Journal Homepage: -www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/19519
DOI URL: http://dx.doi.org/10.21474/IJAR01/19519

RESEARCH ARTICLE

PULMONARY EMBOLISM MIMICKING ASTHMA EXACERBATION

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Manuscript Info

Manuscript History
Received: 21 July 2024
Final Accepted: 24 August 2024
Published: September 2024

Key words:-
Pulmonary Embolism, Asthma, Shortness of Breath, Clinical Diagnosis, Virchow's Triad

Abstract

Pulmonary Embolism poses a significant health threat. Pulmonary Embolism arises from the obstruction of pulmonary arteries, typically by blood clots originating from deep veins. Risk factors mirror those of deep vein thrombosis, encompassing genetic and acquired factors. Timely diagnosis is imperative due to substantial mortality and morbidity rates, with various diagnostic modalities employed. Treatment options include anticoagulants, thrombolytic, surgical embolectomy, and inferior vena cava filters. We present a case from our Emergency department, underscoring the importance of prompt diagnosis and intervention in managing PE. The case involved a 46-year-old male admitted who is known case of Asthma (on inhaled corticosteroids), who presented with dyspnoea, leading to the diagnosis of high-risk PE. Thrombolysis with Reteplase was initiated, highlighting the critical role of identifying the pulmonary embolism.

Introduction:-

Pulmonary Embolism, a blockage of arteries in lung is a life threatening disease. Most common Sources of Pulmonary emboli are Pelvic veins and deep veins of the leg.

Risk factors for pulmonary embolism include heart disease, Surgery, Smoking, Disorders that affect clotting, Covid 19, Smoking, Obesity, Pregnancy and persons having Virchow's triad.

Pulmonary embolism (PE) is a potentially life-threatening condition caused by the obstruction of the pulmonary arteries due to blood clots. It presents a diagnostic challenge because its clinical symptoms overlap with various respiratory conditions, including asthma. This overlap can delay diagnosis and increase the risk of severe complications. Asthma is one of the chronic inflammatory disease associated with pro coagulants and anti-fibrinolytic activities in airways, so therefore asthma is likely a risk factor for pulmonary embolism.

Asthma is a chronic inflammatory disease of the airways that is characterized by episodes of wheezing, shortness of breath, and chest tightness. Pulmonary embolism, on the other hand, may present with similar respiratory symptoms such as dyspnea, tachypnea, and wheezing.

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In this case study the development of pulmonary embolism is collectively thought to be due to Virchow's triad (alterations in blood flow; factors associated with endothelium damage in the vessel wall; and factors affecting blood properties) because of Asthma.

Method:-

This is a case study which was observed and followed up case.

Results/Findings:-

A 46-year-old Male presented to ED with history of chest tightness, shortness of breath and unable to complete full sentences since 3 hours. The patient denies history of hemoptysis, cough, chest pain, fever, Epigastric pain, or lower limb swelling.

On examination
Vitals: Temp: 98 **Pulse: 124 bpm**; **Respiratory Rate: 28**; Blood pressure 110/80 mmHg **Spo2 93%on 3L/min Oxygen via mask.**

No central or peripheral cyanosis, Pallor, Edema or Clubbing. Patient was conscious oriented, wheezes bilaterally were found.

Modified well's Score: 1.5 (Low risk Group) less likely

Patient was investigated in form of **ECG – Sinus tachycardia**; **Trop I 0.034** (Normal value- <0.034)

2D-Echo showed dilated RA, RV; Moderate RV systolic dysfunction; Moderate TR, Severe PAH; Dilated and partially collapsing IVC; Normal LV Systolic function.

Radiological investigations: CT Pulmonary Angiography - Hypo dense non-enhancing filling defect seen involving distal most segment of right and left main pulmonary arteries, bilateral lobar and segmental and sub segmental branches with lower lobar predominance causing partial to complete luminal occlusion. Main pulmonary trunk: 29 mm, right pulmonary artery: 20 mm, Left pulmonary artery: 21 mm.

Fibrotic changes are noted in left upper lobe lung. Areas of ground glass opacity with reticular thickening is noted in basal segments of bilateral lower lobe lung suggestive of gravity dependent atelectasis. Few foci of calcification are noted in Apico-posterior segment of left upper lobe lung.

Suggestive of **Significant bilateral pulmonary arterial thrombo-embolism** as described above. Fibrotic changes in left upper lobe lung. Gravity dependent atelectasis in bilateral lower lobe lungs.

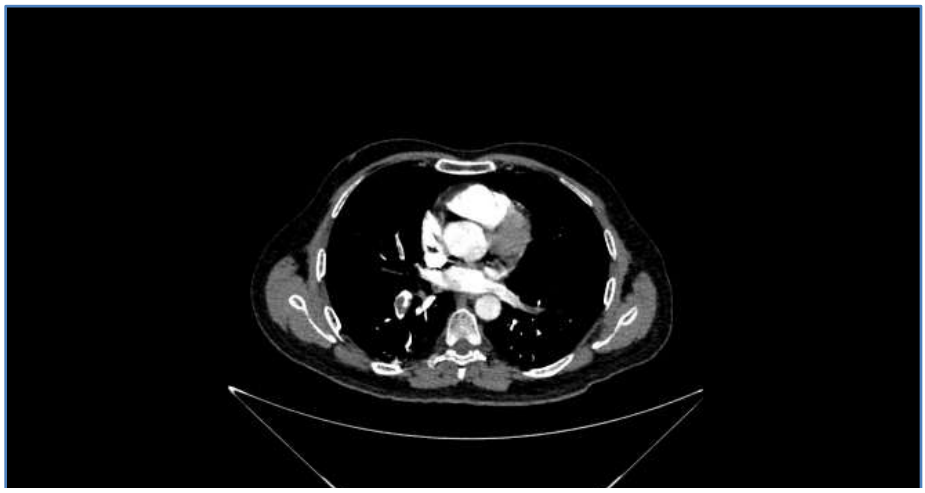

Figure 1:- CT Pulmonary Angiography showing hypo-dense non enhancing defect seen involving right segmental branches and left pulmonary artery.

