the highest ISS and an early rise in IL-6 levels, demonstrates the plausible link between injury severity and FES risk.

### Case 5

A 23-year-old male was admitted following a MVC on November 26th, 2021, sustaining two long bone fractures: An open fracture-dislocation of the right ankle and a displaced fracture of the left shaft of femur. On Day 1, he presented with inapparent

**Table 3: Frequency of key features in all six patients**

Feature	Present (n)	Frequency (%)	Comment
Hypoxia/respiratory distress	6	100	Universal; earliest criterion
Neurological deterioration (GCS drop)	6	100	All had GCS 15/15 on Day 1
Petechial rash	4	67	Absent in Cases 3 and 4
Elevated ESR	4	67	Borderline in Case 3; not measured in Case 5
Thrombocytopenia/low platelets	6	100	All patients, including Case 4 (156 k, borderline)
Anemia/Fall in Hb	6	100	Present in all patients
Oliguria	3	50	Cases 1, 3, 4
Tachycardia (>120 bpm)	2	33	Cases 1, 5
Fever	1	17	Case 5 only (Day 4)
Reduced albumin (<3.5 g/dL)	4	67	—
Elevated IL-6	5	83	Not measured in Case 2
IL-6 $\geq$ 131 pg/mL (proposed threshold) <sup>10</sup>	3	50	Cases 1, 5, both had multiple fracture and one had fatal outcome
Elevated CRP	5	83	Not measured in Case 5
Mortality	2	33	Cases 5 (Day 7) and 6 (Day 16)

**IL-6: Interleukin-6, CRP: C-reactive protein, GCS: Glasgow coma scale, ESR: Erythrocyte sedimentation rate, Hb: Hemoglobin**

hypoxia (SpO<sub>2</sub> 91% in room air) and a heart rate of 112 bpm, with a respiratory rate consistent with mild tachypnea. His initial GCS was 15/15, Hb was 12.6 g/dL, and Plt was 165,000/ $\mu$ L on admission. Serum IL-6 and CRP were not measured on Day 1. Fever was absent. No petechiae were noted on Day 1.

On Day 2, he developed sudden, profound desaturation with SpO<sub>2</sub> falling to 55% in room air with a respiratory rate of 36/min and heart rate of 99 bpm, GCS declined to 12/15, Hb fell to 9.0 g/dL, and Plt dropped to 80,000/ $\mu$ L, serum IL-6 was markedly elevated at 356.6 pg/mL, the highest recorded in this series. Conjunctival petechiae were noted. Non-invasive ventilation was initiated. Fever was noted on Day 4. The patient deteriorated progressively despite all supportive care and died on Day 7. Covid-19 was ruled out by reverse transcription polymerase chain reaction.

### Case 6

A 21-year-old male presented following a road traffic accident on February 24th, 2025, sustaining bilateral shaft of femur fractures, a left distal end radius fracture, and a right-sided rib fracture – the highest fracture burden in this series. On Day 1, he presented with inapparent hypoxia (SpO<sub>2</sub> 94% on 4 L of supplemental oxygen) and a heart rate of 93 bpm with a respiratory rate of 13/min. His initial GCS was 15/15, Hb was 12.3 g/dL, and Plt was 150,000/ $\mu$ L on admission.

On Day 2, serum IL-6 was 117 pg/mL and CRP was 5.9 mg/L, GCS began to deteriorate progressively through the day, atypical petechiae were noted near the axilla. On Day 3, classic petechiae were present over the chest, abdomen, and axilla, Hb fell to 8.9 g/dL and Plt to 75,600/ $\mu$ L, CRP rose to 9.27 mg/L. By Day 4, funduscopy revealed retinal hypoxia and intraretinal hemorrhage, suggestive of retinal fat emboli. The patient developed progressive respiratory failure, acute kidney injury, and multiorgan dysfunction, and died on Day 16 despite all supportive care.

### Discussion

This six-patient case-series reinforces the established epidemiology of FES as a condition predominantly affecting young adults following high-energy road traffic injuries and long bone fractures (Table 1). The mean age in our series was 25.3 years (range 21–32), consistent with published data identifying patients under 40 as disproportionately affected [11,12]. All six patients sustained injuries from road traffic accidents, the dominant mechanism in published literature. All but one had multiple fractures (Table 1).

Both fatal cases had the high fracture burden in the series and

demonstrated the most marked IL-6 elevation; supporting the hypothesis that injury severity and inflammatory load together determine FES severity. Case 5 had an ISS of 29/75, and Case 6 had an ISS of 36/75 (Table 1). The overall case-fatality rate was as low as 5.8% in nationwide Japanese registry data [12]. Our series had high mortality.

Serum IL-6 was elevated in all five patients for whom it was measured (Table 1). In the two fatal cases, IL-6 was the most markedly elevated; 356.6 pg/mL (Case 5) and 117 pg/mL on Day 2 in Case 6. The surviving cases had a range of 37–155 pg/mL. All three patients whose IL-6 exceeded the proposed 131 pg/mL cut-off [10] and had the most severe clinical course, including one fatality.

