

Pulmonary Fat Embolism

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Section: Chest imaging

Imaging Technique: CT

Imaging Technique: MR

Case Type: Clinical Cases

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Patient: 28 years, male

Clinical History:

Polytrauma patient with acute respiratory distress

Imaging Findings:

Case: 28-years old polytrauma patient admitted with multiple fractures of the right tibia and talus, who developed 24 hours after the event a severe dyspnea and confusion. Legends: Fig 1: Admission chest film reveals no lung pathology. Orthopaedic surgery is recognizable by the presence of osteosynthesis material of T6 from a previous trauma. Fig 2: Chest film obtained three days later displays numerous confluent alveolar opacities which predominate in the middle and lower lung fields Fig 3A-B: 5mm CT sections reconstructed with a lung algorithm demonstrate posterior bilateral condensations and diffuse ground-glass patchy ill-defined opacities which slightly predominate in the right lung, highly suggestive of ARDS

Discussion:

Introduction: Fat embolism syndrom (FES) relates to the presence of fat droplets in the bloodstream and clinical symptoms including respiratory, neurological and cutaneous signs, which typically occur 24 to 72 hours after the event.(1-4) Clinical features: FES includes symptoms and paraclinical signs with two types of presentation.(1) The first type relates to a fulminating lethal presentation with pulmonary and systemic fat embolisms with secondary right ventricular failure and cardiovascular collapse.(1) The second type is the most frequent and is characterized by a gradual onset. It includes general symptoms, such as pyrexia and tachycardia, respiratory symptoms such as tachypnea, cough, hemoptysis and hypoxemia and neurological symptoms such as stupor, delirium, seizures and coma. Petechial rash is frequent and typically involves the oral mucous membranes, axillary folds, conjunctiva and retina.(1) The combination of respiratory, neurologic and cutaneous signs constitutes the FES.(1-4) Predisposition: The highest incidence of FES occurs in polytrauma patients with lower limbs and pelvic fractures.(1;3) However FES is also reported in many other conditions such as orthopedic procedures, bone marrow transplant, liposuction, massive hepatic necrosis, acute pancreatitis, acute sickle cell crisis, altitude illness, lipid or propofol infusion and total parenteral nutrition.(1) Pathophysiological mechanisms: Three main theories deal with the origin of fat in the FES but none of them is entirely satisfactory.(1;3) The first theory is mechanical. It relates to fat droplets of bone marrow which pass through lacerated bone medullary veins and disseminate in pulmonary and systemic bloodstream through a patent foramen ovale. This mechanism is enhanced by platelets or red blood cell aggregates.(1) The second theory has been experienced only in dogs. It relates to the plasticity of fat droplets which are wrapped by a marked elevation of the pulmonary arterial blood pressure, allowing them to migrate through the pulmonary capillary bed filter.(1) The third theory is biochemical. It relates to a stress trauma which induces a massive liberation of catecholamines which provoke the mobilisation of free fatty acids (FFA) from the body stock by the activation of the endothelial lipases. Similarly, it is emphasized that FFA mobilisation also results from bone

disruption after trauma. FFA exert a direct toxic action on vessels endothelium . This vascular damage induces activation of the complement cascade, a release of toxins from leukocytes and a platelet activation with further exaggerated endothelial injuries and microthrombosis resulting in multisystemic failure.(1) The second and the third theories appear seem more logical than the mechanical one to explain the presence of systemic fatty droplets embolism in patients with no patent foramen ovale. Radiological patterns: Chest radiograph findings are important for the assesement of the diagnosis of FES, although its appearance is variable and not specific.(2) The chest radiograph remains normal in minor cases. When fat embolism is extensive, a combination of numerous focal alveolar, interstitial and nodular opacities develops within 12-72 hours of latency.(2) These nodules may represent alveolar edema secondary to ARDS or early alveolar hemorrhage secondary to coagulopathy associated with FES(4) The shadows pattern is variable. They can be found in the peripheral lung zones or may predominate in the perihilar areas, simulating a batwing pressure pulmonary edema pattern.(2) CT sections demonstrate a typical ARDS pattern, characterized by focal or diffuse consolidation areas and ground-glass opacities, with small nodules of various sizes. Distribution of lung lesions is even without antero-posterior gradient.(2) Treatment: Treatment is symptomatic and supportive. (1;3)

Differential Diagnosis List: ARDS secondary to fat embolism

Final Diagnosis: ARDS secondary to fat embolism

References:

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Figure 1

a



Description: Admission chest film reveals no lung pathology. Orthopedic surgery is recognizable by the presence of osteosynthesis material of T6 of a previous trauma. **Origin:**

Figure 2

a



Description: Chest film obtained three days later displays numerous confluent alveolar opacities which predominate in the mid and lower lung fields **Origin:**

Figure 3

a



Description: 5mm CT sections reconstructed with a lung algorithm demonstrate posterior bilateral condensations and diffuse ground-glass patchy ill-defined opacities which slightly predominate in the right lung, highly suggestive of ARDS **Origin:**

b



Description: 5mm CT sections reconstructed with a lung algorithm demonstrate posterior bilateral condensations and diffuse ground-glass patchy ill-defined opacities which slightly predominate in the right lung, highly suggestive of ARDS **Origin:**