Dyspnoea after endo-medullary nailing: Fat embolism

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Abstract

Fat embolism is a potentially life-threatening condition that can develop after trauma or lower limb orthopaedic surgery. Characterized as a triad of respiratory, neurological and cutaneous symptoms. High index of suspicion is important. Rapid recognition and supportive treatment are key in improving the outcome. The prognosis is usually good.

DYSPNOEA AFTER ENDOMEDULLARY NAILING: FAT EMBOLISM

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BACKGROUND

Fat embolism is a well-known and life-threatening condition that can develop after long bone fractures and lower limb orthopaedic surgery. It presents in a wide range of respiratory, haematological, neurological and cutaneous symptoms and signs of varying severity, resulting from embolic showering. It is important for clinicians to have a high index of suspicion for fat embolism in patients with respiratory compromise postoperatively. Rapid recognition and supportive treatment are key in improving the outcome of these patients. The prognosis is usually good, except in fulminant fat embolism syndrome.

Key Words: Fat Embolism Syndrome – Trauma – Fracture – Diagnostic Criteria

Consent Statement:

Written informed consent was obtained from the patient to publish this report in accordance with the journal's consent policy.

Conflict of Interest:

None

Disclosures:

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CASE PRESENTATION

A 19-year-old male, without past medical history, was admitted to the emergency department 4 days after an endomedullary nailing of the right femur. He was hospitalized in another hospital after a crash with his motorcycle resulting in a right-sided mid-femoral fracture. An endomedullary nailing with an 340mm long nail was performed and intraoperatively no complications were reported. *(Figure 1)* There was an uncomplicated postoperative course and the patient could leave the hospital the day after the operation.

However, he presented at the emergency department with dyspnoea lasting for 24 hours. He had an important reduced tolerance of physical activity. At admission pulse oximetry revealed oxygen saturation of 89% on room air. On auscultation the chest was clear, his respiratory rate was 24 breaths per minute and he could speak in full sentences. He was agitated and a slightly confused. With three litres of oxygen the saturation was 97%.

Arterial blood gas analyses showed pH: 7.46 – pCO2: 32.2kPa – pO2: 57.3kPa – saturation 89%. Blood analyses revealed a CRP of 27 mg/L, white blood cell count was 15.47 x 10*9/L, normal kidney function, d-dimers of more than 4400ng/ml and elevated CK of 2760 U/L.

Echocardiography showed normal function of the left ventricle, normal valve function and normal contractility of the right ventricle without right ventricle overload.

A Computed Tomography (CT) pulmonary angiogram was performed because of a high suspicion of pulmonary embolism. The CT revealed no pulmonary embolism but demonstrated diffuse patchy ground glass appearance in the lobes of both lungs. The diagnosis of fat embolism was made.

(Figure 2)

The patient was hospitalized at the intensive care unit for observation. He made a good recovery with supportive treatment, which included controlled oxygen therapy and intravenous fluid resuscitation.

DISCUSSION

Dyspnoea in the postoperative period after orthopaedic surgery is not a rare occurrence and can vary in cause and severity. Fat embolism syndrome (FES) is one of the most frequent, but also one of the most overlooked causes of dyspnoea. It is a potential life-threatening complication of long bone fractures and orthopaedic reaming procedures. It is estimated to occur in 3-4% of patients with long bone or pelvic fractures.[1,2] The diagnosis of fat embolism syndrome is often missed because of a subclinical illness or coexisting distracting injuries or diseases. Other causes of dyspnoea after trauma are pulmonary contusions, shock lung or thromboembolism, but also cardiovascular and metabolic causes are possible. The terms fat embolism (FE) and fat embolism syndrome are not interchangeable. Fat embolism refers to the presence of circulating fat globules in the circulation and the pulmonary parenchyma. Fat embolism syndrome is the clinical manifestation of fat embolism. It usually presents as a triad of respiratory insufficiency, altered mental status and petechiae.

In 1861 Zenker reported the first case of fat embolism in an autopsy by describing fat droplets in the lung of a railroad worker who sustained fatal thoracoabdominal injuries.[1] It was only in 1865 that Wagner described the correlation of FE with fractures. The clinical fat embolism syndrome was first described in 1873 by Bergmann as a triad of confusion, dyspnoea and petechiae, following long bone fractures.[2]

It was not until the 1920s that the two main pathophysiologic theories were proposed. The first set of clinical criteria was presented by Gurd in 1970.