Prakash et al. and Yoga et al. demonstrated that serum IL-6 at 12 h post-trauma, with a cut-off of 131 pg/mL, was significantly predictive of subsequent FES development [9,10]. Our series corroborates this finding. Importantly, in Case 6, IL-6 was already 117 pg/mL on Day 2, before the clinical diagnosis of FES was formally established; thus, potentially representing an early biochemical signal of evolving FES that preceded the full clinical syndrome.

The general tendency of rapid fall in Hb and thrombocytopenia was noted in all our cases (Table 2). CRP was elevated in all five patients (Table 1) for whom it was measured (100%), ranging from 5.9 to 90 mg/L. Elevated CRP, alongside rising ESR, possibly reflects the systemic acute-phase response to fat emboli and free fatty acid-mediated tissue injury. In resource-limited settings where IL-6 assays are unavailable, CRP and ESR may serve as accessible surrogates for inflammatory monitoring in at-risk patients.

All patients showed a fall in Hb below normal or a documented drop from admission. Thrombocytopenia or low-normal Plts were confirmed in all 6/6 patients. FES is a clinical diagnosis by a process of exclusion. No laboratory investigation is sufficiently sensitive or specific to confirm the diagnosis in isolation, and no definitive test exists [5,7].

Gurd and Wilson's criteria, the most widely cited diagnostic framework, requires at least two major criteria (respiratory distress, cerebral involvement, petechial rash) or one major and four minor criteria [1,5]. A critical and often underappreciated limitation is that these criteria are retrospective in their applicability: By the time sufficient criteria are met to satisfy the diagnostic threshold, the syndrome is frequently advanced and potentially irreversible. This is illustrated in the current series – all patients had established hypoxia and GCS decline much before petechiae appeared, and the formal diagnosis was confirmed only at an advanced stage of the disease process.

All six patients had intact GCS (15/15) on Day 1; all developed

GCS decline on Day 2 onwards after admission (100%). Petechiae were documented in 4/6 (67%), consistent with published literature (20–50% prevalence). Notably, petechiae, a major criterion, was absent in 2/6 patients (Cases 3 and 4, 33%) and appeared late (Day 2–3) in the remaining four.

This is consistent with the published petechial prevalence of 20–50% in FES [6]. Therefore, the absence of petechiae cannot be used to exclude the diagnosis in a clinically suspicious case. Gurd and Wilson's criteria were fulfilled in all six cases (Tables 2 and 3). All six patients satisfied Gurd's criteria. Case 6 was the only patient to fulfill all three major criteria simultaneously. Hypoxia, GCS drop, fall in Hb, and elevated IL-6/CRP were the most consistent findings. Petechiae, although a Gurd major criterion, were absent in 33% of patients. Thrombocytopenia or low-normal platelets were present in 6/6. Table 3 summarizes the Frequency of the key features of FES in all six patients. Arterial blood gas (ABG) analysis may be normal even in the presence of inapparent hypoxia detectable by CPOM – a finding with significant clinical implications [8]. A normal ABG does not exclude FES. Echocardiography is a useful screening tool in excluding pulmonary venous thromboembolism, which may mimic FES and should be part of the differential workup. Fundoscopy, performed in only one case in our series (retinal fat emboli), may be valuable when performed.

The most important clinical lesson from this series is the concept of the window of reversibility. A pre-clinical or early clinical phase of FES exists – characterised by inapparent hypoxia, rising inflammatory markers, falling Hb, and subtle hematological changes, during which early supportive care and rapid fracture stabilisation may potentially arrest the cascade before progression to irreversible multiorgan failure. Once Gurd's criteria are fully satisfied, the disease is already at an advanced and clinically overt stage, associated with substantially elevated mortality risk. At this point, the therapeutic window has considerably narrowed. The aim must therefore be to recognize and act on early warning features – particularly inapparent hypoxia and early IL-6 elevation, before the fulminant syndrome fully established.

In the two fatal cases, the window of reversibility was compromised by the severity of the initial injury burden. In Case 5, plausible contributing factors included delay in ICU admission (covid era) and inadequate primary-level fracture immobilization before patient transfer. In Case 6, despite appropriate damage control orthopedics, the bilateral high-energy femoral fracture burden possibly generated an inflammatory cascade that progressed to acute respiratory distress syndrome, acute kidney injury, and multiorgan failure refractory to all supportive measures.

Early stabilization of long bone fracture is recommended to

minimize bone marrow embolization into the venous system [13]. Early surgical fixation of long bone within 24 h after trauma has a lower risk and severity than delayed fixation. External fixation or fixation with plates and screws produces less lung injury than intra-medullary nailing, and venting the medullary canal during nailing reduces the emboli. Small diameter nails and unreamed nailing have been mentioned as being useful in the prevention of FES [14].

Current evidence suggests that the use of corticosteroid for the prevention of FES may be considered for initial prophylaxis. A recent meta-analysis of six small randomized, controlled trials found that prophylactic corticosteroid administration reduced risk of FES development (relative risk [RR] 0.16, 95% confidence interval [CI]:0.08–0.35) and hypoxemia (RR 0.34, 95% CI 0.19–0.59) [15].

