

Case Report

A Case of Fat Embolism Syndrome after Elective Hip Arthroplasty

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Doi: [10.5281/zenodo.18039224](https://doi.org/10.5281/zenodo.18039224).

Abstract

Fat embolism syndrome (FES) is a rare clinical entity caused by the release of fat globules in blood circulation. It presents with pulmonary as well as systemic symptoms and is usually observed in connection to long bone fractures and orthopaedic procedures such as arthroplasties. In literature, case reports of FES are numbered, and diagnosis can be challenging as there is no pathognomonic sign or symptom for FES. The authors present the case of a 68-year-old male with dyspnea and altered mental status following elective arthroplasty. After extensive work-up and exclusion of other likely clinical entities, the authors reached the diagnosis of fat embolism syndrome. Supportive care was administered without anticoagulant agents or corticosteroids with subsequent recovery and discharge of the patient. The diagnosis of FES requires a high level of clinical suspicion and can often be elusive. The authors highlight the importance of timely diagnosis and supportive care.

Keywords: fat emboli syndrome, hip arthroplasty, elective arthroplasty, orthopaedics

Introduction

Fat embolism (FE) is the presence of fat globules in pulmonary circulation. Fat embolism syndrome (FES) is a rare clinical syndrome caused by fat released into blood circulation and includes pulmonary and systemic symptoms [1-3]. The majority of cases of FES are related to long bone fractures and orthopaedic procedures such as arthroplasties [1-3]. Incidence is variable with rates of FES ranging from 1% to 10% in trauma patients, 0.1% to 12% after total knee arthroplasty and 0.6% to 10% following total hip replacement [2-4]. Regarding the pathogenesis of FES, there are two main mechanisms described in literature: first, the mechanical theory where fat emboli enter the bloodstream through disrupted tissues and, second, the biochemical theory where intermediaries of fat are released and cause inflammation [3-5]. However, the exact pathophysiology of FES remains unknown. FES can present with a classic triad of non-specific symptoms including hypoxemia, neurologic abnormalities, and the presentation of a petechial rash. Chest radiographs are normal in

the majority of cases. CT scan may reveal well-demarcated ground glass opacities or ill-defined centrilobular nodules [3-5]. The diagnosis requires a high level of clinical suspicion as there is no pathognomonic sign or symptom of FES and in this case further imaging with CT scan is indicated. Treatment of FES is supportive with fluid resuscitation and oxygenation of the patient. The importance of early immobilization of long bone fractures and further staged definitive fixation in polytrauma patients has been well established [6]. The administration of prophylactic corticosteroids remains controversial and is not routinely used [4-6].

In this paper, we present the case of a 68-year-old male with a sudden onset of dyspnea and altered mental status occurring within hours from an elective right hip arthroplasty. After extensive diagnostic investigation and exclusion of other likely diagnoses, the clinical presentation was attributed to FES. Following supportive care and close monitoring, the patient displayed clinical and laboratory improvement leading to his discharge after 10 days of hospitalization. This case report

demonstrates the high degree of clinical suspicion required for timely diagnosis and the sufficiency of supportive treatment for the achievement of a favorable outcome.

Case Presentation

Medical history & clinical presentation

A 68-year-old male presented at the emergency department due to dyspnea and altered mental status with progressive deterioration over a span of 3 days. It was reported that the patient had undergone elective hip arthroplasty 3 days earlier in another hospital while the onset of symptoms was noted a few hours post-operatively. His medical history consisted of arterial hypertension and chronic kidney disease undergoing haemodialysis for the past 8 years.

At the emergency department, the patient was afebrile and haemodynamically stable, mildly tachycardic (approximately 105-110 bpm) and tachypnoeic (approximately 25 breaths/minute) while he presented with severe hypoxemia requiring oxygenation with a non-rebreather mask. Auscultation of the respiratory system revealed crackles in both lungs. From a neurological standpoint, the patient was confused, disoriented in time and place, his eyes were automatically open with pupils of the same size and reactive to light, he responded to verbal commands or questions with inappropriate words, he localized pain while he maintained movement in all four limbs without presenting any specific focal symptoms. The rest of the physical examination revealed rhythmic heart sounds without any additional murmurs, no jugular vein distention or palpable edema, mild sensitivity upon palpation of the abdomen as well as a haematoma around the surgical area/incision without concomitant signs of infection. Laboratory analysis showed a marked inflammatory syndrome with elevated CRP (347 mg/l, NR: < 3) and fibrinogen (1078 mg/dl, NR: 200 - 400), a mildly elevated total neutrophil count (76,4%) with a normal total WBC count (9.400/ μ l), while CPK and d-dimers values were also elevated (1768 u/l, NR: < 170

and 2.72 μ g/ml, NR: < 0.50 respectively).

Differential diagnosis & work up.

