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Case Report

Early bilateral pulmonary embolism following a moderate blunt chest trauma: A case report [☆]

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ARTICLE INFO

Article history:

Received 27 November 2021

Revised 29 November 2021

Accepted 3 December 2021

Keywords:

Blunt chest trauma

Pulmonary embolism

Deep venous thrombosis

Anticoagulation therapy

ABSTRACT

Post-traumatic pulmonary embolism (PE) remains a major problem in cardio-pulmonary diseases and represent the third most common cause of death in trauma patients. Traditional PE occur most commonly between the fifth and the seventh day after a major trauma and are rare before the fourth day. Here, we report a case of acute pulmonary embolism developing 1 day after a moderate thoracic injury in a previously well young man. The diagnosis was made by non-invasive methods and the patient was given anticoagulation therapy with good outcome. The circumstances and the early occurrence of PE in this case is at odds with what is generally reported after trauma.

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Introduction

Trauma represent the third cause of death after cardiovascular diseases and cancer [1]. Up to 25% of trauma mortalities can be attributed to chest trauma and its complications [2]. Post-traumatic pulmonary embolism (PE) is an infrequent but serious complication occurring in up to 5% of trauma patients, usually between the fifth and the seventh day post injury [3,4]. Classically, PE is caused by the development of deep venous thrombosis (DVT) in the lower extremities or pelvis that em-

bolize to the pulmonary circulation [5]. Early post-traumatic PE, however, is uncommon and is limited to case reports and small series [6–10]. It is a separate entity that could be related to an unknown biochemical processes and may not even originate from the peripheral veins [11]. It is a difficult diagnosis, which may be missed because of nonspecific presentation, principally in trauma patients. The management of post-traumatic PE represents a real challenge. The use of anticoagulation therapy must be balanced with the nature and risk of

[☆] Conflicts of Interest: The authors declare no conflicts of interest.

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<https://doi.org/10.1016/j.radcr.2021.12.003>

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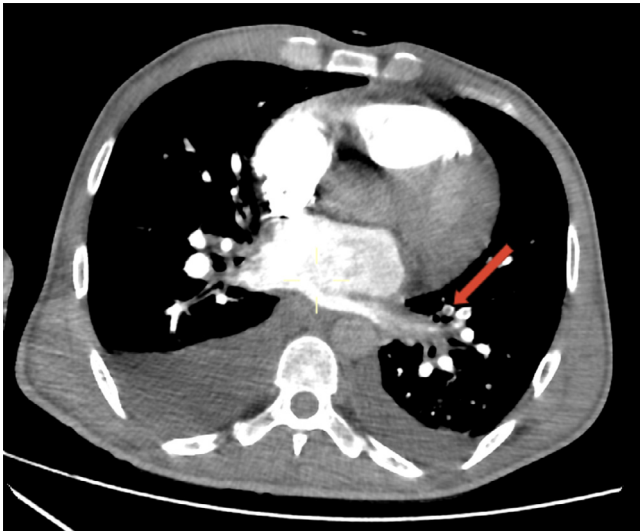


Fig. 1 – Computed tomography pulmonary angiography (CTPA) showing pulmonary embolism (PE) in the segmental branch of the left lower lobe pulmonary artery (A) Axial view (B) Coronal view.



Fig. 1 – Continued

bleeding of the associated injuries. Therefore, it is important to take a multidisciplinary approach to treat the injuries.

Case presentation

A 42 years old man with no significant medical or surgical history, but an active smoker, presented to our emergency department for acute onset of chest pain and dyspnea. Twenty hours earlier, the patient was victim of a road traffic accident while riding a motorcycle, resulting in a chest injury. On physical examination, the patient was normotensive, Glasgow coma scale of 15, with a respiratory rate: 36 cpm, SpO₂: 89% and heart rate: 115 bpm. Cardiovascular examination was unremarkable. No lower limb injury and no clinical signs of DVT were identified.

The patient had no history of recent prolonged immobilization, no personal or family history of prothrombotic conditions, no signs or history of documented COVID-19 infection and was fully vaccinated (second dose administered 3 weeks earlier).

A chest x-rays was performed, showing no signs of fracture, or pneumothorax. Electrocardiogram showed sinus tachycardia with a heart rate of 110 bpm, no repolarization abnormalities were identified.

D-dimer level peaked at 920 mg/L (reference limit 500mg/L), and routine blood investigations including hemogram, coagulation parameters, liver function and renal function tests were normal.

Further investigation with computed tomography pulmonary angiography (CTPA) revealed bilateral PE into segmental branches of the lower lobes (Figs. 1 and 2), with bilateral pleural effusion. There was no sign of active hemorrhage or arterial injury. Doppler ultrasound of the lower extremities was negative for deep vein thrombosis.

