http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v8i9.10

IGM Publication Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Eaton Embolism - Coexistent Pneumonia and Pulmonary Embolism

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Abstract
The coexistence of pulmonary embolism (PE) and pneumonia is known as infarction pneumonia. This condition is relatively rare in adults in comparison to children. The diagnosis of coexistent pneumonia with embolism is challenging as clinical features of fever, pleuritic chest pain and breathlessness maybe common in both conditions. Hence, the presence of coexisting embolism in pneumonia needs a high degree of clinical suspicion .We hereby report a case of a young woman who presented with Mycoplasma pneumonia and ARDS with early diagnosis of concommittant pulmonary embolism. Early recognition and management of both the conditions resulted in a favourable outcome.
Keywords: *Pulmonary embolism, Mycoplasma, Pneumonia, Infarction.*

Introduction

Mycoplasma pneumoniae infections are one of the common etiologies of community-acquired pneumonia (CAP). The Manifestations and clinical features vary widely and systemic involvement is common. Diagnosis is very challenging because the clinico radiological findings of the classical pneumonia is lacking. Moreover, Mycoplasma pneumonia is not routinely tested in the clinical setup. Extrapulmonary manifestations like Arthralgia, pancreatitis, transverse myelitis and severe pulmonary manifestations can lead to long-term sequelae. The treatment becomes more complicated with the increasing emergence of *Mycoplasma* resistance. The current case highlights coexistence of mycoplasma pneumonia and pulmonary embolism and need for early diagnosis and management and can be fatal if not diagnosed and treated early.

History

A 27 year old female, IT professional by occupation came with complaints of high grade fever, cough with mucopurulent expectoration and progressively worsening breathlessness for 1 week. She denied h/o Chest pain /hemoptysis/ wheezing .There was no prior history of any respiratory illness.. No comorbid illnesses. No significant Family history.

Clinical Examination revealed the patient was conscious, oriented, febrile, dyspnoeic and Tachypneic at rest. Patient was not anemic, non icteric, no Clubbing/ cyanosis/ pedal edema. JVP was not elevated.

Vitals showed Spo2-93% in room air, PR-145 beats/min, RR-22 cycles/minute, BP- 110/70 mmHg, BMI-22 kg/m

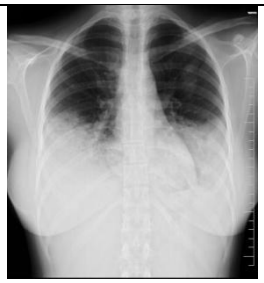
RS-Normal vesicular breath sounds with reduced intensity in right and left infraaxillary and infrascapular areas with bilateral fine basal crepitations

CVS- S1 S2 heard, no added sounds. Other systems normal.

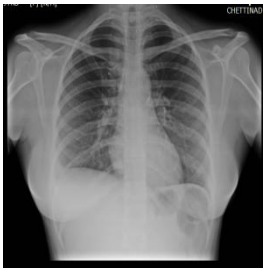
Course in the Hospital: Patient came with above said complaints, in view of desaturation, patient was shifted to ICU and following investigations were done.

Investigations

Routine Blood tests-:Hb – 8.1 gm%
TC, DC, RBS, LFT, RFT, HIV - Normal
Sputum for AFB – Negative
Mycoplasma IgM Antibody -positive
ANA- positive, Coomb's test- positive
ABG- PaO_2-78mmHg, $PaCO_2$-43mmHg,Ph-7.38
CXR PA(pre- treatment) -Bilateral non homogenous opacity in the right and left lower zones with obliteration of both costophrenic angles and cardiophrenic angles.
CECT Chest- Consolidation of bilateral lower lobes with atelectasis and bilateral pleural effusion
CTPA- Thrombus involving segmental and subsegmental branches of posterobasal segment of Right pulmonaryartery. Partial thrombus involving lateral basal segmental branches of Left pulmonary artery
F.O.B -Complete narrowing of left upper lobe bronchus with inflammed mucosa & mucoid secretions
(BAL for AFB & gene expert - negative) biopsy shows no granuloma and malignancy
PFT - Small airway obstruction.

