Fat Embolism in Differential Diagnosis of Acute Cor Pulmonale: Case Report

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Introduction

Fat embolism (FE) is defined as the presence of fat particles in the bloodstream, which have the potential to generate embolic phenomena and local tissue damage, especially in patients suffering from traumas and fractures of long bones. Fat embolism syndrome (FES) is characterized by the appearance of specific signs and symptoms secondary to the involvement of target organs such as lungs, brain and skin, in the presence of FE[1-10].

We discuss the case of a young female patient with fat embolism syndrome and acute cor pulmonale 48 hours after traffic accident with femur fracture.

Case Report

Female 22-year-old patient, white, previously healthy, using solely oral contraceptive, car accident victim with complete diaphyseal fracture of the left femur. At the emergency admission service, as per ATLS protocol of the polytrauma patient, no craniocerebral or chest trauma were evidenced and laboratory tests were normal. After 48 hours, still without surgical repair of the fracture, the patient presented sudden tachypnea, progressing to sinus tachycardia, dyspnea, psychomotor agitation and fall of oxygen saturation in pulse oximetry. On physical examination, the patient revealed heart rate of 145 beats per minute with regular rhythm, respiratory rate of 36 breaths per minute, blood pressure of 110 x 75 mmHg, 85% oxygen saturation with supplemental oxygen. Cardiac auscultation showed regular rate on two sounds with no identifiable murmurs, and lung auscultation, diffuse subcrepitant crackles in both lung fields. Electrocardiogram (Figure 1) revealed sinus tachycardia with signs of overload of the right heart chambers (clockwise rotation with early transition in the precordial leads with R in V2) and secondary repolarization abnormalities.

From these findings, we suggested the diagnosis of pulmonary thromboembolism (PTE), anticoagulation with unfractionated heparin in 10,000 IU bolus (stabilized fracture), laboratory tests, chest X-ray and TTE to search differential diagnoses. Chest X-ray (RX) now showed diffuse bilateral pulmonary alveolar infiltrate (Figure 2) and arterial blood gas analysis showed hypoxemia with PaO_2 of 45 mmHg. TTE showed findings consistent with acute cor pulmonale: dilation and right ventricular systolic dysfunction with right ventricular fractional area estimated at 24%, probable pulmonary hypertension - estimated pulmonary artery pressure of 40 mmHg, moderate tricuspid regurgitation and normal left ventricular function (Figure 3). Thirty minutes afterwards, chest CT angiography was performed, which showed no filling defect in pulmonary arterial branches, but showed multiple areas of opacity with frosted glass appearance on the edge of both upper lobes, lower lobes, lingula and middle lobe (Figure 2).

With this clinical and laboratory picture, the definitive diagnosis of EG leading to FES was reached. The patient was transferred to the intensive care unit, where she received supportive treatment, with no indication of heparin, and presented favorable evolution of the clinical picture, and was able to undergo surgical repair of the fracture ten days after the onset of symptoms.

Discussion

FES is a rare condition and is considered an exclusion diagnosis in suspected cases of pulmonary embolism[9]. It follows the fracture of long bones, especially the femur, tibia and pelvic bones, after 24-48 hours from the onset of the trauma, with an incidence of 0.5%-2% in these patients[2,7,9]. Other common causes of FES are orthopedic procedures, manipulation of the bone marrow, liposuction, burns and pancreatitis[10]. It is believed that the pathophysiological mechanism of the disease has two phases: firstly, mechanical obstruction of the pulmonary capillary arteries leading to progressive pulmonary hypertension; and, secondly, a biochemical phase, in which there is transformation of fat microparticles into fatty acids in the affected tissue, which probably activates chemotactic factors and local inflammatory response[1,4].

The most affected organs in FES are the lungs and heart (pulmonary hypertension, cor pulmonale and diffuse alveolar damage - SARA), skin (petechiae) and the brain (mental confusion)[5,6].

Some authors describe FES with two different presentations: "Acute fulminant", when the patients are affected by a high embolic load, usually post-traumatic, leading to high mortality rates; "Subacute", when the patient is in the first 16-36 hours

Keywords

Embolism fat/physiopathology; Pulmonary artery disease/physiopathology; Femoral fractures; Young adult.

