

**Case Report** 

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# Lung Bullae, Cavitation and Right Ventricular Thrombus Formation with Saprophytic Aspergillus Colonisation: A Rare Presentation of Idiopathic Pulmonary Embolism

Soumik Ghosh\*, Prabhat Kumar, Pushpa Kumari, Varun Rehani, Ratnakar Sahoo and Adesh Kumar Gadpayle PGIMER, Dr.Ram Manohar Lohia Hospital, India

#### Abstract

Pulmonary Embolus (PE), in association with Deep Vein Thrombosis (DVT) is one of the three major cardiovascular causes of death. There are several risk factors for PE but still many cases are idiopathic. Cavity, bullae and lung abscess formation are unusual finding in PE. Right Ventricular (RV) thrombus secondary to PE itself is also rare. Here, we present a case of a young male patient with no prior risk factors, who came to us with a large bullae and cavitation in lung which was secondary to PE. Later he developed RV thrombus and saprophytic fungal colonisation of affected lung which was all attributed to PE. Patient was managed conservatively with anticoagulation and his repeat echo after two months showed clearance of RV thrombus.

Keywords: Deep vein thrombosis; Pulmonary embolus

# **Case Report**

A 23 year old previously healthy adult male presented with complains of cough since 5 days which was insidious in onset, gradually progressive in nature; with muco-purulent expectoration often blood tinged and non foul smelling. He described that 1 day prior to these symptoms he had an episode of sudden onset choking and breathlessness which lasted in 5 minutes after taking rest. There was no history of pain or swelling of lower limb, trauma, recent surgery or prolonged immobilisation, drug intake and significant weight loss. He had no previous medical illness. There was no history of any illicit sex or intravenous drug abuse. None of his family members had similar illness.

## **Clinical Examination**

On examination he was febrile with a temperature of 99.4 F, pulse rate was 120/minute, regular and all peripheral pulses were felt. B.P-130/70 mm Hg, respiratory rate of 32/minute and pulse oximetery saturation was 89%. On cardiovascular examination, Second heart sound was loud in pulmonary area and a Grade 2 pansystolic murmur was present in tricuspid area. Respiratory system examination revealed a central trachea with grossly diminished air entry on right side with fine crepitations in right axillary and mammary area, left side was normal. Central nervous system examination was normal.

#### Investigations

On routine haematological examination, ESR was 31 mm, Hb-13 gm/dl, TLC-15,000/cu mm of which 81% were neutrophil, 16% lymphocyte and 2% eosinophil. Arterial Blood Gas analysis showed respiratory alkalosis with a pH of 7.52,  $pCO_2$ -21.3 mm Hg,  $pO_2$ -50.1 mm Hg,  $SO_2$ -90.1%. His random blood sugar, renal function tests, serum electrolytes and liver function tests were normal. ELISA for HIV, blood culture and Mantoux test was negative. Sputum gram stain and culture was negative.

Electrocardiography showed sinus tachycardia, SIQ3T3 pattern and T wave inversion in V1-V3 leads (Figure 1). Chest X-ray taken 5 days prior to admission was suggestive of ground glassing in right upper/middle zone with irregular opacities in left perihilar region (Figure 2). Chest X-ray on day of admission showed a large thick walled cavitatory lesion in right mid and lower zone and consolidation in right lower zone lung parenchyma, with lucency in right upper zone suggestive of bulla formation, right costophrenic angle was blunted and cardia was pushed to left side (Figure 3). Chest X-ray after 7 days of hospital stay showed a well defined air fluid level in the cavity along with resolution of right lower zone pulmonary infiltrates (Figure 4).

