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Pulmonary infarct masquerading as community acquired pneumonia in the COVID - 19 scenario: A case report

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Abstract

BACKGROUND

Pulmonary embolism (PE) requires a high degree of clinical suspicion for the diagnosis and can mimic pneumonia due to its clinical, radiological and laboratory findings. Co-existence of both PE and pneumonia can also occur which is surprisingly commoner than appreciated.

CASE SUMMARY

Here we report a case of young male, who initially presented during peak of Covid-19 pandemic with features of pneumonia, kept under observation and was later diagnosed and treated for a main pulmonary artery embolism without any identifiable source of thrombosis.

CONCLUSION

Pulmonary embolism and pneumonia share common clinical, radiological, and laboratory finding that may delay the diagnosis of pulmonary embolism.Hypoxia disproportionate to the extent of radiological involvement could be an indicator of an underlying pulmonary embolism.

INTRODUCTION

Pulmonary embolism mimic with pneumonia because of the considerable overlap in their clinical and radiological picture. Moreover, pneumonia may occasionally mask PE, particularly in patients with predominant systemic symptoms such as fever, and with no evidence of deep vein thrombosis (DVT) or trauma.Here we discuss a case of young male presented with during peak covid-19 pandemic with initial signs and symptoms of community acquired pneumonia , and on further evaluation due to clinical worsening showed extensive pulmonary embolism.

CASE PRESENTATION

Chief complaints

A 26-year-old healthy male presented with fever,cough,progressive exertional dyspnoea and vomiting for 5 days.

History of present illness

+ADw-html+AD4APA-p+AD4-Symptoms started as high grade fever with temperature recording of 101F without rigor or chills, cough noted from next day ,it was with scanty mucoid sputum ,no history of hemoptysis or copious purulent sputum .Dyspnea progressed slowly over 5 days to MMRC grade 2 .There was no orthopnea or paroxysmal nocturnal dyspnea or angina symptoms associated .He also had vomiting 2 -3 episodes per day after the fever , vomiting was non bilious and there was no associated abdominal pain or loose stool or blood in stool .No history of any leg pain , calf swelling or any prolonged immobilization .+ADw-/p+AD4APA-/html+AD4-

History of past illness

He had no other comorbidity. No history previous recurrent respiratory infection , asthma , or frequent hospitalizations.There was no history of recent travel outside his native place.

Personal and family history

He was non smoker and engaged in painting job during last 2 years.

Physical examination

On examination patient was febrile with temperature of 100F , tachypneic with respiratory rate of 36/minute, pulse rate 112/minute, blood pressure 110/70 mmHg and oxygen saturation of 85% at room air. Other Respiratory, cardiovascular, neurological and abdominal examinations were normal.

Laboratory examinations

His hemogram showed a total white blood cells count of 23090 [neutrophil 88%], and platelet count of 3.3 Lakh/cmm, blood urea and creatinine were 43mg/dL and 1.5 mg/dL respectively. Liver function test was normal. Electrocardiogram showed sinus tachycardia.

Imaging examinations

Chest radiograph showed left upper zone non-homogenous opacity with air bronchogram and right lower zone opacity obliterating hemi diaphragm silhouette [Figure -1].

A repeat chest radiograph documented resolution of left upper and right lower zones opacities [figure- 2].

CT Pulmonary angiography (CTPA) was done. CTPA revealed thrombus completely occluding right main pulmonary artery just after bifurcation, hypodense filling defect of 2.8×2 cm in right atrium suggestive of thrombus [Figure 3].

FINAL DIAGNOSIS

Pulmonary Embolism ,Hypoxic respiratory failure , Shock due to massive Pulmonary embolism .

TREATMENT

Considering the current pandemic, he was initially treated as a COVID-19 suspect and nasopharyngeal swab test was performed. Empirically antibiotics (intravenous Co-amoxyclav and oral azithromycin) were initiated along with high flow oxygen at 10 L/min and enoxaparin 40 mg subcutaneously once daily. His nasopharyngeal swab RTPCR for SARS Co2 was reported negative and he was transferred to pulmonary ward for further management. Sputum and blood culture were sterile. Sputum for XpertMTB was negative. Gradually, patient improved clinically and by day four his fever subsided, leukocytosis decreased and oxygen requirement reduced to 4 L/min. On fifth day of admission, he had new onset hemoptysis of minimal quantity. A repeat chest radiograph documented resolution of left upper and right lower zones opacities [figure- 2]. Repeat electrocardiogram showed sinus tachycardia and T wave inversion in V1 to V3. However, Troponin I was negative.