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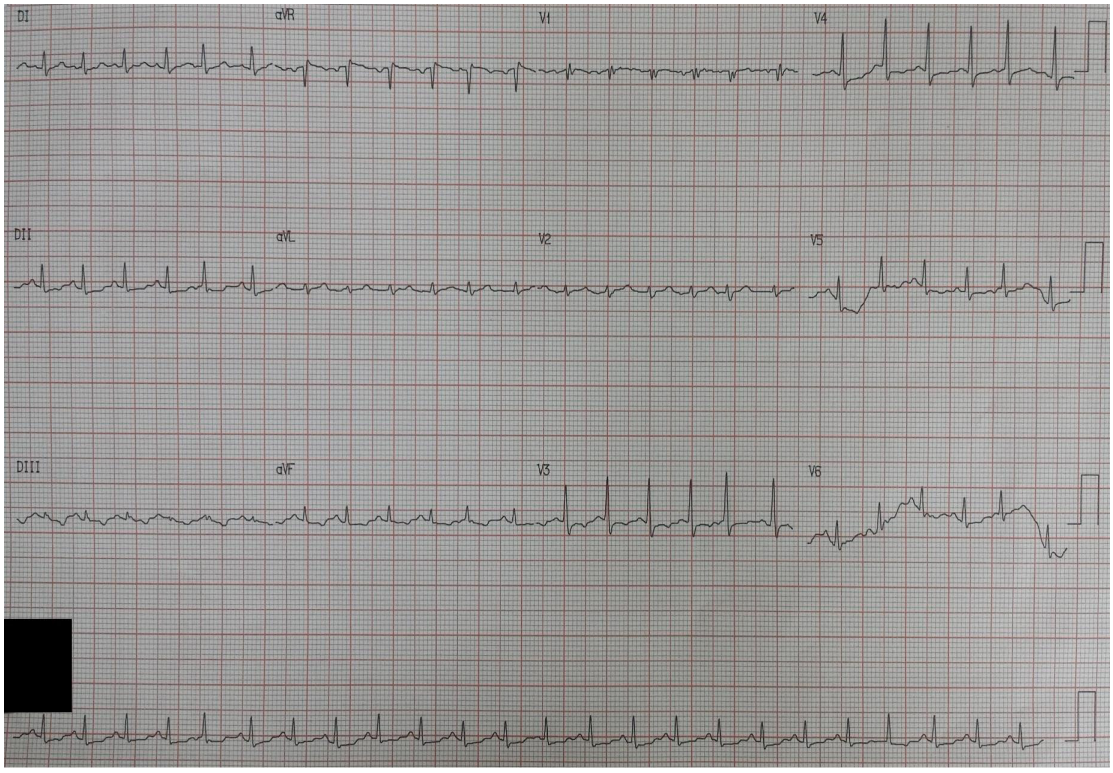

Figure 1 – *Sinus tachycardia with signs of overload of the right heart chambers (clockwise rotation with early transition in the precordial leads with R in V2) and secondary repolarization abnormalities.*

with progressive respiratory symptoms, sometimes with cutaneous and cognitive disorders[1,5,6].

Confirmation is based on clinical parameters[10]. Lindeque et al[9] developed diagnostic criteria in post-trauma patients with respiratory distress, in which the presence of one of the criteria makes the differential diagnosis of FES important (Table 1)[9].

Treatment of FES is still an issue of debate. It is known that early correction of fractures reduces the incidence of subsequent episodes, and the treatment recommended should be only a source of support[7,8]. More recent studies suggest that the use of corticosteroids has a beneficial effect on the clinical course of these patients. However, there is no concrete evidence of this[3]. The use of anticoagulation with heparin in FES is known to increase mortality, but it should be administered until the suspected PTE is fully ruled out[1,5].

The differential diagnosis of FE with PTE is often difficult. However, these conditions differ in some important points: the onset of symptoms in the PTE is delayed, usually from 72 hours of immobilization, while the manifestations of FES tend to emerge earlier from 24-48 hours[1,7,9,10]. In addition, in the PTE, there is mechanical obstruction of the pulmonary arteries on chest computed tomography angiography, justifying the pulmonary hypertension picture. In the FES, there is involvement of the lung parenchyma triggered by fatty microembolism of pulmonary capillary arteries and local inflammatory response.

During the clinical investigation, differential diagnoses of lung injury and SARA were ruled out, which have radiological patterns of pulmonary involvement similar to those found in FE, but with a distinct clinical history.

Conclusion

FE and FES are a diagnosis of exclusion. However, such conditions should be put next to the diagnosis of PTE in the context of a young patient, multiple trauma with long-bone fractures and sudden respiratory symptoms in less than 72 hours of immobilization, with findings of pulmonary hypertension on TTE without a mechanical obstruction confirmation of pulmonary vessels in computed tomography angiography. The clinical implication of this can be translated into delayed diagnosis time and even the maintenance of anticoagulation therapy, hence increasing mortality in patients with FES. FE treatment to date consists of hemodynamic and ventilatory support measures to the patient and surgical repair of fracture, if any, and the prognosis is directly related with the embolic load, with a mortality of about 20% in the subacute form and almost 100% in the fulminant form[1].

