Scientific Foundation SPIROSKI, Skopje, Republic of Macedonia
Open Access Macedonian Journal of Medical Sciences. 2022 Dec 19; 10(C):331-335.
https://doi.org/10.3889/oamjms.2022.11169
eISSN: 1857-9655
Category: *C - Case Reports*
Section: *Case Report in Internal Medicine*

Fat Embolism Syndrome Without Bone Fracture: Is It Possible?

Mustafa Bajraktari[1,2]*, Majlinda Naco[2], Gentian Huti[1,2], Blerim Arapi[1], Rudin Domi[1,2]

[1]Department of Anaesthesia Intensive Care, American Hospital, Tirana, Albania; [2]Department of Anaesthesia Intensive Care, Faculty of Medicine, University of Medicine, Tirana, Albania

Edited by: Mirko Spiroski
Citation: Bajraktari M, Naco M, Huti G, Arapi B, Domi R. Fat Embolism Syndrome Without Bone Fracture: Is It Possible? Open Access Maced J Med Sci. 2022 Dec 19; 10(C):331-335. https://doi.org/10.3889/oamjms.2022.11169
Keywords: Fat embolism syndrome; Prophylactic measures; Adult respiratory distress syndrome; Long bone fracture
***Correspondence:** Mustafa Bajraktari, Department of Anaesthesia Intensive Care, American Hospital, Tirana, Albania. E-mail: m.bajraktari@live.com
Received: 26-Oct-2022
Revised: 09-Dec-2022
Accepted: 12-Dec-2022

Funding: This research did not receive any financial support
Competing Interests: The authors have declared that no competing interests exist

Abstract

BACKGROUND: Fat embolism syndrome is a life challenge syndrome. Early diagnosing and treatment can significantly improve the patient's prognosis and likelihood of success. This syndrome occurs mainly after long bones fractures or orthopedic surgery up to 95% of diagnosed cases, but in unusual situation can be faced as well. These rare situations include diabetes mellitus, video-assisted thoracoscopies, fatty liver, and fat injection in plastic and cosmetic procedures. The likelihood of this syndrome can be increased if multiplex long bones fractures occur in the same patient simultaneously. This syndrome is usually manifested with respiratory changes (hypoxemia and ARDS), neurological focal symptoms (confusion, headache, aphasia, and hemiplegia), and skin abnormalities (petechias, and rush in conjunctiva and oral mucosa). The clinical scenario begins typically after 24−72 h of injury, and mainly, respiratory changes are the first, followed by neurological abnormalities and finally petechias as the most significant sign.

CASE REPORT: In this case, we report a rare case of unexpected fat embolism syndrome after soft-tissue minimal trauma. This is the first case that we faced according to literature, and the aim of reporting this case is to emphasize that fat syndrome embolism can happen perhaps in every trauma patient even in minor soft-tissue trauma in absence of bone fractures.

CONCLUSION: We strongly suggest that this case should make the physicians taking in consideration fat embolism syndrome even if bone fracture missed, to early diagnosing and adequately treating the patient, and optimizing his chances to survive.

Introduction

The anesthesiologists and intensivists can often face several life-threating situations during their daily practice. Embolisms of all origins are some of these critical events. Fat embolism is relatively rare and generally related to long bone fractures and orthopedic surgery [1], [2]. It is recently reported that 95% of fat embolism are due to significant trauma [3]. Several non-traumatic situations are related to fat embolism as liposuction [4], bone marrow biopsy and transplant [4], video-assisted thoracoscopy [4], [5], pancreatitis and gastrectomy [6], [7], and massive soft tissue [7]. There is only one interesting paper in the literature that describes cerebral fat embolism in the absence of long bone fractures [6]. Hence, our case according to literature would be a rare case that after minor soft-tissues trauma, fat embolism syndrome can be developed. Clinical scenario typically begins 24−72 h after trauma and usually is firstly presented with respiratory signs and symptoms such as hypoxemia, dyspnea, and tachypnea. The pulmonary abnormalities can progress from mild forms to adult respiratory distress syndrome (ARDS) making immediate respiratory support up to mechanical ventilation and endotracheal intubation [8]. The second group of symptoms is neurological symptoms usually presented later than respiratory changes. The most common neurological symptoms are headache, confusion, aphasia, and focal deficits (hemiplegia) due to the occluded brain territory [9]. The latest group of symptoms is petechias in oral mucosa, conjunctiva, chest, axilla, and face, occurring by embolization of small vessels causing extravasation. Petechias are faced up to 60% of diagnosed patients, but it occurs late during the disease. Nevertheless, petechias are considered almost pathognomonic sign of fat embolism syndrome [10]. Pathophysiology of fat embolism syndrome is based in two theories: mechanical obstruction and biochemical theory. Biochemical theory supported idea that neutral fat produces free fatty acids which cause vasodilatation and increased vascular permeability and bleeding. The mechanical theory is based on phenomena of obstruction due to aggregation of fat with platelets and fibrine resulting in pulmonary vascular obstruction and hypoxemia [11].