Epidemiology

The true incidence and mortality rate of FES is unclear, mainly because there are often concomitant injuries and pre-existing problems. Fat embolism and milder forms of fat embolism syndrome may stay clinically undetected. In retrospective studies the incidence of FES is less than 1%, in contrast to the incidence of 11%-29% in prospective reviews.[4-8] Differences in the diagnostic criteria and the over-diagnosis in prospective studies and under-diagnosis in retrospective studies are possible explanations for this varying incidence. The incidence of fat embolism in post mortem examinations rises up to 20%.[9] It is estimated that fat embolisms occur in more than 90% of patients with fractures of the long bones or multiple injuries, patients undergoing intramedullary reaming and nailing, and patients undergoing total hip arthroplasty. These estimates are based on both postmortem findings in trauma victims as well as prospective studies that used either transthoracic or transesophageal echocardiography to monitor the “showering” of micro emboli through the heart and lungs that took place during certain corrective orthopedic procedures.[10]

Etiology

Fat embolism syndrome develops most commonly after orthopaedic trauma and orthopaedic procedures such as intramedullary reaming and pelvic or knee arthroplasty. However, it has also been reported following other forms of trauma such as severe burns, liver injury, thoracic compressions, bone marrow transplants and liposuction. Non-traumatic causes are very uncommon.

Pathophysiology

The pathophysiology of fat embolisms is still controversial and many theories have been proposed. Why some patients develop fat embolism while others don't is not exactly understood and no single theory explains all the pathophysiological features of FES. The mechanical and biochemical theory are most accepted. The mechanical, or "infloating" theory, proposed by Gauss in 1924 states that as pressure increases in the intramedullary space, during trauma or surgical manipulation, yellow fat is forced out and enters the venous circulation through breaks in the vessel walls, where it can clump and form thrombotic masses.[12] Limitations of the mechanical theory are that is does not explain the 24 to 72 hour interval until symptoms occur and it does not explain nontraumatic FES. The biochemical theory, also known as the free fatty acid and lipase theory, was proposed by Lehmann in 1927. This theory suggests that an inflammatory cascade occurs when fat embolisms are degraded into free fatty acids, which are known to injure pneumocytes and capillaries resulting in pulmonary inflammation.[13] The combination of the mechanical and the biochemical theory, thus initiation of the symptoms by fat globules followed by the inflammatory cascade, is also described.

Risk factors

It is not clear why some patients develop fat embolism while others don't. Closed fractures, multiple fractures, conservative treatment for long bone fractures, reaming of endomedullary cavity, over-enthusiastic intra-medullary nailing, increased velocity of reaming are predisposing factors for FES.[11]

Altered intrinsic metabolic changes predispose patients to FES following FE, are yet to be defined.

An injury severity score of more than 16, femoral fracture, combination of extremity and abdominal trauma or abnormal vital signs at admission are independently risk factors for the development of ARDS (acute respiratory distress syndrome) due to FES. [11]

Clinical features

Most recent studies show that clinical signs and symptoms occur only in 1-10% of patients with fractures. Clinical presentation includes a wide range of symptoms and thus severity. A high level of suspicion should be taken into account when a patient presents with the classic triade of hypoxia, confusion/neurological abnormalities and petechial rash.[3] The clinical manifestations are preceded by an asymptomatic latent period of about 12–48h, but it can occur intraoperatively or as late as two weeks after the inciting event. Embolization begins rather slowly and attains a maximum in about 48hours. Most commonly the onset is gradual but sometimes it can be fulminant with pulmonary and systemic embolization, right ventricular heart failure and collapse.[3]

Most commonly and primarily involved is the respiratory system. Up to 75% of patients with FES present with some degree of respiratory failure, ranging from nearly asymptomatic hypoxemia to pulmonary distress requiring ventilatory support.[6] The most fulminant and lethal form of FES presents as acute cor pulmonale with respiratory failure within a few hours of injury. Usually, the lung recovers by the third day. Acute right heart failure is seen if the embolism occludes 80% of the pulmonary capillary meshwork.[5]

The central nervous system is the second most commonly involved system, usually in combination with pulmonary disturbances. The symptoms are highly variable, usually nonspecific and ranging from a simple headache to rigidity, disorientation, confusion, convulsion, stupor, and coma. These symptoms are usually non lateralizing, tend not to respond to O2 therapy but are transient and fully reversible.[6,8]Some propose that smaller globules may traverse the pulmonary microvasculature and reach the systemic circulation, leading to the common neurological manifestation of FES.[6,8]

In 50%–60% of patients a petechial non-blanching rash is present on the upper anterior area of the body, axillae, neck, upper arms, and shoulders.[6] It may also be present in the oral mucous membranes and conjunctivae. It has never been described on the back. The rash results from occlusion of the dermal capillaries by fat causing increased capillary fragility. It tends to be transient and disappears after 24 hours.[5,6]

An invariable cardiovascular sign of FES is tachycardia, but this does not often help with the diagnosis of FES since there are many causes of tachycardia in the trauma patient.

