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Case Report

Post-traumatic pulmonary embolism in the setting of cough-variant asthma ☆

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ABSTRACT

Post-traumatic pulmonary embolism (PE) poses diagnostic complexities, especially with underlying lung pathologies and delayed symptoms. We report a 43-year-old male who presented with cough, frothy sputum, and dyspnea following blunt chest trauma 2 weeks ago. Due to a history of asthma, an asthma exacerbation was suspected but he failed to respond to bronchodilator therapy. Doppler USG (ultrasonography) was negative for deep venous thrombi, however, elevated D-dimer levels prompted further investigation with computed tomography pulmonary angiography (CTPA), confirming the diagnosis of PE. Prompt initiation of anticoagulation and thrombolysis resulted in clinical improvement. This case underscores the need to rule out posttraumatic PE, irrespective of age, type of, or time since injury, and underlying lung disease, to ensure timely diagnosis and intervention.

Introduction

Pulmonary embolism (PE) poses a diagnostic challenge due to its varied and often nonspecific clinical presentation. Post-traumatic pulmonary embolism (PE) is a rare yet grave complication that affects up to 5% of trauma patients, typically manifesting between the fifth and seventh days following the injury [1]. The incidence of post-traumatic VTE can be up to 13 times more than that of nontraumatic patients [3]. However, the risk is highest at 1 week after the injury with most patients being diagnosed within a few days or 24 hours post-trauma. A recent study indicates that 41.5% of all cases of pulmonary thromboembolism (PTE) in trauma patients occur within 72 hours of the trauma, with PTE patients experiencing a notably higher mortality rate compared to non-PTE patients beyond this timeframe [4].

Delayed presentation and the presence of underlying pulmonary co-morbidities such as Asthma, make the diagnosis particularly more challenging. Prompt recognition and intervention are imperative to mitigate associated morbidity and mortality. Physicians must maintain a high level of suspicion of pulmonary embolism (PE) even when symptoms and signs are nonspecific [5,12]. We present a case highlighting the diagnostic intricacies encountered in identifying PE following chest trauma in the presence of comorbidities such as

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Asthma, underscoring the significance of clinical evaluation and careful utilization of investigative modalities. The overlap of symptoms between these conditions can obscure the diagnosis of PE, making it challenging to distinguish from other potential issues like asthma exacerbation or trauma-related injuries.

Case presentation

A 43-year-old male, presented to the outpatient department with a 2-week history of cough with frothy sputum production and progressive dyspnea (mMRC- Grade 4) following a road traffic accident (RTA) with blunt trauma to the chest.

Clinical examination revealed central cyanosis, tachycardia, and hypoxemia. Further evaluation unveiled no overt signs of heart failure or pneumonia. A thorough systemic examination yielded no significant abnormalities. All Lab investigations, including complete blood count (CBC), were within normal limits.

Chest X-ray (PA view) was essentially normal, showing no signs of pneumothorax, Pleural effusion, rib fractures, lung contusions, or any other pathology (Fig. 1).

The patient had a long history of cough-variant Asthma, which was initially suspected to be the cause of his respiratory symptoms. However, the failure of response to bronchodilator therapy raised suspicion of an alternative diagnosis.

Post-traumatic pulmonary embolism was suspected, however, Doppler ultrasonography only revealed incompetent saphenofemoral, and popliteal junctions on the right side along with ectasia of the bilateral great saphenous veins but without any evidence of thrombosis or obstruction.

With an elevated D-Dimer of 700 mg/dL, we calculated the Well's score to be 4.5 which indicates a moderate probability of pulmonary embolism.

A CTPA (pulmonary angiogram) was performed which revealed a saddle embolus at the pulmonary artery bifurcation with extension into the main pulmonary arteries and partial occlusion of their lumen (Figs. 2, 3 and 4).

The patient was commenced on low molecular weight heparin (enoxaparin). Upon stabilization, he was discharged with rivaroxaban and symptomatic management. Thrombolysis was pursued at a tertiary cardiac center to expedite clot resolution. Financial constraints precluded comprehensive thrombophilia profiling.