Case Report

A 21-year-old man presented in emergency department after suffered a minor motor vehicle accident causing a distortion of his left talocrural

articulation. Due to minor trauma, he did not fortunately suffer head or thoracic trauma. In admission, he was hemodynamically stable. Biochemistry findings revealed hg 11.7 g/dl, hematocrit of 38 %, an arterial partial pressure of oxygen (PaO2) of 112 mmHg, and apparently normal chest X-Ray. 18 h later, he was readmitted in hospital for developing tachypnea (respiratory rate 38−41), tachycardia (heart rate 132 bpm), febrile 38.6, and confusion. Gas analyses monstered a PaO2 of 54 mmHg in admission. The patient remained hemodynamically stable, afebrile, confused, and conjunctival petechiae which were noted. Due to persistent hypoxemia, the patient was intubated and transferred to ICU. Before being fully sedated, neurological examination revealed no slight tendence for opening eyes under pain, abnormal flexion by painful stimulation, so GCS 7−8 points was determined taking in consideration the anesthetic drugs effects duration. The patient was put in sedation and muscle relaxants to be better mechanically ventilated. After 6 h, we tried to wake up the patient stopping sedation, but his saturation was 60−65% putting him again in SIMV, FiO_2 of 80%, PEEP 8 cm H_2O, Tidal volume 500 ml, respiratory rate 16, and I: E 1:2. Hemodynamics remain stable despite hemodynamically negative effects of ventilation, PEEP, and sedation. His vital signs were BP around 95/50 mmHg, heart rate 124 bpm, sinus rhythm, O_2 saturation 96%, and a PaO2 85−94 mmHg under FiO_2 80%. Laboratory examinations found hemoglobin level of 10.2 g/dl, white blood cells 9.60 × 10^3/uL with 75% neutrophiles and 23% lymphocytes, platelets 102.00 × 10^3 uL, normal values of electrolytes, BUN 28 mg/dl, and creatinine 1.1 mg/dl. Chest X-rays (Figure 1) demonstrated diffuse pulmonary infiltrates confirmed by significant consolidations in CT scan (Figure 2). Fat embolism syndrome was suspected, and the patient remained mechanically ventilated, methylprednisolone 10 mg/kg, PPI drugs, antibiotics (cefuroxime and amikacin), sedation with propofol and fentanyl, anticoagulants (enoxaparin 0.4), and mannitol. Due to the severity of hypoxemia, we followed the patient by bedside chest X-ray instead of CT scan. Fat globules on bronchoalveolar lavage confirmed the diagnosis of fat embolism syndrome. On the second day of admission, the patient continued to require ventilatory support. Vital signs remained stable with BP 125/75 mmHg, HR = 106 bpm, RR = 14 bpm, UO 100 ml/h, PaO2 137−149 mmHg, SO_2 = 99% under FiO_2 60%, and PEEP 7 cm H_2O. On 3rd day, the patient was remarkable stable, and after reduced FiO_2 up to 50%, we began weaning from ventilator. The sedation was stopped, and the ventilatory regimen was changed to pressure support ventilation with PS 15, PEEP 6, and FiO2 50%. On the 4th day, the patient began to open his eyes in response to painful stimuli. The pupils also began reacting to light and accommodation. A decision was made to extubate due to stable gas analyses (PaO_2 177−192 mmHg with FiO_2 45%, spontaneously respiratory rate 19, decreased PEEP at 5 cm H_2O and pressure support at 9−10), improved conscious, stabilized hemodynamic, and UO approximately 100−110 ml/h. After the 8th day, the patient was significantly improved being afebrile, hemodynamically, and respiratory stable, and his hemorrhaging petechia was resolved. His GCS was improved to GCS 10–11 points (E4/M4-5/V2). By this time, he was scoring a GCS of E4M4V2 = 10/15. Within the next 2 days, the patient was stable enough to be transferred to surgery service, where he spent another 5 days before he was uneventfully discharged in home.