Case Report

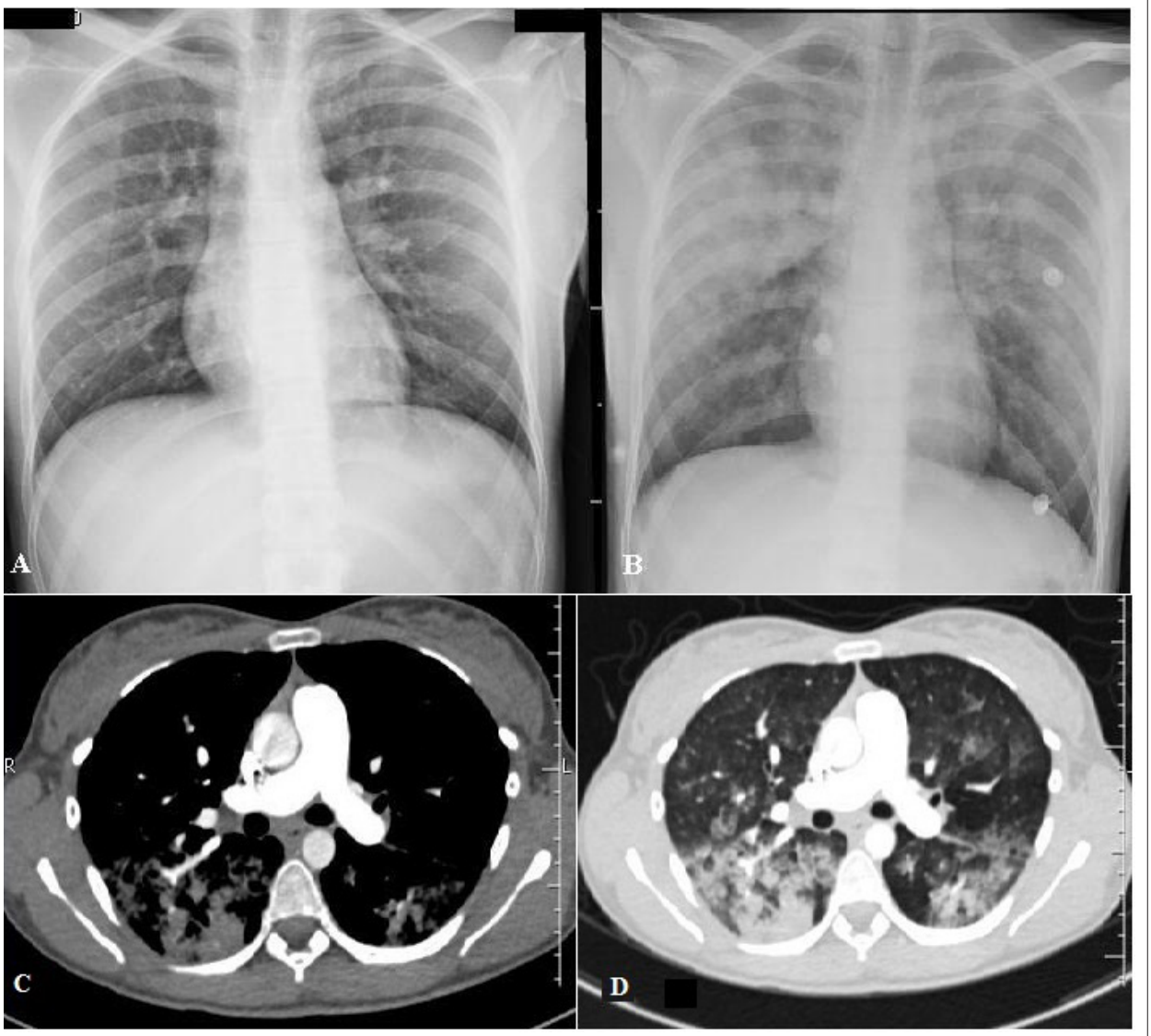

Figure 2 – *A) Chest X-ray on admission, normal. B) Radiography after 48 hours of evolution with the presence of diffuse alveolar infiltrate. C) Chest computed tomography angiography with no filling deficits by contrast in the pulmonary arteries and their main branches. D) Chest computed tomography angiography with diffuse alveolar infiltrate with a bilateral "ground glass" standard.*

Authors' contributions

Research creation and design: Pretto JLCS, Oliveira AC, Spilmann D, Marcon EDM, Almeida E, Bajesrski LM; Data acquisition: Pretto JLCS, Oliveira AC, Spilmann D, Marcon EDM, Almeida E, Bajesrski LM; Analysis and interpretation of data: Pretto JLCS, Oliveira AC, Spilmann D, Marcon EDM, Almeida E, Bajesrski LM; Manuscript drafting: Pretto JLCS, Oliveira AC, Spilmann D, Almeida E; Critical revision of the manuscript as for important intellectual content: Pretto JLCS, Oliveira AC, Spilmann D, Roman RM.

