

Pulmonary Embolism Mimicking Acute Severe Asthma

DP Bansal¹, M Maazuddin², M Viquasuddin³

Abstract

When a patient presents with wheezing, pulmonary embolism is not usually considered as a possible cause. However bronchoconstriction can be caused by the embolism which produces wheezing that may be so obvious to make a diagnosis of bronchial asthma. Here we present a case of sub-massive pulmonary embolism presenting predominantly with wheezing without any previous history of cardio pulmonary diseases. The patient was treated for acute severe asthma in the beginning later suspected to have pulmonary embolism because of poor response to therapy and was confirmed by appropriate investigations. The present case thus emphasizes that pulmonary embolism can mimic acute severe asthma.

Introduction

Venous thromboembolism which encompasses Deep vein thrombosis (DVT) and Pulmonary embolism (PE) is one of the three major cardiovascular causes of death along with myocardial infarction and stroke.¹ Evidence of DVT is found in about 70% of patients who have sustained PE. The most common presentation of

minor PE includes dyspnoea with or without pleuritic pain and hemoptysis. The second presentation includes hemodynamic instability which is associated with acute massive PE. Lastly for subacute massive PE, common

symptoms include dyspnoea and chest discomfort mimicking heart failure/ acute coronary syndrome.² Isolated wheezing is uncommon but could be possible presentation of pulmonary thromboembolism.³ In the PIOPED trial, 11% of patients with pulmonary embolism had wheezing at admission.⁴

Here we report a case of PE presenting as acute severe asthma.

Case History

A 60 year old obese female, known hypertensive on telmisartan, non diabetic admitted with history of breathlessness for 1 week prior to admission. There was history of dry cough and wheezing with no history of chest pain, fever, orthopnoea and PND (paroxysmal nocturnal dyspnoea). On

¹Consultant Pulmonologist and Intensivist, ²Clinical Pharmacologist, ³Senior Resident -Internal Medicine, Olive Hospital, Hyderabad, Telangana
Received: 18.02.2017; Accepted: 06.02.2018



Fig. 1: Filling defect noted in the right pulmonary artery extending distally into segmental branches

examination patient was tachypnoeic, tachycardiac. Blood pressure was 130/80 mm of hg. Accessory muscles of respiration were active; SpO₂ was 80% on room air which improved to 95% with oxygen therapy. CVS – S₁S₂ heard. No murmur or gallops. Lungs showed bilateral diffuse rhonchi. Per abdomen and CNS – within normal limits. Clinically diagnosis of late onset asthma with exacerbation V/S left ventricular failure was considered and she was investigated and treated

for the same.

Investigations

Complete blood picture – haemoglobin: 12.0 gm%; Total leukocyte count: 11500; Platelets: adequate.

Liver function test - within normal limits

Renal function test - within normal limits

Chest X-ray - Normal

Arterial blood gases - hypoxia with respiratory alkalosis.

2D echo - normal LV function, mild PAH, no pericardial effusion.

ECG - Sinus tachycardia

D-Dimer - positive

Venous Doppler of lower limbs - no evidence of any venous thrombosis

CT chest with pulmonary angiogram revealed thrombus in the lower division of right pulmonary artery Figures 1 and 2.

Course in the Hospital

Patient was treated for acute severe asthma with intravenous (I.V) steroids, oxygen therapy and bronchodilators. However patient's breathlessness and

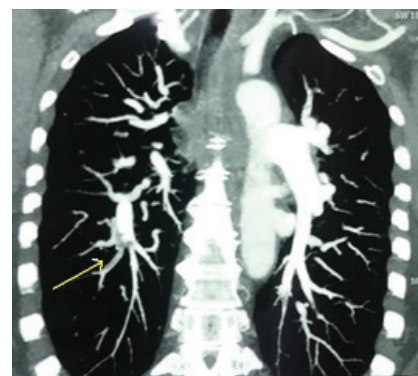


Fig. 2: Filling defect noted

bronchospasm were persistent despite optimum treatment for asthma. In view of obesity and limited physical activity diagnosis of pulmonary embolism was considered that was objectively supported by positive D-Dimer. CECT chest with pulmonary angiogram was done which revealed right sided pulmonary artery (segmental) embolism. Patient was started on anticoagulation therapy (injection enoxaparin 60mg twice daily for 5 days). Thrombolysis was not needed in view of normal blood pressures and normal right ventricular functions.

Patient showed improvement in her breathlessness and she was started on oral anticoagulant therapy (warfarin 6mg). Patient was discharged in a stable condition on oral anticoagulant and asked to come for regular follow-ups.

Discussion

PE is known as “the great masquerader”. Due to non specific signs and symptoms diagnosis becomes difficult. The most common symptom is unexplained breathlessness and sign is tachypnoea.¹ Bronchospasm can occur in patients of PE which could be the initial presentation in some patients. In the index case, patient had no past history of any cardiopulmonary disease including bronchial asthma. Patient presented with sudden onset of breathlessness and on clinical examination patient had bilateral wheeze making diagnosis of asthma more likely. The relationship of bronchoconstriction to PE is well known but the mechanism of bronchoconstriction is not very clear. In an experimental work on animals,⁵ it

is subtle that bronchospasm is related to release of serotonin from platelets of the clot. The release of serotonin can be prevented by heparin. The other mechanism for development of bronchospasm in PE could be due to the reduction in PaCO₂ which can cause bronchoconstriction in that affected areas of lung. It may be difficult to differentiate whether the wheezing in PE is due to embolus or patient has underlying airway disease. Some of the features may suggest PE as a cause of wheezing, it includes risk factors of DVT and PE like pregnancy, immobilisation, malignancy, morbid obesity etc, and failure to respond to optimal therapy of bronchial asthma. In one study about 85% patients that complained of wheezing were later diagnosed with recent pulmonary emboli,⁶ while another study showed about 4% of patients with pulmonary Emboli had wheezing.⁷

Sudden onset of breathlessness and wheezing could be the only presenting symptoms in patients of PTE. It is important for the clinician to know that

wheezing is not only because of airway diseases like asthma, COPD, heart failure or upper airway pathology but it could be only presenting symptoms and sign in patients of PE.

References

1. Goldhaber SZ. Deep venous thrombosis and pulmonary thromboembolism .In: Harrison's principles of internal medicine vol 2 19th ed. McGraw hill education 2015; 1631-36.
2. Riedel M. Acute pulmonary embolism 1: pathophysiology, clinical presentation, and diagnosis. *Heart* 2001; 85:229-240.
3. Calvo-Romero JM, Pérez-Miranda M, Bureo-Dacal P. Wheezing in patients with acute pulmonary embolism with and without previous cardiopulmonary disease. *Eur J Emerg Med* 2003; 10:288-9.
4. Stein PD, Terrin ML, Hales CA, et al. Clinical, laboratory, roentgenographic and electrocardiographic findings in patients with acute pulmonary embolism and no pre-existing cardiac or pulmonary disease. *Chest* 1991; 100:598-603.
5. Boyer NH, Currey JJ. Bronchospasm associated with pulmonary embolism respiratory failure. *Arch Intern Med (Chic)* 1944; 73:403-9.
6. Sasahara AA, Cannilla JE, Morse RL, Sidd JJ, Tremblay GM. Clinical and Physiologic studies in pulmonary thromboembolism. *Am J Cardiol* 1967; 20:10-20.
7. Windebank WJ, Boyd G, Moran F. Pulmonary thromboembolism presenting as asthma. *British Medical Journal* 1973; 1:90-94.