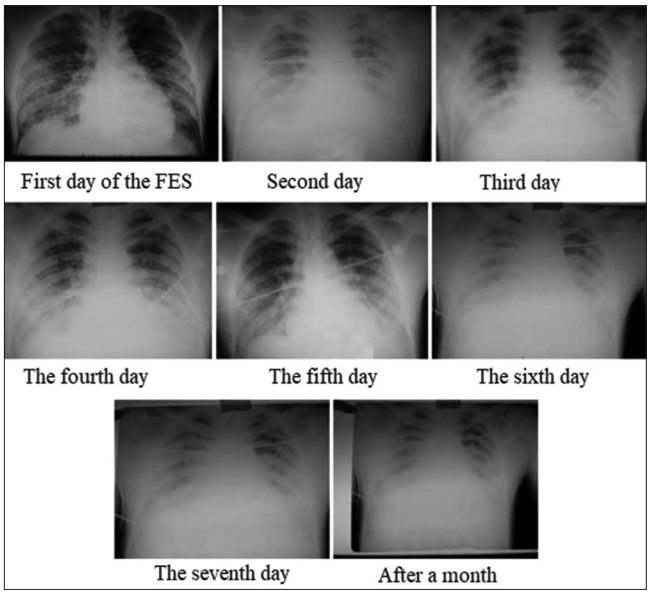

Figure 1: X-rays

Discussion

It is recently ported that the incidence of fat embolism syndrome (FES) after bone fractures varies 0.9−22% [3], [4], [12]. This syndrome was firstly described by Guld in the late 1970 remaining as the principial detailed described model. Bulger *et al*. published an interesting paper, collecting data of 10 years. The authors described in detail the clinical features, diagnosis, pathophysiological mechanisms, and treatment of this syndrome. Fat embolism syndrome is faced mainly in patients with long bone fractures or in those who undergo orthopedic surgery and procedures [4], [12]. According to the literature hypoxemia, deteriorating mental status and petechiae are the main diagnostic criteria. The secondary diagnostic signs include tachycardia, fever, anemia, and thrombocytopenia [3], [13]. Bronchial lavage sustained the diagnosis of FES by the presence of fat globule in pulmonary secretion. Our patient had no bone fracture after a minor vehicle accident, and in the earlier admission had no compromised vital signs. After 18 h, the patient developed suddenly hypoxemia and impaired mental status. Due to the persistent refractory