Contrast Enhanced Compute D Tomography (CECT) chest was done on the very day of admission which revealed a subacute thrombus in right Main Pulmonary Artery (MPA) extending into the sub-segmental artery of right lower lobe, right lower lobe collapse/ consolidation with abscess cavity, large bullae in right upper lobe and loculated hydro-pneumothorax in right lower lobe (Figure 5). Echocardiography done on the same day which showed dilated Right Atria (RA) and Right Ventricle (RV) with a dimension of 4.3



\*Corresponding author: Soumik Ghosh, PGIMER, Dr. Ram Manohar Lohia Hospital, India, Tel: 011 2336 5525; E-mail: soumik13joy@gmail.com

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**Figure 3:** Chest X-ray on day of admission showed a large thick walled cavitatory lesion in right mid and lower zone and consolidation in right lower zone lung parenchyma.



**Figure 4:** Chest X-ray after 7 days of hospital stay showed a welldefined air fluid level in the cavity along with resolution of right lower zone pulmonary infiltrates.

and 4.7 cm respectively, hypokinetic free wall of RV, moderate Tricuspid Regurgitation (TR) with dilated MPA with thrombus in Right Pulmonary artery (RPA). However, all cardiac chambers were echofree (Figure 6). Repeat echocardiography after a week revealed, severe TR, severe pulmonary arterial hypertension (PASP- 72 mm Hg). A large thrombus was noted at RV apex measuring  $3.5 \times 2.6$  cm with

both above and below moderator band attachment. RPA was full of thrombus with no clot in SVC and IVC. Left ventricular systolic and diastolic functions were normal with an Ejection Fraction of 60%. Trans-Esophageal Echocardiography (TEE) showed large crescentic mass of approximately 4 cm in Right Ventricle near Right Ventricle Outflow Tract (RVOT). RPA was full of thrombus.

CardiacMagneticResonance(CMR)showedhypokineticandakinetic wall of right ventricle with systolic dysfunction. A soft tissue intensity lesion suggestive of thrombus seen in right ventricular apex measuring  $2 \times 1.7$  cm. A large thrombus was seen occluding Right pulmonary artery along with a wedge shaped area of consolidation in right paracardiac region (Figure 7).

Sputum culture was repeated and reported positive fungal growth for Aspergillus fumigatus and Aspergillus flavus after two weeks hospitalisation. However serum precipitin and Galactomannan tests were negative.

Echocardiography, after one month of anticoagulation, showed persistent horse-shoe shaped mobile thrombus attached to right ventricular apex, size was smaller than earlier. Mild TR, moderate pulmonary hypertension with RPA thrombus which was partially recanalized and RV size smaller than earlier (Figure 8). Repeat CECT chest showed RPA thrombus with partial recanalization and RV thrombus, with markedly destroyed right lung (Figure 9).

Any other source of emboli was ruled out by venous colour doppler of both lower limbs and abdominal veins which was normal. Tests for



Figure 5: Large bullae in right upper lobe and loculated hydropneumothorax in right lower lobe.



Figure 6: Echocardiography done on the same day which showed dilated Right Atria (RA) and Right Ventricle (RV) with a dimension of 4.3 and 4.7 cm respectively.

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**Figure 7:** A large thrombus was seen occluding Right pulmonary artery along with a wedge shaped area of consolidation in right paracardiac region.



Figure 8: Mild TR, moderate pulmonary hypertension with RPA thrombus which was partially recanalized and RV size smaller than earlier.



recanalization and RV thrombus, with markedly destroyed right lung.

hypercoagulable states (Protein C and S, Factor V leiden mutation, Antithrombin 3, Prothrombin time, activated partial thromboplastin time, lupus anticoagulant and homocysteine levels) were normal. His connective tissue profile (ANA, c ANCA, p ANCA and rheumatoid factor) were negative. Ultrasound abdomen was normal. Serum immunoglobulin and complement levels were also within normal limits.

# Diagnosis

Idiopathic pulmonary thromboembolism with lung bullae, cavitation and saprophytic *Aspergillus* colonisation with secondary right ventricular thrombus formation.

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## Treatment

Patient was treated empirically with intravenous Amikacin and Piperacillin/Tazobactam for lung abscess. Injection enoxaparin and oral anticoagulation with warfarin was started spontaneously as soon as pulmonary artery thrombosis was diagnosed in CECT chest. Enoxaparin was stopped after 5 days and warfarin continued to maintain an INR 2-3.