The patient showed clinical improvement post-thrombolysis with resolution of dyspnea and normalization of oxygen saturation. The significance of long-term anticoagulation was emphasized to avert recurrent thromboembolic events.

Discussion

Pulmonary embolism, one of the most common clinical manifestations of Venous-Thromboembolism is a fatal but preventable complication frequently seen in Trauma Patients. Patients usually present with dyspnea, tachypnea, chest pain, and hemoptysis [2], but it can be difficult to diagnose in post-traumatic cases with relatively delayed presentation, especially in the setting of underlying respiratory conditions such as Asthma.

Identifying acute pulmonary embolism is one of the most complex challenges in clinical practice [14]. This is especially true when the presentation is delayed and the patient has an underlying lung disease, such as Asthma in our case, which can present similarly. Cough can be the primary or presenting symptom in such patients which is why it is important to maintain a high level of suspicion to identify and diagnose pulmonary embolism correctly [13]. Moreover, reviewing the literature, the risk factors for post-traumatic PE such as severe injury, long bone fractures, obesity, and elderly age [7] were all absent in our patient which initially made an Asthma exacerbation a more likely cause of the patient's respiratory distress.

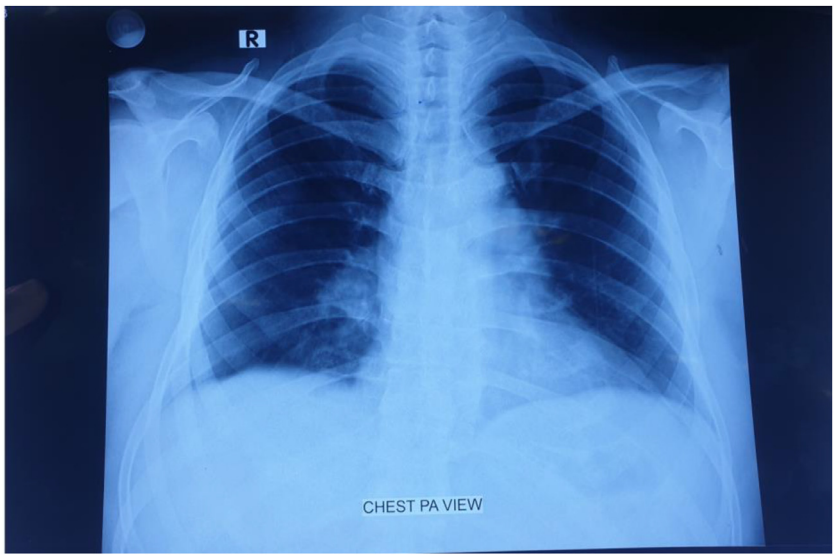

Fig. 1 – Chest X-ray, postero-anterior (PA) view showing no signs of consolidation, lung contusion, or rib fractures.