Taking under consideration the aforementioned clinical presentation and history, our differential diagnosis included the following: septic syndrome due to infection (pulmonary infection - possibly inspiration post extubation, CNS infection, vascular infection, surgical site infection), pulmonary embolism, pulmonary oedema, stroke (possibly ischaemic), fat embolism syndrome.

Examining the possibility of infection, blood cultures were obtained although no pathogen was isolated. Urinalysis did not reveal specific findings while urine culture was sterile. To rule out a CNS infection, a brain CT was performed and showed no abnormalities. Lumbar puncture revealed 1 lymphocyte and slightly elevated glucose in the CNS fluid, while PCR and culture tests were negative. Finally, the surgical site did not present any sign of infection throughout the duration of the hospitalization of the patient.

Due to the clinical presentation (severe hypoxaemia, tachycardia) and the relevant history of recent surgery, a lung CT was performed which excluded the possibility of pulmonary embolism but showed ground-glass lesions in both lungs. Furthermore, cardiology consultation was conducted with an echocardiogram which did not reveal any signs of volume overload or pulmonary hypertension. Moreover, a brain MRI was performed after admission which did not show any specific pathologic findings thus excluding the diagnosis of a cerebral event.

Treatment & outcome

After thorough diagnostic investigation which excluded all other possible differential diagnoses, the presenting signs and symptoms of the patient were attributed to fat embolism syndrome post-arthroplasty of the right hip. Although no petechiae or other type of rash was recorded, our patient met 2 out of 3 major criteria set by Gurd and Wilson thus making fat embolism syndrome a probable diagnosis in this

case. Indeed, the patient received supportive treatment (oxygenation through a non-rebreather mask) without any anticoagulant agents or corticosteroids. Throughout his hospitalization, he remained haemodynamically stable with no need for administration of intravenous fluids or vasopressors. Consequently, clinical improvement was noted with return of level of consciousness and communication to pre-surgery status and complete weaning of oxygen therapy. The patient was discharged after 10 days of hospitalization

Discussion

FES is considered to be a post-traumatic complication most commonly associated with long bone fractures or pelvic fractures. Fat embolism was first described by Zenker in 1861 [1, 7], who first diagnosed the presence of fat droplets in lung capillaries of a railroad worker that sustained crush injury and eventually passed away [7].

Aetiology of FES is mainly divided into traumatic and non-traumatic causes. Traumatic causes include orthopaedic conditions such as long bone or pelvic fractures, blunt force trauma, soft tissue injuries, burns, cardiopulmonary resuscitation compressions, etc. Although rare, non-traumatic conditions such as corticosteroid therapy, pancreatitis, haemoglobinopathies (sickle cell disease), liposuction, bone-marrow or lung transplantation and hip or knee arthroplasties are also associated with FES [1-4]. Although fat embolism is believed to occur to a greater or lesser extent in the majority of patients sustaining trauma, long bone or pelvic fractures and relevant arthroplasties, the clinical manifestation of fat embolism syndrome is variable [3, 4, 8, 9]. Existing bibliography consists mainly of case reports of FES whereas prospective or retrospective cohort studies are numbered. Indeed, there are several cases with FES presented in the literature with sustained long bone or pelvic fractures in major trauma centres, with bilateral fractures being associated with a higher incidence of FES

compared to unilateral long bone fractures [6, 7, 10]. According to published literature, 1%-5% of patients sustaining long bone fractures or undergoing orthopaedic procedures can present with FES [3, 7, 11, 12]. There are numbered case reports in the literature describing fat embolism after elective total hip or knee arthroplasty [8, 13-15].

Symptom manifestation is variable and can occur from the first hour's post-admission to as long as 2 weeks after initial trauma or surgery, although usually symptoms present within 24 to 72 hours after the causative incident [1, 3, 7]. In many cases, FES has an insidious onset with dyspnea, tachypnoea and hypoxemia which may progress to acute respiratory distress syndrome (ARDS). Fifty percent of patients with FES develop respiratory failure that requires mechanical ventilation [7]. Neurologic sequelae are usually subsequent to respiratory symptoms, may be present in 80% of patients and are non-specific [7]. They may include confusion or agitation as well as neurologic deficits, seizures or even coma. Finally, the petechial rash, which is considered pathognomonic for FES, is present in 20%-50% of cases and is typically located at the neck, thorax, axillae, subconjunctival space and oral mucous membranes [1, 3, 7]. Patients can also present with other non-specific signs and symptoms such as fever, tachycardia, jaundice, retinal changes, oliguria, or anuria. Death usually occurs due to respiratory failure or right heart failure [7].