Transthoracic echocardiogram showed normal walls motion, as well as normal left ventricular ejection fraction (LVEF: 62%). There was no evidence of right heart strain or pericardial effusion.

In the absence of bleeding risk, systemic anticoagulation with low molecular weight heparin was started immediately and the patient was then admitted for observation and follow-up for 24 hours with a diagnosis of post-traumatic PE. He had an uneventful recovery and vitamin K antagonists were prescribed for the ensuing 6 months.

Discussion

Post-traumatic PE is an acute respiratory disorder caused by the occlusion of the pulmonary arterial bed, leading to a life threatening condition, due to a potentially reversible right ventricular failure [12]. Diagnosing PE may be difficult in injured patients. Clinical presentation is variable, symptoms are often masked by multiple painful injuries, and altered mental status. Patient classically present dyspnea, tachypnea, chest pain and hemoptysis. Severe cases may also involve circulatory instability, or shock. Computed tomography pulmonary angiography (CTPA) is the gold standard for the diagnosis of PE, showing obstruction in the pulmonary artery or its branches.

It is a well-described clinical entity occurring in up to 24% [5,6,13]. Reviewing studies, we found no uniform definition of “early” or “late” post-traumatic PE. Nevertheless, PE occur commonly between the fifth and the seventh day post injury and is rare before the fourth day [3,4,7,8]. It is classically caused by the development of DVT in the lower extremities

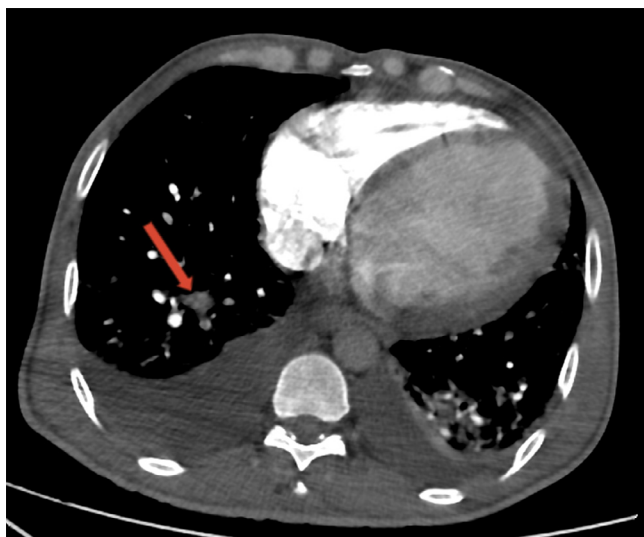


Fig. 2 – Computed tomography pulmonary angiography (CTPA) showing pulmonary embolism (PE) in the segmental branch of the right lower lobe pulmonary artery (A) Axial view (B) Coronal view.



Fig. 2 – Continued

or pelvis that embolize to the pulmonary circulation [3]. However, Velmahos et al. [11] reported that the majority of patients with early PE did not have evidence of DVT and suggested that early PE may have a different pathophysiology. Localized inflammation, occult vascular injury and the low flow state that occur after thoracic injury are a possible etiology for ‘in situ’ formation of a clot in the pulmonary arteries [5,11,14]. This

theory may explain why thoracic injury is associated with PE but not significantly with DVT.

The factors associated with early post-traumatic PE have been understudied. Reviewing the literature, risk factors included elder age, obesity, long bone fractures, and severe injury [7–10]. None of these were present in our patient. Therefore, multidisciplinary discussion questioned the diagnosis of early post-traumatic PE because of the less severe nature of the trauma and the timing so soon of occurrence. The possibility of pre-injury PE was considered, however, the patient had no thromboembolic risk factors and no prior history of COVID-19 infection. As hypercoagulability is present acutely after trauma due to direct tissue injury, this theory may explain the early development of PE in our patient.

Early diagnosis and treatment of post-traumatic PE is very important because the mortality rates is estimated at 50% [15]. The use of anticoagulation therapy must be balanced with the nature and risk of bleeding of the associated injuries. Thus, the management of these patients is complex and requires a multidisciplinary approach.

Conclusion

Post-traumatic PE remains relatively common and can occur early after trauma and in the absence of lower extremity fractures. PE are usually associated with severe trauma. However, in mild/moderate blunt chest trauma like in our case, physicians should maintain a high index of suspicion and consider PE in patients with unexplained dyspnea/hypoxia early after injury. We believe that a better understanding of risk factors could lead to earlier recognition and, possibly, prevention of early PE, but further studies are required.

Consent for publication

Obtained.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Acknowledgments

We would like to thank the team of cardiology and radiology of university hospital for their management and availability.

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