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Case Report

Fat Embolism with Negative CTPA Rare Presentation

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Abstract
Fat embolism is a rare clinical entity. the majority of cases represent microscopic embolism and are not detectable at CT pulmonary arteriography (CTPA).The diagnosis is largely clinical ,with the imaging studies supporting the clinical diagnosis. Here we present the case of a 76 year old male who presented with history of fall and developed sudden onset of shortness of breath, chest pain and hemoptysis. His clinical and investigative findings which shows high D dimer and urine fat globules presence with negative CTPA we came to diagnosis of fat embolism.
Keywords: *fat embolism, negative CTPA.*

Introduction

Fat embolism syndrome was first described in 1873 by Von Bergman[1] in patients with fracture of the femur. Whereas two decades earlier than this description of fat embolism syndrome, fat emboli were noted by Zenker[2] in a crush injury patient. CT pulmonary arteriography (CTPA) is the optimal modality for diagnosis of pulmonary embolism (PE). The majority of cases of fat embolism are due to microscopic fat post long bone fractures or orthopaedic surgery with implants. Microscopic fat embolism does not

result in a filling defect in the pulmonary arteries and diagnosis cannot be made on CTPA alone[1]. Macroscopic pulmonary fat embolism is a rare manifestation of fat embolism where macroscopic fat is present within the pulmonary arteries.

Case Report

A 76 year old patient non diabetic non hypertensive presented with complaints of shortness of breath which was sudden in onset gradually progressive in nature since three days.chest pain which was sudden in onset gradually progressive not relieved with rest since three days, hemoptysis since two days.

Patient had history of fall three days back

Upon physical examination patient was moderately built, pulse rate 67bpm,blood pressure 158/90mmhg,respiratory rate 18/min,spo2 90%on room air ,GCS 15/15,temperature 37c

Patient was alert, oriented and interactive.

Cardiovascular and respiratory examination was otherwise normal. There was no petechial rash.

Initial investigation were significant for D dimer 1130ng/ml, urine for fat globules present, x-ray of left foot AP and LATERAL view shows fracture of fifth metatarsal. CBP, LFT and RFT were normal.

Upon further investigations CT pulmonary angiogram was negative for pulmonary thromboembolism, multiple patchy areas of ground glass opacities in right and left lung lower lobe with few patchy areas of consolidation in left lung lower lobe

Few small soft tissue density nodules in bilateral upper lobes and left lower lobe

Fibrocalcific areas with adjacent fissural, pleural thickening in right lung upper lobe/likely sequelae of previous infection.

Mediastinal lymphadenopathy. Patient was started on enoxaparin 60 mg/sc/bd for one week, followed by capsule ecospirin75mg/po/od at bedtime for one month and patient was on follow up after one month.

CBP(COMPLETE BLOOD PICTURE)

Parameter	Result Values	Biological Reference Intervals	Method
Haemoglobin	: 8.1 gms%	13.5 - 17.5 gms%	SLS-Hemoglobin method
PCV	: 23.8 vol %	40 - 54 vol %	Calculated
RBC Count	: 2.97 mil/cumm	4.0 - 5.5 mil/cumm	DC detection
Total WBC Count	: 9,710 cumm	4000 - 11000 cumm	Flow cytometry
DIFFERENTIAL COUNT			
Neutrophils	: 70 %	40 - 75 %	Flowcytometry,microscop
Lymphocytes	: 20 %	20 - 45 %	Flowcytometry,microscop
Eosinophils	: 02 %	1 - 6 %	Flowcytometry,microscop
Monocytes	: 08 %	2 - 10 %	Flowcytometry,microscop
Basophils	: 00 %	0 - 1 %	Flowcytometry,microscop
PLATELET COUNT	: 2.02 Lakhs/cumm	1.5 - 4.5 Lakhs/cumm	DC detection,microscopy
PERIPHERAL BLOOD SMEAR			
RBC	: Normocytic / Hypochromic with anisocytosis		Microscopy
WBC	: Within normal limits		Microscopy
Platelets	: Adequate		Microscopy

*** End Of Report ***

Parameter	Result Values
Urine for fat globules	: Present

*** End Of Report ***

D-DIMER

Parameter	Result Values	Biological Reference Intervals
D Dimer	: 1130 ng/mL	< 550
Method	: Immunoassay	

Interpretation:
Elevated D-dimer levels are obsvered in all disease and conditions with increased cogulationactivation. e. Thromboembolic disease, DIC, acute aortic dissection, MI, malignant diseases,obstetric complications, ird trimester pregnancy, surgery and polytrauma. D-dimer testing is done due to exclude thromboembolic events suc t.

eference: Dacie & lewis practical hematology, 11th edition.
INNOVANCE D-dimer SIEMENS kit insert.