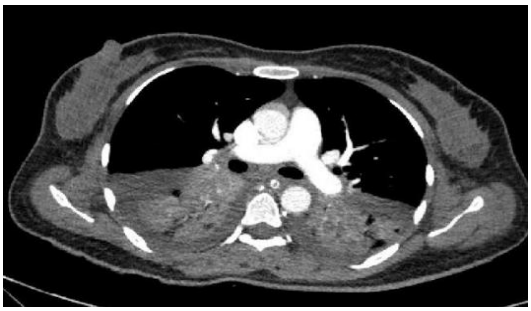


PRE- Treatment xray



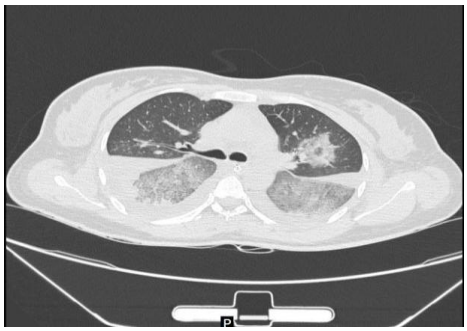
POST-Treatment xray

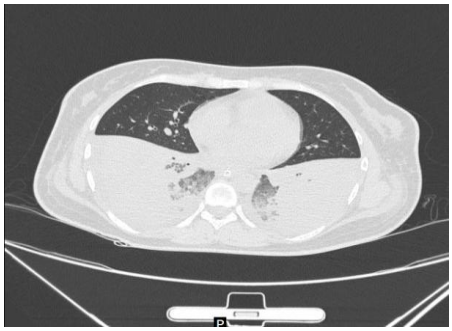
CTPA



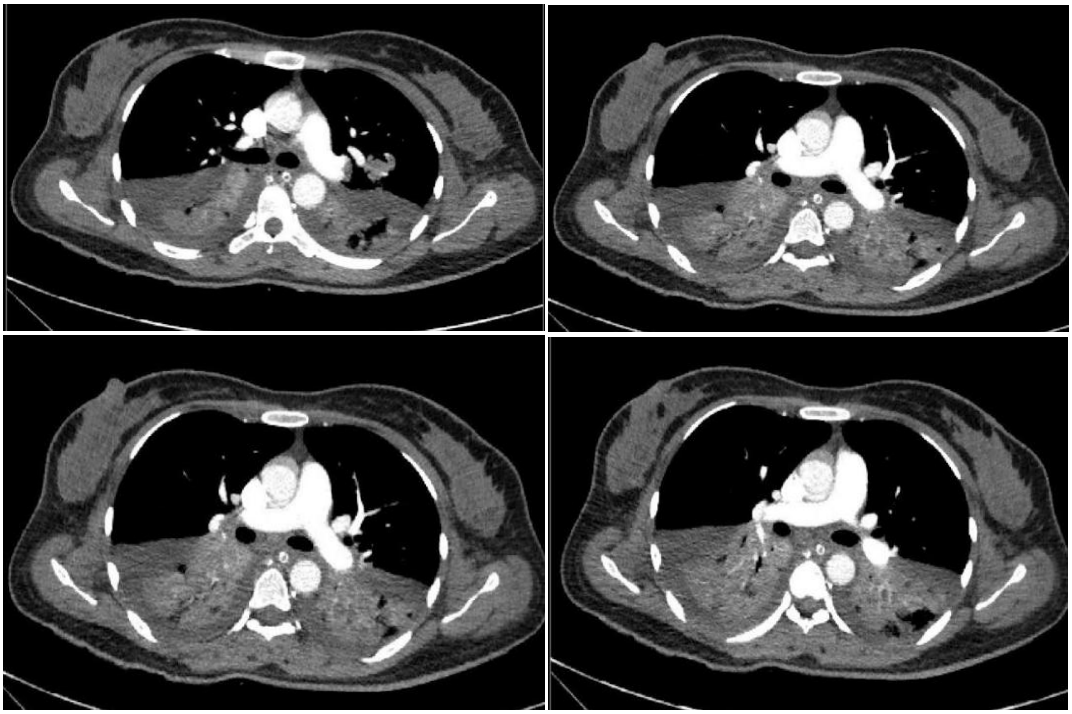
Results & Treatment

CECT Chest





CTPA



Treatment

Patient clinically presented with signs and symptoms of pneumonia and ARDS and was initially started on broad spectrum antibiotic. In view of worsening hypoxemia, patient intubated following a trial with NIV. Since patient had persistent tachycardia and pleural based lesion, pulmonary embolism was suspected and CTPA was done and revealed thrombus involving segmental and subsegmental branches of posterobasal segment of Right pulmonaryartery. Partial thrombus involving lateral basal segmental branches of Left pulmonary artery. All cultures were negative but Mycoplasma IgM antibody was positive, Patient was started on anticoagulants and Clarithromycin. Patient significantly improved with treatment and discharged with oral anticoagulants and Antibiotics.

Discussion

Mycoplasma pneumonia accounts for 7-20% of community acquired pneumonia. Community acquired pneumonia causing pulmonary embolism is known as infarction pneumonia which is the most important cause for mortality. It can cause hemophagocytic syndrome which leads to disruption of vessel wall integrity resulting in pulmonary embolism and ARDS. The probable pathophysiology postulated in our case is Mycoplasma vasculitis leading to pulmonary embolism.

Pathogenesis can be either direct or indirect type. The mechanism of direct pathogenesis is that mycoplasma pneumoniae blood-borne metastasis may induce cytokines such as tumor necrosis factor-α, chemotactic factor and interleukin-8 which affect vessel wall and lead to vascular occlusion or local angiitis. The indirect pathogenesis is that mycoplasma pneumoniae causes immunologic derangement which produces phospholipids and IgM anticardiolipin antibodies to form a temporary hypercoagulable state, leading to deep vein thrombosis. These findings reflect a systemic infection-related prothrombotic states. The clinical features for infection is chills, purulent sputum, or bacteremia, whereas pulmonary embolism is confirmed by CTPA. Therefore, the possibility of pulmonary infarction should be considered when there is continued clinical and radiological worsening in case of pneumonia despite treatment with antibiotics and optimal management.

Conclusion

This case highlights the importance of suspecting pulmonary embolism in pneumonias with atypical presentation not responding to standard care of management. A high clinical suspicion and early diagnosis of coexisting pulmonary embolism prevents complications and mortality.

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