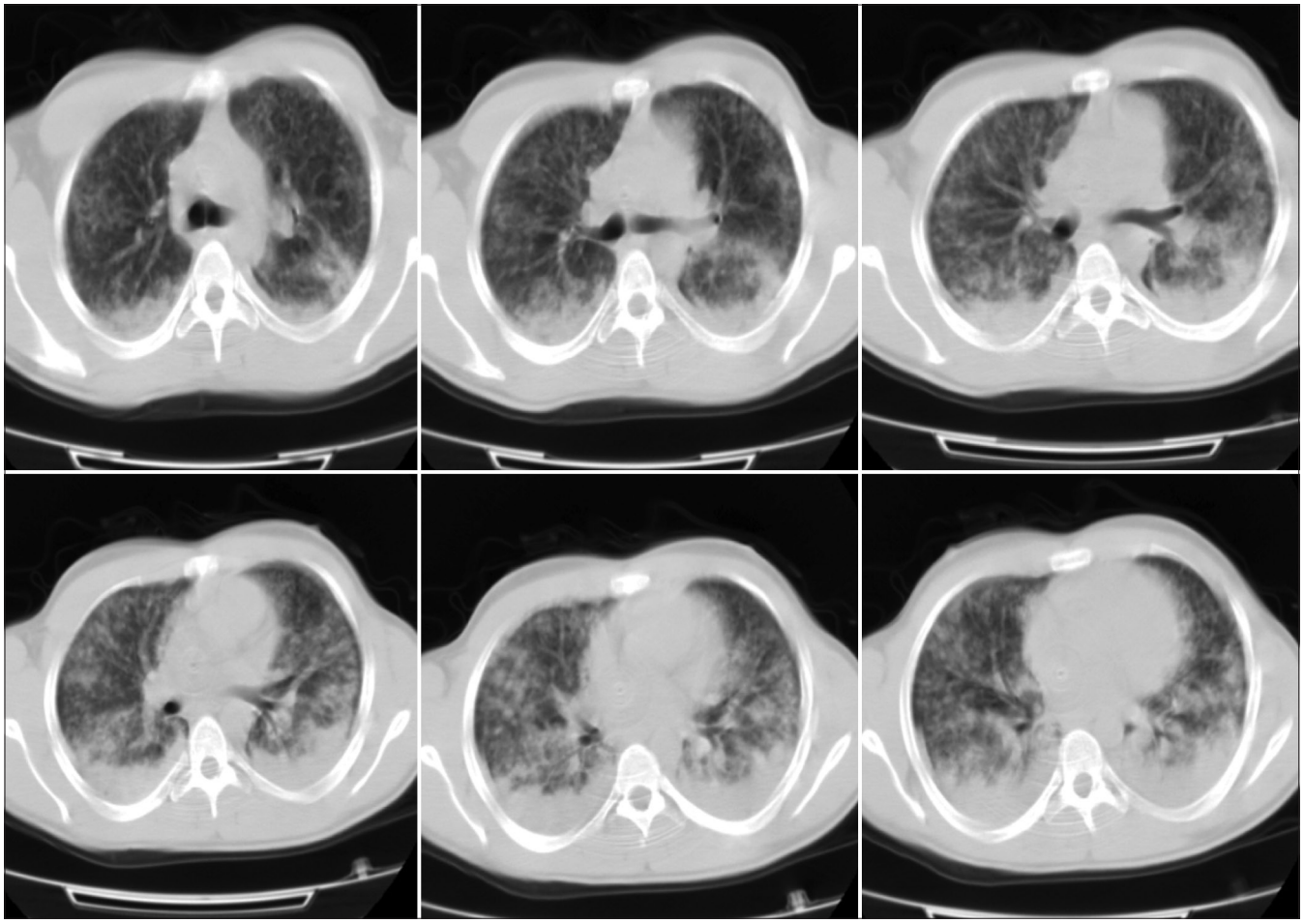

Figure 2: Chest CT scan

hypoxemia and altered conscience, the patient was intubated and mechanically ventilated. After he was intubated, several hours petechia was noticed. This is a normal clinical diagnosis according to the literature [14] even the bone fracture missed. Kwiatt *et al*. published an interesting paper regarding FES, which summarizes all the aspects from clinical elements up to diagnosis and management of this syndrome. That was the reason of highly suspected FES when the patient was readmitted in hospital. Once his clinical presentation was completed by petechia, the diagnosis of FES was clear even unexpected after a minor soft-tissue trauma without bone fracture [15]. The diagnosis was certified by lung imaging (CT scan and chest X-ray), and from the presence of fat globulus in pulmonary secretion.

There are two theories explaining the pathogenesis of FES [16]. According to the "mechanical" theory, free fat particles from the bone marrow can enter vein sinusoids at the site of the fracture and embolize the pulmonary arterioles. The "biochemical" theory suggests that the degradation of fat can produce free fatty acids. Free fatty acids can induce endothelial severe dysfunction associated with increased permeability, vasodilatation, and bleeding. The last mechanism occurs mainly in pathological situations, minor trauma without long bone fractures, and may be the mechanism in our patient. The "biochemical" mechanism is more silent, and this explains the situation in our patient.