CT PULMONARY ANGIO

Technique
Serial axial sections of Chest were obtained from the arch of aorta to the diaphragm after administration of 90 cc of I.V contrast injected at the rate of 4 ml per second.

Findings
No evidence of filling defect noted in the main pulmonary artery, right & left pulmonary arteries and bilateral segmental & subsegmental pulmonary arteries.

Main pulmonary trunk (measures :24 mm) is normal in calibre and shows normal contrast opacification. No e/o filling defects.
Right pulmonary artery (measures :19 mm) is normal in calibre and shows normal contrast opacification. No e/o filling defects.
Left pulmonary artery (measures :20 mm) is normal in calibre and shows normal contrast opacification. No e/o filling defects.

Focal fibrotic areas with calcification noted in the right lung upper lobe posterior segment with adjacent fissural thickening and pleural thickening - Likely sequelae of previous infection (Koch's etiology).

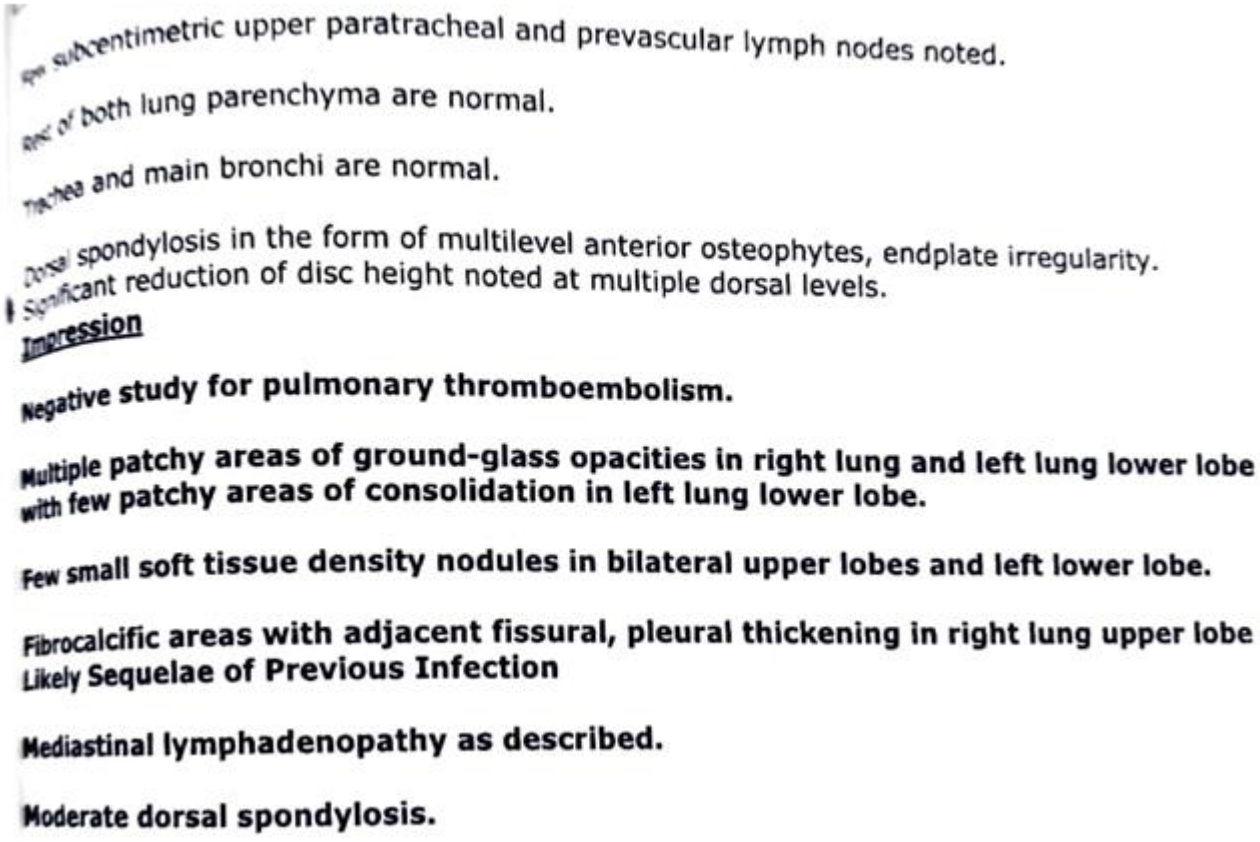
Multiple patchy areas of ground-glass opacities with intralobar septal thickening noted in right lung and left lung lower lobe predominantly in bilateral lower lobes

Few patchy areas of consolidation in left lung lower lobe.