Retinal manifestations of FES are present in about 50% of patients, most of these findings disappear within a few weeks. They consist of cotton-wool exudates and small hemorrhages along the vessels and macula.[6,8]

Other less common and nonspecific manifestations are anemia, fever, myocardial depression or hypotension.[6]

Differential diagnosis

The list of differential diagnosis in a patient with signs and symptoms of FES is exhaustive. The clinical symptoms are vague and non-specific. In the differential diagnose we have to include pulmonary thromboembolism, pneumonia, acute respiratory distress, pulmonary oedema, heart failure and atypical infection.

Diagnosis

Diagnosis often follows a process of elimination. Diagnostic criteria for FES are wide in the literature but none is routinely used in practice.[15] Most accepted are the modified Gurd's criteria.[14] When using modified Gurd's criteria, the chance of underdiagnosis is greater, however fat droplets could be found in many patients in lab results without any clinical significance.

In 1983 Schonfeld proposed a clinical score. He assigned scores to seven clinical signs. A cumulative score >5 is required for a diagnosis of FES. [15] *(Table 1)* Several other scoring systems were still proposed but all these criteria are based on small series and none of them are validated on prospective studies.

Arterial blood gas analysis will show hypoxia along with the presence of hypocapnia. Thrombocytopenia, anaemia and hypofibrinogenemia are seen in FES but are all nonspecific findings. ECG is usually non-specific, but ECG changes can be detected if FES leads to myocardial necrosis.

On chest radiography bilateral pulmonary infiltrates, fleck-like pulmonary shadows (snowstorm appearance) are seen.

High-resolution Computed Tomography (CT) shows patchy ground glass opacities and consolidation with intralobular thickening. The extent of the CT findings is well correlated with the disease severity.[15]

POCUS (Point-of-care ultrasound) may aid to establish the diagnosis. Few recent case reports have described that transthoracic echocardiography (TTE) can detect fat emboli, seen as flowing hyperechoic particles in inferior vena cava.[16]

Brain CT is mostly normal or may reveal diffuse white matter petechial haemorrhages consistent with microvascular injury.

Bronchoalveolar lavage may also aid in the diagnosis, although fat in the lungs is nonspecific and can be seen in multiorgan failure and sepsis.

Treatment

The mainstay of treatment is supportive, including sufficient oxygenation and fluid resuscitation with maintaining good intravascular volume, as shock can exacerbate lung injury. Albumin has been recommended for volume resuscitation in addition to balanced electrolyte solution, because it not only restores blood volume but also binds with the fatty acids and may decrease the extent of lung injury. There are no drugs that have proved to give better outcome. Some data support the prophylactic administration of corticosteroids to patients with an elevated risk of FES, by decreasing the body's inflammatory response to the embolisms, thus supporting the biochemical theory.[17,18] There is no consensus about the dosage and duration of the

prophylactic corticosteroid therapy. Furthermore, there is no current evidence supporting the benefits of corticosteroids administered following a diagnosis of FES.[17,18] Recent experimental studies on the renin-angiotensin pathway are promising, angiotensin II acts as a vasoconstrictor but also proinflammatory and profibrotic. Patients in a state of alcoholic intoxication had less incidence of FES than sober ones, however there have been no prospective studies on use of alcohol as a drug for FES.[18]

Prevention

Because of the lack of treatment options, prevention is very important. The use of profylactic corticosteroïds is studied widely, but because of many different study protocols it is difficult to interpret. Routine prophylaxis is not recommended because the lack of high quality evidence, as well as potential risks of corticosteroid treatment and the low incidence of FES.[17,19]

Timing of surgery has been a point of interest for many decades. A randomized trial in 1989 compared patients with isolated femur fractures that were randomized to fixation either before 24 hours or after 48 hours. Significantly more pulmonary complications were reported with late intervention.[21] Recent literature supports data showing that timely fixation of fractures decreases the incidence of FES.[9,21]

Studies on the use of intramedullary nailing versus external fixation and reamed versus unreamed nails have been performed but were all underpowered and non-conclusive.[22] Early fracture stabilization, slow advancement of the intramedullary nail and technical revolutions like e.g., the reamer head are strategies to limit FE.[23]