Cerebral fat embolism seems to be catastrophic complication but often is an incomplete an uncommon form of FES. CFE has an incidence that varies from 0,9% to 2.2%, but the incidence increases if FES occurs after fractures [17]. Even in majority CFE is limited, the mortality rate is reported up to 10%. Aman *et al*. have in their paper focused on cerebral fat embolism after long bone fractures. They described this important component of fat embolism syndrome, diagnosis, and management. The authors emphasized the role of MRI in diagnosing the cerebral aspect of FES. Our patient had altered mental status due to hypoxemia and no CFE was detected in head CT scan and MRI [18]. Hence, the cerebral abnormality was missing in our patients and not fulfilled the original Gurd description.

FSE was firstly described in 1970 by Gurd *et al*. [19], but our patient totally fulfilled the Schonfeld's fat embolism criteria whose include petechial rash, diffuse pulmonary infiltrates, hypoxemia, confusion, fever above 38°C, and respiratory rate over 30/min [20]. Schonfeld had modified the diagnostic criteria making them more clinical criteria, describing fat embolism syndrome as a clinical scenario especially after long bone fractures. To diagnose, our patient was too

difficult since pulmonary complications may be adult respiratory distress syndrome or severe pneumonia as well. Our patient had hypoxemia, thrombocythemia, fever 38.6, heart rate 132 bpm, tachypnea 38−41 in a minute, pulmonary infiltrates in CT scan, and X-ray, and lastly but determined in diagnosis petechial rush. The presence of petechial rush finally completed the Schonfeld criteria and assuring us for the real diagnose. The only thing missed in our patient's medical history was long bone fracture. Hence, the real diagnose was far from medical history, and differential diagnosis was difficult to be made. After petechial rush, and positive bronchoalveolar lavage for fat globules, the situation was clear to us even we suspected the diagnosis before but not able to confirm fat embolism syndrome.

Differential diagnosis with other forms of embolisms is an outstanding issue to deal with. There are some forms of embolism as thromboembolism, air embolism, amniotic embolism, fat embolism, and tumoral embolism. Our patient is a young man so amniotic embolism is excluded from the study. All other embolisms forms may be manifested with severe hemodynamic derangements due to the fact of acute right heart failure. Fat embolism is the only form typically associated with petechial rush. In thromboembolic embolism, the diagnosing process can be facilitated by medical history, echocardiography, and pulmonary Angio CT scan finding very specific culprit lesionin on pulmonary artery, but not pulmonary infiltrates typically seen in ARDS and fat embolism. Airway embolism is another form of embolism. This type of embolism is faced in all the situation that right atrium is at least 10 cm under the surgical site as for example in sitting position during posterior fossa neurosurgical procedures, in head and neck surgeries, even rare during hysterectomy if the uterus is higher than 10 cm from the right atrium level. Hence, air embolism is often faced during specific procedures, mainly presented with severe hemodynamic abnormalities, with mild hypoxemia, suddenly decreased of ETCO2, and no pulmonary infiltrates in pulmonary CT scan. Our case presented after minor trauma, no surgery, no bone fractures, hemodynamically stable, severe hypoxemia, pulmonary infiltrates in CT scan, febrile, and thrombocytopenic. After bronchial lavage and petechial rush, the diagnosis was clear to us. Meanwhile, we tended to support the patients with standard measures. If the medical history would include bone fracture, then our diagnostic idea would be earlier clear, and the prophylactic use of steroids would be earlier instituted improving the patient's prognosis. The treatment regimen is the same for fat embolism syndrome regardless bone fracture, but early diagnosing and early use of methylprednisolone, which is a cornerstone of treatment, may improve patient outcome.

We strongly suggest that this case should make the physicians taking in consideration fat embolism syndrome even if bone fracture missed, to early diagnosing and adequately treating the patient, and optimizing his chances to survive.

Authors' Contributions

Mustafa Bajraktari collected data, Rudin Domi write article, Majlinda Naco revised literature, Gentian Huti, and Blerim Arapi supervised article.

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