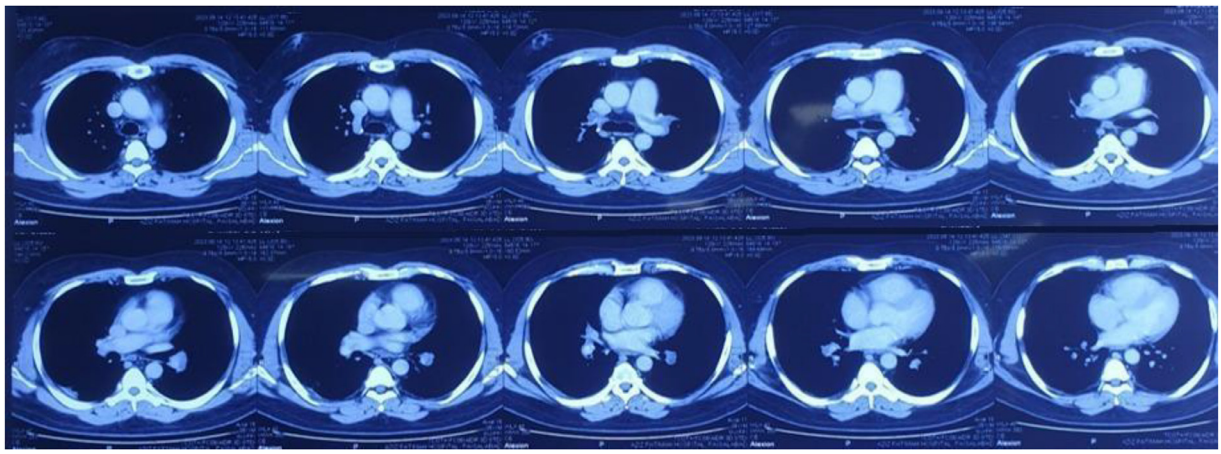

Fig. 2 – Computed tomography pulmonary angiography—axial view shows intraluminal fillings defect in the main pulmonary artery, also extending into left and right pulmonary segmental arteries partially occluding their lumen.

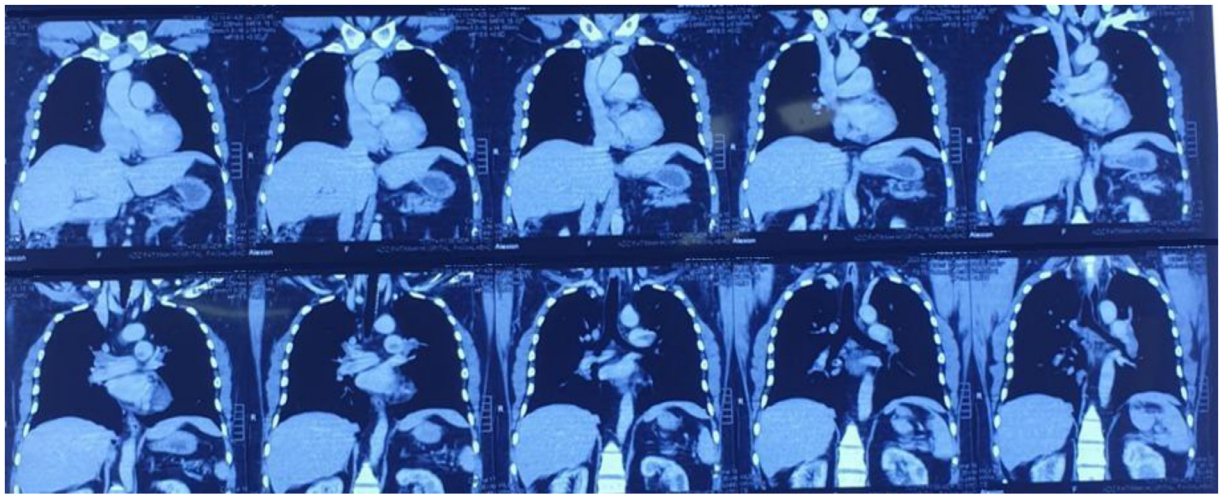

Fig. 3 – CTPA—Coronary view showing multiple intraluminal filling defects in segmental and lobar branches of the pulmonary arteries, partially occluding their lumen.

What makes the case even more challenging is that there was no evidence of venous thrombosis, including in the deep veins of the legs, iliac and renal veins, or the inferior vena cava [11]. Potentially, in situ clot formation in the pulmonary vasculature could be attributed to localized inflammation, concealed vascular injury, and the diminished blood flow state ensuing from thoracic injury [8–10]. which may explain the origin of the PE in our patient.

Moreover, Pulmonary Embolism usually presents within the first week after trauma while our patient presented 2 weeks after the accident. However, it is interesting to note that the hypercoagulable state remains much longer than this period, as shown in a retrospective study involving 267,743 trauma patients, which found that the highest incidence of venous thromboembolism (VTE) was observed within the first 3 months postinjury, gradually returning to normal levels between 12 to 15 months thereafter [6]. Therefore, even after the first 3-7 days have passed, the patient is still at risk for developing a VTE or PE, such as in our patient. Diagnosing PE in trauma patients is particularly challenging because many are unable to communicate or report symptoms due to being unconscious or intubated. Additionally, other existing pathological conditions can often account for the subtle signs of PE, further complicating the diagnosis. This complexity increases the risk of delayed or missed diagnosis, potentially leading to severe consequences. Therefore, heightened clinical vigilance and advanced diagnostic tools are crucial in identifying PE in high-risk groups [15].