USG Abdomen-
Fatty liver (Grade-I). Left kidneys show mild hydronephrosis. Right renal simple cyst. Faecal loaded colon. Bilateral Lower Limb Venous Doppler Study showed Hemodynamically, normal both lower limb venous Doppler study with No evidence of venous thrombosis seen.

Discussion:-

The patient was initially treated as Acute Moderate Asthma, however after 2D Echo, CT pulmonary angiography findings and Vital examination collectively pointed us towards a diagnosis of Pulmonary embolism. As per study published by Po-Hsin Lee and Pin-Kuei Fu in 2019 Asthma is one of the chronic inflammatory diseases associated with procoagulants and antifibrinolytic activities in the airways, which increases risks of venous thromboembolic events.

Pulmonary embolism (PE) is a potentially life-threatening condition caused by the obstruction of the pulmonary arteries due to blood clots. It presents a diagnostic challenge because its clinical symptoms overlap with various respiratory conditions, including asthma. This overlap can delay diagnosis and increase the risk of severe complications.

Clinical Presentation and Diagnostic Challenge
Asthma is a chronic inflammatory disease of the airways that is characterized by episodes of wheezing, shortness of breath, and chest tightness. Pulmonary embolism, on the other hand, may present with similar respiratory symptoms such as dyspnea, tachypnea, and wheezing. Both conditions share common triggers such as respiratory infections, exertion, and stress, which can further confound the diagnostic process. Patients with PE may present with non-specific symptoms, leading to misdiagnosis as asthma, particularly in patients with pre-existing respiratory diseases. A study by Tapson (2008) found that the presentation of dyspnea and chest pain in PE can be confused with asthma, delaying the correct diagnosis. This is especially relevant in young adults who present with acute onset respiratory distress but have no prior history of asthma (Lualdi & Goldhaber, 1995).

The absence of typical asthma findings, such as significant response to bronchodilators, or the presence of risk factors for venous thromboembolism (e.g., immobility, recent surgery, or thrombophilia), should raise suspicion of PE in these cases. The role of diagnostic tools such as D-dimer levels, computed tomography pulmonary angiography (CTPA), and ventilation-perfusion (V/Q) scanning becomes essential in differentiating PE from asthma.

Pathophysiology and Mechanism of Confusion
The pathophysiology behind the overlap of symptoms can be attributed to airway obstruction in both conditions. In asthma, this is due to inflammation, bronchoconstriction, and increased mucus production, while in PE, hypoxemia resulting from impaired gas exchange due to vascular occlusion may lead to reflex bronchoconstriction. This may present clinically as wheezing, typically associated with asthma, but also observed in patients with PE (Rashid et al., 2017). Furthermore, hypoxemia and hyperventilation in PE can result in respiratory alkalosis, a finding that can also be seen during acute asthma exacerbations (Simonneau et al., 2019).

Importance of Differential Diagnosis
The need for accurate differential diagnosis between PE and asthma is crucial for timely and appropriate treatment. Mistreatment of PE as asthma, such as administering bronchodilators or corticosteroids without addressing the thrombotic component, can lead to worsening of the patient's condition. A retrospective analysis by Carson et al. (1992) suggested that patients with undiagnosed PE who were treated for other respiratory conditions had a significantly higher mortality rate.

The importance of considering PE in patients presenting with atypical asthma symptoms, especially those with risk factors for thromboembolism, cannot be overstated. Clinical guidelines recommend that any sudden onset of unexplained dyspnea or chest pain should prompt an investigation for PE, particularly if the patient's history does not support a diagnosis of asthma (Stein et al., 2017).

In this case study as the diagnosis of Pulmonary Embolism was confirmed patient was Thrombolysed with Injection Reteplase 18 mg IV stat Followed by another 18 mg IV stat 30 mins apart and after 2 hours of thrombolysis patient

was symptomatically better with decreasing oxygen requirement to maintain oxygen saturation and Tachycardia settled down to Normal heart rate.

Conclusion:-

Our case provides an important information that asthma is a risk factor for pulmonary embolism and patients having frequent exacerbations of asthma should be evaluated for having a pulmonary embolism after an acute dyspnea. The clinical overlap between pulmonary embolism and asthma presents a significant diagnostic challenge. Both conditions may present with dyspnea, and chest pain, which can lead to misdiagnosis and delayed treatment. Clinicians must maintain a high index of suspicion for PE in patients with atypical asthma symptoms, especially in the presence of risk factors for thromboembolism. Early use of diagnostic tools such as D-dimer testing and CTPA can help differentiate between these conditions and lead to more effective management strategies, ultimately improving patient outcomes.

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