Potential Conflicts of Interest

No relevant potential conflicts of interest.

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Academic Association

This study is not associated with any graduate program.

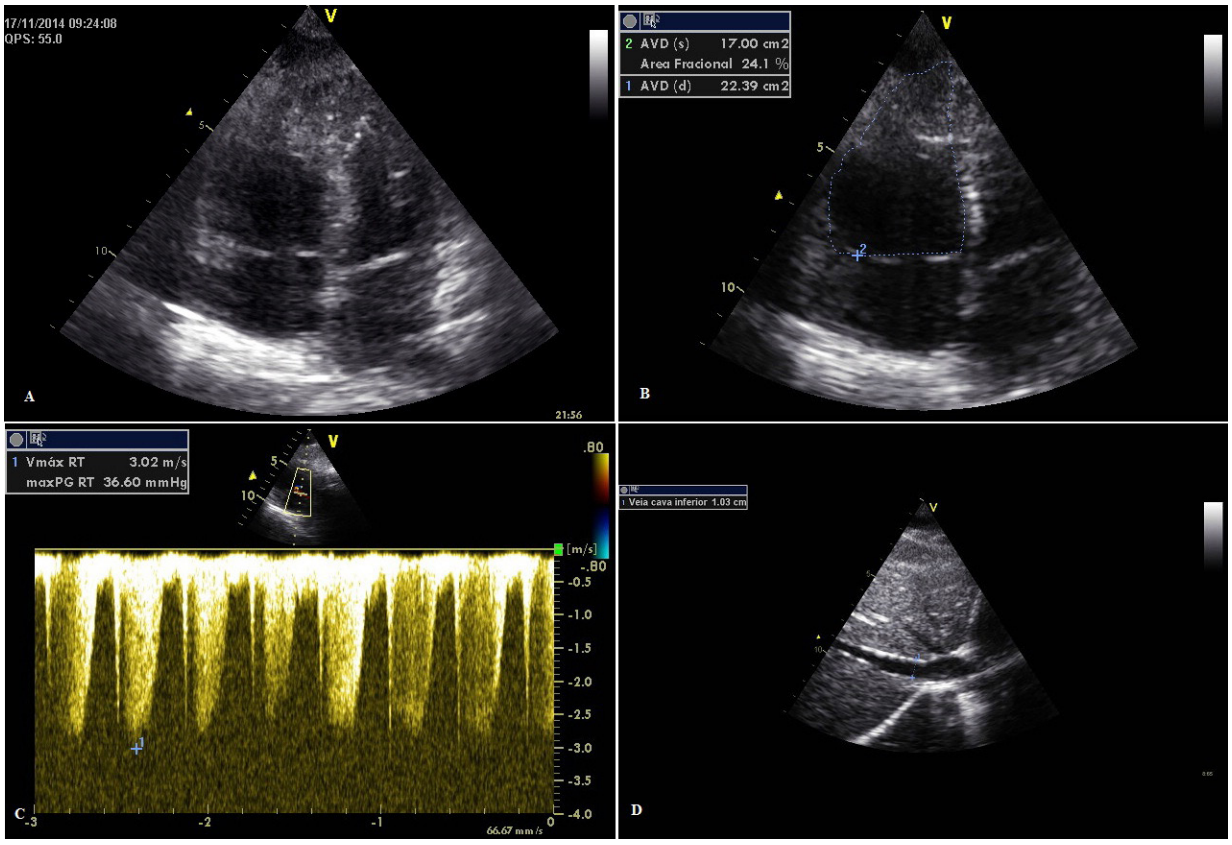

Figure 3 – *Transthoracic echocardiography: A) Increase of the right ventricle with normal left ventricle. B) Right ventricular systolic dysfunction with fractional area estimated at 24%. C) Tricuspid regurgitation to estimate the RV-RA gradient. D) Lower vena cava diameter to estimate right blood pressure.*

Table 1 – Criteria Proposed by Lindeque et al.

PaO_2 < 60 mmHg
$PaCO_2$ > 55 mmHg
Intense dyspnea: breathing rate > 35 rpm
Labored breathing requiring the use of accessory muscles

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