Regardless of the origin, the high mortality rates warrant prompt diagnosis and treatment of pulmonary embolism, with careful consideration of the appropriate therapy to minimize bleeding risk and prevent clot formation simultaneously.

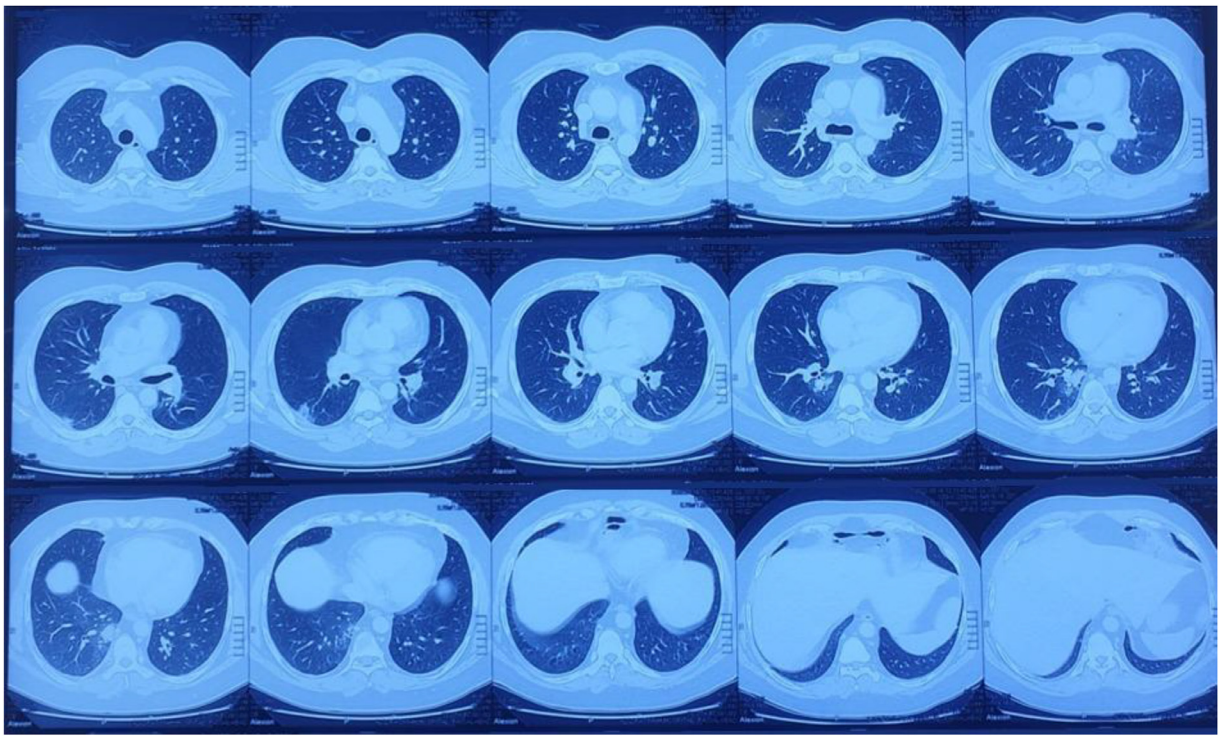

Fig. 4 – Lung window of CT—axial view showing wedge-shaped opacities in the superior region of the lower lobe of the right lung.

Conclusion

Pulmonary embolism represents a significant and potentially fatal complication, particularly prevalent among trauma patients. Despite the challenges in diagnosis, prompt recognition and treatment are crucial to mitigate mortality rates associated with PE. It is important to remember that risk factors such as severe injury or long bone fractures may not always be present and PE should always be ruled out in cases of blunt chest injuries, regardless of the patient's age, underlying lung disease, or the time that has elapsed.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the author(s) used [CHAT GPT / OPEN AI] in order to [improve language and readability]. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

Patient consent

Written, informed consent was obtained from the patient for publication of their case.

REFERENCES

[1] Menaker J, Stein DM, Scalea TM. Incidence of early pulmonary embolism after injury. J Trauma 2007;63(3):620–4. doi:10.1097/TA.0b013e31812f60aa.

