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Fat Embolism in Orthopedic Surgery

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Abstract

Every long bone fracture in orthopedic surgery represents a possible scenario for development of embolism complication, especially the fat embolism. There is no scientific explanation why fat embolism occurs and what are the hypotheses for development of fat embolism or the proper way of prevention, but just speculations and possible theories in the evolution of the clinical picture of fat embolism syndrome. Throughout this chapter, the authors will explain the possible theories of development of fat embolism, risk factors, pathology, and pathophysiology during progress of the clinical picture and signs of the fat embolism syndrome and therapy.

Keywords: fat embolism, orthopedic surgery, complications, fractures

1. Introduction

Every long bone fracture in orthopedic surgery represents a possible scenario for development of embolism complication, especially the fat embolism.

2. History

In 1861, fat embolism was first described by Zenker after a railroad accident and a worker who sustained crush syndrome injuries [1]. At the time when it was first described, it was believed that fat from the bone marrow, after a long bone fracture, embolized in the lungs causing pulmonary deficiency. On the other hand, Fenger and Salisbury believed that fat embolized from

fractures to the brain causing death [2]. Von Bergmann first clinically diagnosed fat embolism in a patient with a fractured femur in 1873 [3]. Fat embolism was thoroughly monitored and described during World Wars I and II. It was noted by Wong and his colleagues that patients with long bone fractures had a couple of desaturation episodes during the day with prolonged period of total desaturation [4].

3. Pathophysiology of fat embolism

The pathophysiologic mechanism of fat embolism is comprised of two theories—mechanical obstruction and biochemical injury. After a long bone fracture, fat emboli together with erythrocytes and thrombocytes can occlude pulmonary or brain blood vessels. The release of free fatty acids from fat causes local toxic effect on the endothelium, while the activation of platelets and granulocytes causes vascular incident.

Mechanical obstruction of the pulmonary blood vessels occurs because of the size of the embolized particles. In a dog model, Teng and colleagues found 80% of fat droplets to be between 20 and 40 μm , while vessels in the lungs that are smaller than 20 μm become obstructed [5]. Fat globules of 10–40 μm have been found after human trauma [6].

The biochemical theory suggests that mediators from the fracture site alter lipid solubility, causing coalescence, because normal chylomicrons are smaller than 1 μm . Many of the emboli have a histological composition consisting of a fatty center with platelets and fibrin adhered [7]. Large amounts of thromboplastin are liberated with the release of the bone marrow, leading to the activation of the coagulation cascade.

Peltier hypothesized that elevated serum lipase levels present after the embolization of neutral fat hydrolyzes this neutral fat to free fatty acids and causes local endothelial damage in the lungs and other tissues, resulting in FES [8]. This chemical phase might in part explain the latency period seen between the arrival of embolic fat and more severe lung dysfunction. Elevated serum lipase levels have been reported in association with clinically fatal FES [9, 10].

Mudd and colleagues did not find any myeloid tissue in any of the lungs at autopsy in patients with FES and suggested that the soft tissue injury, rather than fractures, was the primary cause of FES [11].

4. Incidence

The incidence of fat embolism is still not completely known. From the studies done on this topic so far, it happens in younger patients more often with lower limb fractures. Its incidence rises with the number of fractured long bones and severity of suffered injuries. Chan and associates found an incidence of 8.75% of overt FES in all fracture patients, with a mortality rate of 2.5% [12]. The incidence rose to 35% in patients with multiple fractures. Other investigators reported the incidence of FES between 0.9 and 3.5% in patients with long bone fractures [13–15].

5. Clinical presentation

Clinical presentation of the fat embolism syndrome starts with hypoxia, abnormalities in the neurological status, as well as development of petechiae that can be found in the region of the head, neck, and chest. Characteristic for development of petechiae is that they cannot be found in all patients, and some studies have shown their presentation in only 20–50% of cases. Also it is important to emphasize fever that follows the clinical presentation from the beginning. For the survival and adequate care of the patient, it is extremely important to recognize the development of fat embolism syndrome at the beginning. It is known that the development of this syndrome starts 1–2 days after the trauma in a period that can be referred to as a latent period. In some studies, the authors have concluded that fat embolism syndrome develops in the first 24 h (in around 60% of all cases), while in the rest of the traumatized patients, it can be recognized in the first 72 h.

For the proper understanding and better and faster diagnosis of fat embolism syndrome, Gurd and Wilson have developed a classification of the symptoms, where they have divided all symptoms in major or minor signs [16]. For diagnosing a fat embolism syndrome, it is necessary to notice one major and four minor signs that are shown in **Table 1**.

Nowadays, some authors in their studies have noticed the rigidity of the criteria mentioned above. In the study done by Lindeque and colleagues, development of the fat embolism syndrome was noticed with following signs: (1) pCO₂ of more than 55 mg Hg or pH of less than 7.3, (2) sustained respiratory rate of more than 35 breaths/min, and (3) dyspnea, tachycardia, and anxiety which they have suggested to be added in the criteria of Gurd and Wilson [17].

5.1. Major signs of FES

Hypoxia – even though pulmonary symptoms can be developed in a traumatized patient by the pulmonary embolism, heart failure, aspiration, or medication reaction, when none of the mentioned pathogenesis can be connected with patient’s clinical presentation, fat embolism syndrome must be suspected in differential diagnosis. For a patient, it is very important to be on an oxygen support from the time of admittance in a hospital.

Major signs	Minor signs
Hypoxemia	Tachycardia
Depression of the central nervous system	Fever
Petechiae	Fat in urine
	Fat in sputum
	Retinal emboli
	Decreased hematocrit
	Thrombocytopenia

Table 1. Major and minor signs of fat embolism syndrome.

Neurological status – in order to be monitored on a proper way, a full neurological status must be examined from the time of admittance of a patient. Every deviation in neurological status must be a suspicion on the development of FES. By the studies published so far on this topic, it is noted that in around 80% of patients with FES, some alterations in neurological status have been noticed. It is also of great importance to exclude the factors that can lead to changes in neurological status and that are not connected with FES (hypoxia, head trauma, etc.). Alterations of consciousness or seizures are considered as a bad prognostic sign.

Petechiae – as mentioned before, not all patients with developed fat embolism syndrome have petechiae as a major sign. They are presented in 20–50% of patients with FES, and their distribution can affect not only the head, neck, and anterior aspect of chest but also the axillar region, palate, and conjunctivae and are caused by embolic fat.

6. Treatment options and prevention

There is no strict protocol regarding possible prevention of fat embolism syndrome. It is considered that immobilization of long bone fracture, fast transport to the hospital unit, and immediate stabilization of the fracture can be ways to prevent the development of fat embolism syndrome. Also, fluid compensation and oxygen support from the moment of admittance into the hospital also reduce the risk of development of FES. It is known that oxygen support has value in prevention of FES.

In order to properly follow up the patient's condition regarding development of FES, it is recommended to daily monitor blood pressure, complete blood count, blood gas values, diuresis, and arterial oxygen on room air together with daily clinical examination.

From the moment of admittance into the hospital, increased fluid compensation (saline, Ringer solution, or hypertonic glucose) and low-molecular-weight heparin or acetylsalicylic acid are measures that possibly prevent the development of fat embolism syndrome [18].

Some studies have shown efficiency in preventing FES by giving large doses of corticosteroids immediately after the injury.

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