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NOTES ON CASE HISTORY

Blunt Chest Trauma and Pulmonary Thromboembolism, as a Rare Complication

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Study Area: Mashhad, Iran
Coordinates: 36°18'N; 59°36'E

Key words: Hypercoagulable state

Approved by the local Ethics Committee of Mashhad University of Medical Sciences (IR.MUMS.REC.1394.304).

Introduction:

One of the factors that enhance fibrinogen synthesis and promote its catalysis to fibrin is Events of trauma. The triad of venous injury, slow blood flow, and hypercoagulability are the cardinal inciting mechanisms for venous thromboembolism (Rogers *et al.*, 2000). Although there is no definitive explanation for the cause and timing of PE in trauma patients, However, Studies have shown PE is a complication that on average 4-7 days after serious injury (Menaker *et al.*, 2007).

We report a case of a PE that develops after 3 days of a right lower chest blunt trauma. Our goal from presenting this patient is that the physicians working in the trauma department should consider pulmonary embolism when dealing with a patient with chest trauma.

Case study:

A 25-year-old man complaining of pleuritic chest pain was presented to the emergency department at Emam Reza hospital. The patient had fallen for three days before the 2-meter ladder and had a right lower blunt chest trauma, had referred to a physician in an outpatient center for chest pain. The physician had provided a chest x-ray, and since the pathologic findings had not been seen, he was cleared. After three days, due to increased chest pain, for further investigation, chest Computed tomography was performed.a peripheral-based opacity in the right lower zone, which was not seen in a previous study done three days ago, suggestive of Hampton's hump due to pulmonary infarction was seen on chest CT scan (Plate-1b).

Physical examination findings were as follows; PR: 98, RR: 18, O_2 Sat: 97%, T: 36.7°C, and BP: 170/80 mmHg. A 12—lead ECG, pathologic findings were not observed. In laboratory findings, leukocytosis was not seen. TPI was negative and D-dimer was reported 1200 ng/ml. For the patient was performed CTPA scan. Computed tomography pulmonary angiography confirmed the diagnosis of pulmonary embolism in a right lower lobe segmental branch, pleural effusion with adjacent collapsed lung, consistent with lung infarction (Plate- 1a,b,c). The patient was started on heparin injection and three days later with a good general, he was discharged.

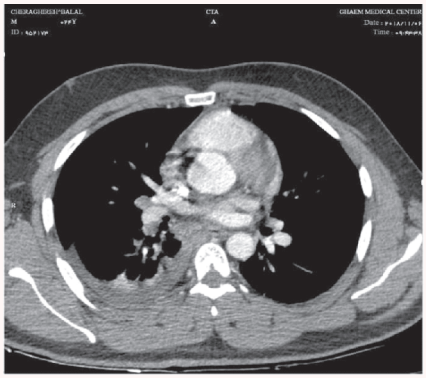

Plate-1 (a): Pulmonary embolism in a right lower lobe segmental branchand coronal (c) computed tomography angiography

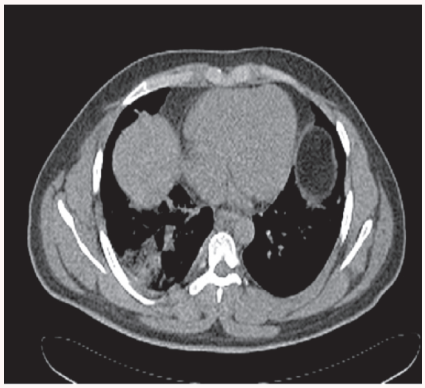

Plate-1 (b): Pulmonary embolisHampton's hump due to pulmonary infarction

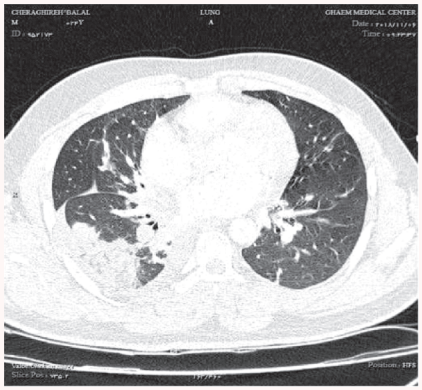

Plate-1 (c): Pleural effusion with adjacent collapsed lung, consistent with lung infarction

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Discussion:

InSoft tissue injury may lead to endothelial damage, which in turn can induce hypercoagulability. Trauma can produce a hypercoagulable state and physicians working in the trauma department should consider pulmonary embolism when dealing with a patient with chest trauma. Symptoms vary widely during PTE, ranging from no symptom to cardiovascular collapse. The patient can feel focal, sharp, pleuritic pain, and exhibit a splinting response to breathing. Over several days, the infarcted segment becomes consolidated on chest radiography and exudes a pleural effusion, manifesting an intense underlying inflammatory process (Attar et al., 1969; Schmidt et al., 1992).

The most common signs and symptoms of PE include dyspnea and tachypnea (Menaker et al., 2007; Lewis et al., 2013). As stated above, our patient only complained of chest pain and did not have tachypnea and dyspnea. However, studies have shown that up to 25% of PE's may occur within the first four days after injury, with one author reporting a diagnosis of PE within several hours after injury (O'Malley & Ross, 1990). In our patient, the symptoms of PE appeared on the third day.

Tachypnea, hypoxia, pleuritic pain within a few days after blunt chest trauma prompt most clinicians to think of atelectasis or pneumonia. Pulmonary thromboembolism must now be added to that differential diagnosis.

In several studies, the incidence of early post-traumatic thromboembolism in trauma-patients who need ICU care reported ranges from 10% to 30% (Geerts *et al.*, 1996).

PE is the third most common cause of death after the first 24 hours of trauma in patients who survive (Geerts *et al.*, 1996). Injury severity, comorbidities, and thrombosis extent are the most common cause of crude mortality in trauma patients (Goldhaber *et al.*, 1999).

Several studies reported that pharmacological prevention is more effective than mechanical devices (Knudson et al., 1992; Knudson *et al.*, 1994). Given that anticoagulant therapy can cause bleeding complications, prophylactic treatment needs further studies (Bahloul *et al.*, 2018).

Conclusively, what is important for the medical staff that works at the trauma center is this- when treating a patient with trauma, consider PE. Although the typical signs and symptoms of a PE may be explained by additional traumatic injuries or may even be absent, PE cannot be excluded.

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