Prognosis

Most cases are self-limiting, with full recovery of most patients, but mortality has been reported as high as 5-15%.[5,8]Patients with increased age, decreased physiologic reserves and comorbidities have obviously a higher chance of a negative outcome.[5,8]

Conclusion

Although rare, FES is a potentially fatal complication that can occur secondarily to trauma and certain orthopedic procedures. Identifying this syndrome remains a challenge, mainly because FES is a diagnosis of exclusion. Awareness of the key features, rapid recognition and supportive treatment are imperative in preventing poor outcomes, thereby decreasing mortality.

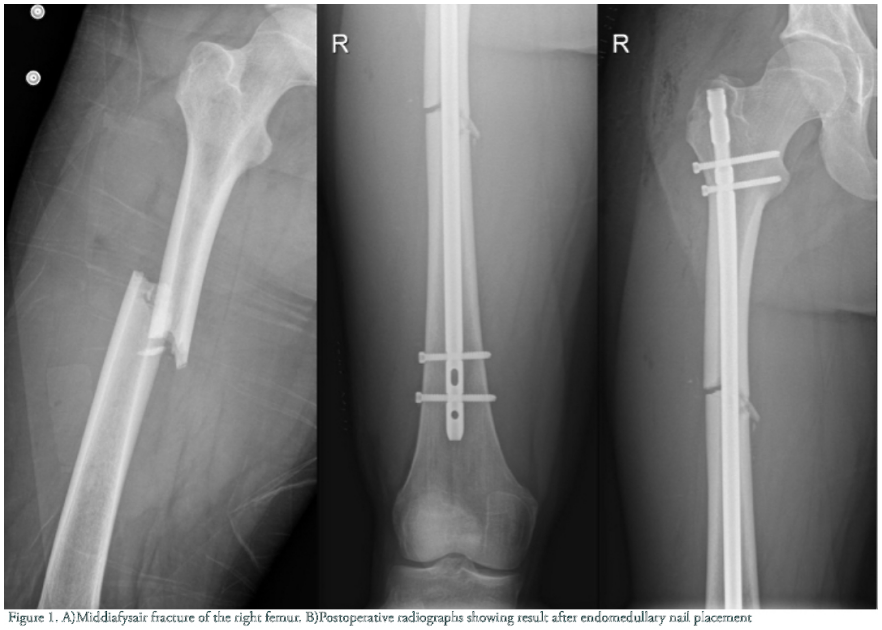

Figure 1. A)Middiafysair fracture of the right femur. B)Postoperative radiographs showing result after endomedullary nail placement

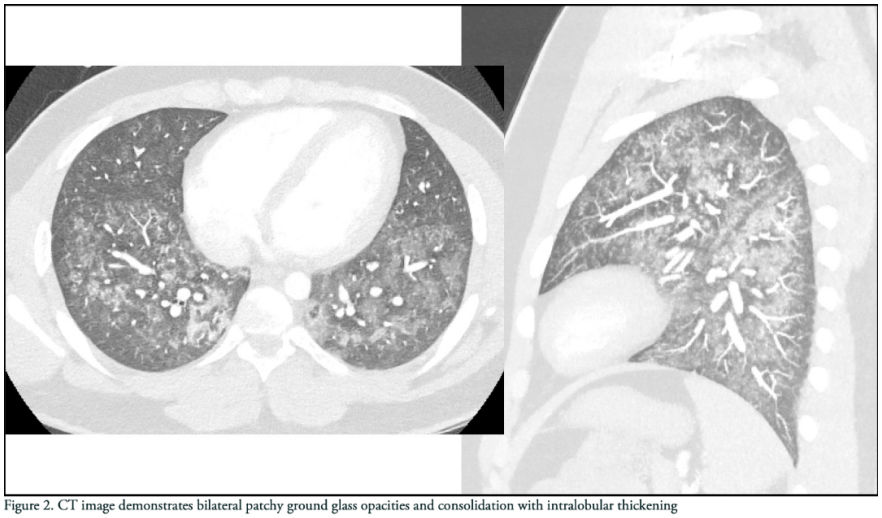

Figure 2. CT image demonstrates bilateral patchy ground glass opacities and consolidation with intralobular thickening

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Table 1- Diagnostic criteria for Fat Embolism Syndrome.pptx available at https://authorea.com/users/511823/articles/588530-dyspnoea-after-endo-medullary-nailing-fat-embolism