In view of persisting hypoxia, radiological opacity and higher Wells score, CT Pulmonary angiography (CTPA) was done. CTPA revealed thrombus completely occluding right main pulmonary artery just after bifurcation, hypodense filling defect of 2.8×2 cm in right atrium suggestive of thrombus [Figure 3]. Deep vein screening and compression ultrasound was normal. Echo cardiography revealed dilatation of right atria and right ventricle with increased right ventricular systolic pressure. Pulmonary embolism was confirmed, as patient was hemodynamically stable hence Injection Enoxaparin 40 mg subcutaneous twice daily was Initiated. Antibiotic escalated to piperacillin tazobactam 4.5 gm Q 6 hly and linezolid 600 mg twice daily in view of pulmonary opacity and elevated total white cell count of 21180/cmm. On fourth day of anti-coagulation, patient had increasing tachypnea and oxygen requirement increased from 4 Lit to 10 Liter per minute. Over the next six hours patient dyspnoea increased

further and intubated and mechanically ventilated due to severe hypoxia despite high flow oxygen and shifted to intensive care unit. Patient was hemodynamically stable except sinus tachycardia initially after 12 h patient developed hypotension requiring fluid resuscitation and noradrenaline support, patient was immediately considered for thrombolysis after counseling the benefits and risks of thrombolysis, due to financial constrain of patient's family was only afford for streptokinase. Injection streptokinase 2.5 Lakh loading dose followed by one lakh per hour was started. After 3 h of thrombolysis there was progressive worsening of hypotension and patient developed pulmonary edema and refractory shock and succumbed to death on 14th day of admission.

OUTCOME AND FOLLOW-UP

Succumbed to death on day 14th of admission.

DISCUSSION

In the present case a young male admitted with presumptive diagnosis COVID-19 pneumonia, was later diagnosed as pulmonary embolism. We missed the diagnosis of PE initially in the context of rising COVID-19 cases and time elapsed while waiting for RT-PCR result. Initial clinical diagnosis was non-COVID pneumonia due to presence of fever, elevated TLC and initial clinical improvement with antibiotic. There was possibility of concomitant PE and pneumonia also. Pneumonia is one the illness due to which PE often misdiagnosed due to similar clinical presentations. Few retrospective studies have already discussed about the patients with pneumonia have about 2–3-fold increased risk of venous thrombosis [1].

Due to lower sensitivity of RT-PCR for COVID-19, our clinical suspicion continued due to prevailing trend of ever rising COVID pandemic [2]. Even fever, leukocytosis, elevated CRP, procalcitonin which commonly used to differentiate PE from pneumonia is often exist in both conditions [3]. In one study 80% of angiographically proven PE reported leucocytosis and two third reported fever [4]. Initial radiological work up like

chest radiograph may often be helpful for diagnosing pneumonia, but data from ICOPER study including 2000 PE patient reported normal chest radiograph in one quarter of patient and patchy infiltrated were present in 17% patients [5]. Radiographic findings of PE have been extensively documented but are frequently nonspecific, it can present as normal findings, focal oligemic, pleural effusion, variable zones of atelectasis, pulmonary consolidations, large hilar vessels, elevated hemi diaphragm [6]. Airspace consolidation may result from infarction and actual necrosis of the lung tissue but more commonly occurs from "incomplete infarction" and haemorrhage into the air spaces and size of an infarct can vary from barely perceptible up to 10 cm in diameter [7]. Elevated D-dimer with PE probability scoring from the first day of treatment may help to take decision regarding CTPA. But D-dimer can also be elevated in pneumonia and sepsis along with another inflammatory marker. This elevated D-dimer associated with ICU admission and increased 30-day mortality [8].ECG changes like right ventricular strain, S1Q3T3 pattern, right bundle branch block, atrial fibrillation also can see in community acquired pneumonia [9].

In this case in the initial clinical evaluation there was no sign of DVT and the first day Well's score was only 1.5 (low probability for PE). Bed side echocardiography is usually done and

helps to detect early signs of PE like enlarged RV size with reduced functionality, abnormal septal wall movement, and tricuspid regurgitation. RV thrombi can be visualised in echocardiography similar to our case and usually signify a poor prognosis [10]. A timely CT pulmonary angiography will help to confirm the diagnosis. Secondly there are large scale autopsy of 1455 cases demonstrated concomitant presentation of PE and pneumonia, and out of 54 patients identified with anatomically major PE at autopsy, only 30 % had correct ante mortem diagnosis. However, their accuracy was more in postoperative patients. In 80% of lung scanning and pulmonary angiography associated with an increased tendency to correct clinical diagnosis of PE. Among 21 patients with autopsy-proved major pulmonary embolism who also had pneumonia and no pulmonary embolism was diagnosed before death, which indicate the

importance of this discussion [11]. As the patient had some clinical and radiological improvement with antibiotics, the diagnosis of PE was delayed in this case. Hypoxia that is out of proportion to clinico-radiological assessment is the key for early suspicion for PE as this case.

CONCLUSION

Pulmonary embolism and pneumonia share common clinical, radiological, and laboratory finding that may delay the diagnosis of pulmonary embolism. Our case suggests that persisting hypoxia even after clinical and radiological improvement even during peak of the COVID pandemic and hypoxia disproportionate to the extent of radiological involvement could be an indicator of an underlying pulmonary embolism.

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