Although a combination of antibiotics and anticoagulants as part of supportive care is suggested [16]; in the setting of trauma and pre-existing hematologic abnormalities, anticoagulants may be harmful if used routinely [12]. Choice of antibiotics may be based on updated institutional protocols for open wounds. Pharmacological prophylaxis may, however, be recommended for prevention of deep vein thrombosis and pulmonary embolism; based on risk-benefit analysis.

Based on the current understanding of available literature and the collective lessons learnt from this series, the following prevention and early detection strategies may be recommended for patients presenting with long bone fractures:

1. Rapid fracture stabilisation: Primary-level immobilization (Thomas splint, slab) must be applied before transfer. Delayed or inadequate immobilization allows ongoing fat embolization from unstable fracture ends. [17]. Early stabilization of long bone fracture is recommended (external fixator or plate-screw construct or unreamed nail) to minimize bone marrow embolization into the venous system [13]. Early surgical fixation of long bone within 24 h after trauma has lower risk and severity than delayed fixation.
2. Adequate trauma resuscitation: Goal-directed resuscitation from the point of first contact. Hypovolemia worsens FES by compounding hypoxia and impairing end-organ perfusion. Albumin may be added at an early stage of resuscitation, to bind free fatty acid to form lipoprotein.
3. CPOM: All patients with long bone or pelvic fractures should be placed on CPOM from the time of admission. Inapparent hypoxia detectable by CPOM may precede the full clinical syndrome by many hours, even when ABG is normal [8].
4. Serial IL-6 measurement at 6-, 12-, and 24-h post-injury: In patients with bilateral or high-energy long bone fractures and high ISS, serial serum IL-6 measurement (particularly at 12 h)

may allow early identification of those at risk of progressing to FES. The 131 pg/mL cut-off appears to be a clinically useful threshold [9,10].

5. Universal team awareness of Gurd's criteria and evolving FES: Every member of the trauma team – orthopedic surgeons, anesthesiologists, ICU physicians, nursing staff, and ward doctors must be aware of Gurd's criteria and the early warning signs of evolving FES. Delays in recognition at any level of the care chain can result in potentially preventable mortality.

6. Consider prophylactic corticosteroids: Recent meta-analyses suggest prophylactic corticosteroids may reduce the risk of FES by up to 78% and reduce hypoxia and morbidity [15,18,19]. Brief low-dose steroid administration early in the course may be carefully considered based on the levels of inflammatory markers (CRP, IL-6) in high-risk patients, acknowledging that this approach remains under investigation.

7. The standard of contemporary treatment is supportive: With good early care, one can expect a good outcome [20,21]. However, days of mechanical ventilation may be required before full recovery [21].

### Conclusion

This six-patient case series of FES managed between 2020 and 2025 supports the following:

- First, FES predominantly affects young adults following high-energy road traffic injuries involving long bone fractures. All six patients in this series were aged 21–32 years.
- Second, hypoxia and neurological deterioration (Altered GCS) were universal findings present in all six patients (100%). Both preceded the Gurd's criteria threshold in every case, making them the most reliable indicators of evolving FES.
- Third, elevated IL-6 and elevated CRP were present in all patients for whom these were measured (100%). IL-6 exceeding 131 pg/mL was associated with more severe disease

and was found in the two fatal cases and the most severe FES survivor (Case 1). Serial IL-6 monitoring at 6-, 12-, and 24-h post-injury, combined with CPOM, represents the most promising early warning strategy currently available.

- Fourth, petechiae were absent in 33% of patients and appeared late in those who did develop them. A normal ABG does not exclude FES. Neither finding is required before clinical suspicion and acting upon.

- Fifth, Gurd's criteria confirm FES only after the disease is clinically established and potentially irreversible. They function as a confirmation tool rather than an early warning tool. Positive criteria should be understood as a signal that the disease has already advanced to a potentially high-risk of mortality.

- Sixth, the two fatal cases demonstrate that mortality in FES is associated with high fracture burden, high IL6 levels, delay in adequate primary stabilisation and ICU-level admission, and such cases may rapidly progress to multiorgan failure. Not all mortalities are preventable, but the prevention window potentially lies in the early hours after injury.

- Seventh, prevention of FES mortality requires a systems-level approach: early fracture stabilisation from the point of first contact, goal-directed resuscitation, CPOM, serial IL-6 monitoring, and universal awareness of early FES signs among all healthcare providers involved in trauma care.

### Clinical Message

FES is a rare but potentially fatal complication of major long bone fractures. An early diagnosis of an evolving FES is paramount before it progresses to an irreversible fulminant FES. Hypoxia and markedly elevated biomarker serum IL-6 levels appear to be the earliest detectable indicators of evolving FES, preceding the full clinical syndrome. Team awareness of such early warning sign may potentially prevent FES-related morbidity and mortality.

**Declaration of patient consent:** The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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