### Course in hospital

Patient stayed in hospital for almost a month and was discharged on anticoagulation to maintain INR 2-3.

### Follow-up

On follow up, chest X ray after 2 months, showed evidence of right middle zone collapse with right sided upper and middle zone lucency with absence of vascular markings and blunting of costophrenic angle suggestive of loculated pneumothorax with pleural effusion (Figure 10). Subsequent echocardiography after 2 month showed RV cavity free of thrombus, with normal sized all cardiac chambers, mild TR and pulmonary hypertension with haziness in RPA.

He is asymptomatic and is leading a normal life. He is well aware of the future risk of PE and is on regular follow up.

# Discussion

PE is part of the spectrum of diseases that along with DVT, falls under the spectrum of Venous Thromboembolism (VTE). The clinical presentation may be varied and masquerades other disease symptomatology. In most cases, PE is secondary to DVT. Other rare etiologies are tumours, air bubbles, amniotic fluid, or fat from a long bone fracture. The causes for PE are multifactorial and apparently cannot be attributable in many cases. Hypercoagulable states are one of the causes which may be acquired or congenital. Factor V Leiden mutation causing resistance to activated protein C is the most common risk factor. An inherited hypercoagulable can be detected in approximately 60% of patients presenting with idiopathic VTE. However, one-third of them have unremarkable test results, which

Figure 10: On follow up, chest X ray after 2 months, showed evidence of right middle zone collapse with right sided upper and middle zone lucency with absence of vascular markings and blunting of costophrenic angle suggestive of loculated pneumothorax with pleural effusion.

may be due to a disorder or defect that has not yet been discovered or characterized [1]. Many patients of PE do not have ultrasonographic evidence of DVT, probably because clot has already embolized to lungs.

Cavitation due to pulmonary emboli has been described earlier, although it is also an unusual finding [2-4]. Infection in these cavities is common and can cause abscess formation. Bullae formation in PE is a rare occurrence with only few reported cases in past [5]. However, to the best of our knowledge both bullae and cavity formation in a same patient of PE has not been reported in medical literature so far.

Invasion of necrotic tissue is a rare form of aspergillus lung disease that manifests as colonization of non-viable lung parenchyma from pulmonary infarction [6-7] or necrotizing pneumonia. Antifungal treatment is not required in such a clinical scenario, unless invasion of viable lung tissue occurs, which is rare in immune-competent patients.

Echocardiographic findings in PE include RV dilatation and hypokinesia, inter-ventricular septal dys-synchrony, TR and lack of inspiratory collapse of the inferior vena cava [8]. Right ventricular thrombus can be present unusually in peripheral embolism. RV thrombi are of two types- type A and B. Type A thrombi occur when peripheral thrombus during their course to the lungs, get entrapped in a patent foramen ovale, tricuspid chordae or Chiaris network, they have marked mobility and are characteristically localized in the right atrium. Patients with type A thrombi have a poor prognosis and higher early mortality of 44%. Type B thrombi are found attached to the walls of the cardiac chambers and are probably of local origin [9] and patients with type B thrombi constitute a low risk group with thrombus related mortality of 4% [10]. In our case it was type B thrombi as it was not present in initial echocardiography which was done on the day of admission.

Thrombolytic therapy is unanimously advocated in cases of proven PE with cardiogenic or with systemic hypotension without shock. The use of thrombolysis in clinical context apart from the above, i.e. PE causing RV dilatation and hypokinesis without systemic hypotension is doubtful.

In the literature, there is lack of an accurate evaluation of right

ventricular thrombi due to PE and no consensus regarding the optimal treatment for these patients. It is due to evaluation of anecdotal case reports or short series, collected retrospectively in a metaanalysis which frequently varies in the severity of the underlying PE and homogeneity of thrombus morphology. The timing of the echocardiographic examination for follow up is still to be standardized [11]. Further randomized controlled trials are necessary to optimize the management of RV thrombus formation following PE [9].

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