[2] Stein PD, Beemath A, Matta F, Weg JG, Yusen RD, Hales CA, Hull RD, Leeper KV Jr, Sostman HD, Tapson VF, Buckley JD, Gottschalk A, Goodman LR, Wakefied TW, Woodard PK. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med 2007;120(10):871–9. doi:10.1016/j.amjmed.2007.03.024.

[3] Hamada SR, Espina C, Guedj T, Buaron R, Harrois A, Figueiredo S, Duranteau J. High level of venous thromboembolism in critically ill trauma patients despite early and well-driven thromboprophylaxis protocol. Ann Intensive Care 2017;7(1):97. doi:10.1186/s13613-017-0315-0.

[4] Bahloul M, Dlela M, Bouchaala K, Triki A, Chelly H, Hamida CB, Haddar S, Bouaziz M. Early post-traumatic pulmonary embolism in intensive care unit: incidence, risks factors, and impact outcome. Am J Cardiovasc Dis 2020;10(3):207–18.

[5] Ekici A, İleri Ş, Aslan H, Ekici M. Troublesome cough as the sole manifestation of pulmonary embolism. Respir Med Case Rep 2019;28:100861. doi:10.1016/j.rmcr.2019.100861.

[6] Godzik J, McAndrew CM, Morshed S, Kandemir U, Kelly MP. Multiple lower-extremity and pelvic fractures increase pulmonary embolus risk. Orthopedics 2014;37(6):e517–24. doi:10.3928/01477447-20140528-50.

[7] Bělohlávek J, Dytrych V, Linhart A. Pulmonary embolism, part I: Epidemiology, risk factors and risk stratification, pathophysiology, clinical presentation, diagnosis and nonthrombotic pulmonary embolism. Exp Clin Cardiol 2013;18(2):129–38.

[8] Knudson MM, Gomez D, Haas B, Cohen MJ, Nathens AB. Three thousand seven hundred thirty-eight posttraumatic pulmonary emboli: a new look at an old disease. Ann Surg 2011;254(4):625–32. doi:10.1097/SLA.0b013e3182300209.
[9] Velmahos GC, Spaniolas K, Tabbara M, Abujudeh HH, de Moya M, Gervasini A, Alam HB. Pulmonary embolism and deep venous thrombosis in trauma: are they related? Arch Surg 2009;144(10):928–32. doi:10.1001/archsurg.2009.97.
[10] Gelbard RB, Karamanos E, Farhoomand A, Keeling WB, McDaniel MC, Wyrzykowski AD, Shafii SM, Rajani RR. Immediate post-traumatic pulmonary embolism is not associated with right ventricular dysfunction. Am J Surg 2016;212(4):769–74. doi:10.1016/j.amjsurg.2015.08.027.
[11] Bělohlávek J, Dytrych V, Linhart A. Pulmonary embolism, part I: Epidemiology, risk factors and risk stratification, pathophysiology, clinical presentation, diagnosis and nonthrombotic pulmonary embolism. Exp Clin Cardiol 2013;18(2):129–38.
[12] Ricketti PA, Ricketti AJ, Cleri DJ, Seelagy M, Unkle DW, Vernaleo JR. A 41-year-old male with cough, wheeze, and dyspnea poorly responsive to asthma therapy. Allergy Asthma Proc 2010;31(4):355–8. doi:10.2500/aap.2010.31.3344.
[13] Prakash UBS. Uncommon causes of cough: ACCP evidence-based clinical practice guidelines. Chest 2006;129(1) 206S–219S. doi:10.1378/chest.129.1_suppl.206S.
[14] Wang SY, Chen H, Di LG. Caution for acute submassive pulmonary embolism with syncope as initial symptom: a case report. J Thorac Dis 2014;6(10):E212–6. doi:10.3978/j.issn.2072-1439.2014.08.11.
[15] Owings JT, Kraut E, Battistella F, Cornelius JT, O'Malley R. Timing of the occurrence of pulmonary embolism in trauma patients. Arch Surg 1997;132(8):862–6. doi:10.1001/archsurg.1997.01430320064010.