Multiple small soft tissue density nodules noted in bilateral upper lobes and left lower lobe, largest measuring 6x5mm in left upper lobe.

Multiple enlarged lower paratracheal, precarinal, subcarinal and aortopulmonary lymphnodes noted, largest measuring 30x12mm .

Partially calcified bilateral hilar and right paraoesophageal lymphnodes noted.



Discussion

Fat embolism results from disruption of intramedullary fat following long bone fractures or orthopaedic surgery[(1)]. Pulmonary fat embolism (PFE)is the presence of fat globules in the pulmonary circulation. Although the fat emboli can reach the vessels of virtually any organs, the organs mainly affected are lungs, brain and skin which correspond to the classic trait of Fat Embolism Syndrome (FES): dyspnea, neurological abnormalities and petechial rash. Fat embolism syndrome which is a clinical diagnosis based on hypoxia, confusion, and a petechial rash, and can progress to adult respiratory distress syndrome (ARDS). Microscopic fat embolism can be detected in the blood and urine of almost all patients with long bone fractures, but only a minority of these develop clinical symptoms. Pathophysiology appears to relate to a combination of mechanical obstruction due to fat particles obstructing capillaries with ventilation perfusion mismatch and fatty acid induced inflammatory damage to alveoli with ARDS[(1)]. For diagnosis of fat embolism there is no universal criteria, various criteria were proposed by different authors.

According to Gurd's *et al.*[(6)], diagnosis of FES need at least two major criteria or one major and four minor criteria to be present in order to diagnose FES [Table 1].

Table 1 Gurd's Criteria

Major criteria	Petechial rash
	Respiratory insufficiency
	Cerebral involvement
Minor criteria	Fever
	Retinal changes
	Jaundice
	Renal signs
	Thrombocytopenia
	Anaemia
	High ESR
	Fat macroglobinneamia

Diagnosis of pulmonary fat embolism by CT visualization of macroscopic fat in the pulmonary vasculature is rare[2,3].

Pulmonary fat embolism was confidently diagnosed due to CTPA demonstration of fat attenuation filling defects in the pulmonary arteries. Fat typically has an attenuation value of -50 HU to -150 HU, and can be distinguished from both acute and chronic pulmonary thromboembolism which have a mean attenuation value of 33 HU (95% confidence intervals 26, 41 HU) and 87 HU (95% confidence intervals 66, 107 HU) respectively[7].

Chest radiograph is generally the first imaging test in patients with respiratory distress but findings of fat embolism are nonspecific and indistinguishable from pulmonary oedema, infection or aspiration. CT lung parenchymal findings in fat embolism are also generally nonspecific and include patchy ground glass opacities with consolidation and small centrilobular nodules.[4,5]

CTPA is the test of choice to diagnose pulmonary embolism and in contrast to ventilation perfusion scintigraphy allows direct visualization of embolic material permitting distinction of thromboembolism and fat embolism.

But in our case CTPA was negative and fat embolism can be diagnosed with clinical symptoms and high d dimer values and presence of fat globules in urine.

In cases of isolated fat embolism, routine use of heparin is not recommended due to lack of evidence, risk of haemorrhage and the potential, theoretical risk of exacerbation due to production of free fatty acids. Diagnosis of PFE over thrombotic pulmonary embolism may avoid the risks of prolonged treatment with anti-coagulation. This is particularly important in postoperative patients and patients with severe trauma due to the risk of hemorrhage as a complication of treatment. Treatment of isolated fat embolism is predominantly supportive with the majority of patients recovering in 1-2 weeks .

Fat embolism is a rare clinical manifestation as shown in our case. This case was a clinical conundrum as our patient presented with acute SOB with positive D-dimers, and without skin petechiae. But fat embolism also can rarely present with this symptoms. Even though it is not very well identified in fat embolism, it can appear secondary to acute bronchospasms. So high clinical suspicion should be there to diagnose fat embolism when respiratory distress occurs in a

day or more after major trauma or orthopedic surgery to focus on the management of FES.

Conclusion

Without specific tests and validated clinical criteria, diagnosis of FES is challenging. One should have a high degree of suspicion despite small fractures not usually associated with FES. Although most patients recover fully, if not timely intervened mortality rates are high. Early diagnosis and treatment of symptoms are of paramount importance for a successful outcome.

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