Laboratory analysis may reveal anaemia, thrombocytopenia, elevated bilirubin, acute kidney injury as well as elevated inflammatory markers (CRP, ESR) [4, 7]. Radiographic imaging can be helpful, mainly in the sense of excluding alternative diagnoses. Chest radiography is usually normal [1]. In patients with respiratory compromise, it may reveal bilateral infiltrates (diffuse or patchy) mimicking ARDS, pulmonary oedema, or infection [4, 7]. CRX can be useful in monitoring progression of pulmonary infiltrates in patients with pulmonary FES manifestations [16]. Findings in computed tomography of the thorax most commonly consist of ground glass

infiltrates (either patchy, well-demarcated or diffused) which can be accompanied with interlobular septal thickening resulting in the so-called “crazy paving” pattern but, they can also include pulmonary consolidation and nodular opacities (usually centrilobular) [4, 7, 14, 16]. In patients with altered mental state, brain CT can reveal diffuse oedema with scattered haemorrhagic elements but in the majority of cases no abnormality is observed [4]. Further evaluation with MRI of the brain can be helpful for early diagnosis, especially when the “starfield pattern” is observed (multiple, small, nonconfluent, hyperintense lesions on T2 weighted images located around the ventricles, under the cortex or in the deep white matter) [4, 6, 7].

It is important to note that there is no gold standard diagnostic test for FES thus making its diagnosis one of exclusion [1]. As the diagnosis can often be elusive, a high degree of clinical suspicion is a necessary prerequisite parallel to the exclusion of more common entities such as septic syndrome or pulmonary embolism. In this context, diagnosis of FES is made using diagnostic tools such as certain sets of clinical and laboratory criteria, although none of them are clinically validated or universally accepted [1-4]. Most commonly, the Gurd and Wilson’s criteria are used [3, 9, 17].

Treatment is mainly supportive. Important aspects include oxygenation support (as far as mechanical ventilation, should that be deemed necessary) and haemodynamic stabilization through fluid resuscitation with or without the administration of blood products and vasopressors, as needed [4, 6]. Several protocols for targeted treatment (dextran, heparin, steroids) have been applied throughout the years but they have not demonstrated any benefit as far as outcome and morbidity is concerned [4, 6].

Depending on the severity of initial presentation, the outcome of patients with FES can vary from full recovery to death. Even though there is an important degree of variability in existing literature, FES is associated with high mortality with overall mortality rates

ranging from 5%-20% [7]. Older people seem to be more gravely affected with mortality rates as high as 17.6% in patients over 65 years old [18]. However, it is documented that rates of full recovery are high when patients receive appropriate supportive treatment [6]. This finding highlights the importance of clinical suspicion of FES, timely and appropriate differential work-up and administration of proper supportive care.

In recent years, there has been a significant decrease in the incidence of FES in trauma patients. The establishment of treatment protocols such as damage control orthopaedics and early total care led to early stabilization of fractures within the first few days post injury that reduced the incidence of FES in polytrauma patients [6, 7]. However, there are not many clinical cases described in the literature regarding the association between FES and arthroplasties. Interestingly, there are 5 clinical cases of patients developing symptoms of FES following elective arthroplasty described in the literature over a 20-year period [3, 19]. The definitive diagnosis was made using clinical criteria, head and chest CT and brain MRI [3, 19]. Modifications of surgical techniques such as cementless arthroplasties have not shown to have lower incidence of FES however, irrigation and aspiration of bone marrow contents could decrease the incidence of FES [3, 19].

There are two main theories described in the literature regarding the mechanism of FES. On one hand, the mechanical theory suggests that fat droplets from bone marrow or adipose tissue enter systemic circulation through torn venules after trauma or medullary preparation during surgery. In hip arthroplasty the insertion of the femoral stem causes mechanical compression of the medullary canal [5]. In addition, during knee arthroplasty, the use of long-stemmed components and intramedullary alignment guides also lead to an increase in intramedullary pressure [9, 14, 19, 20]. On the other hand, the biochemical theory proposes those toxic intermediaries of fat cause inflammation and release cytokines [7, 12].

Gurd and Wilson's criteria	
Major	Minor
<ul style="list-style-type: none"> ● Petechiae ● Hypoxemia ● Altered mental status 	<ul style="list-style-type: none"> ● Tachycardia >120 bpm ● Fever ● Retinal changes (fat or petechiae) ● Jaundice ● Anuria or oliguria ● Thrombocytopenia ● Anemia ● High erythrocyte sedimentation rate ● Fat macroglobulinemia
FES diagnosis	2 major or 1 major + 4 minor

Table 1. Gurd and Wilson's criteria for the diagnosis of Fat embolism syndrome [3]

Conclusion

We presented the case of a 68-year-old male with FES after elective total hip arthroplasty. Main symptoms comprised of mental status alteration and acute respiratory failure with symptom manifestation occurring a few hours post-operatively. Through clinical examination and laboratory and imaging analysis, alternate diagnoses were excluded. The patient received supportive therapy with oxygenation without any anticoagulant agents or corticosteroids. Clinical suspicion of FES in combination with timely and appropriate supportive treatment led to a full recovery and discharge of the patient after 10 days of hospitalization.

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