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IMMEDIATE PULMONARY EMBOLISM OR PULMONARY ARTERY THROMBOSIS FOLLOWING BLUNT CHEST INJURY?

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ABSTRACT

Introduction: A pulmonary embolism usually appears as early as two to three days post-trauma, but in rare cases, it can manifest immediately. It may be a result of *in situ* thrombosis instead of dislodged deep vein thrombosis.

Case description: We present a 24-year-old patient involved in a motorcycle and car collision with blunt chest trauma, mild desaturation and a right clavicle fracture. A thorax computed tomography scan seven hours post-trauma revealed bilateral main pulmonary arteries thrombosis extending to the proximal segmental branches with no evidence of deep vein thrombosis or major thoracic injury. The patient was treated with low molecular weight heparin and discharged home after five days with a one-year follow-up.

Discussion: This could be related to an inflammatory cascade secondary to the trauma which led to endothelial damage and formation of a de novo thrombus in the pulmonary vessels.

Conclusion: this case highlights the occurrence of immediate pulmonary embolism after minor blunt chest trauma in the absence of classical risk factors and has been treated successfully by low molecular weight heparin.

KEYWORDS

Pulmonary embolism, pulmonary artery thrombosis, traumatic injury

LEARNING POINTS

- Pulmonary thrombosis may develop within hours of injury – even mild trauma and may occur *de novo*.
- Absence of risk factors (medical or traumatic) does not exclude pulmonary embolism or thrombosis.
- Despite major pulmonary vessel thrombosis, low molecular weight heparin can be used successfully.

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INTRODUCTION

Pulmonary embolism (PE) is an acute respiratory disorder caused by the occlusion of the pulmonary arterial bed. Pulmonary thrombosis refers to a localised clot that obstructs blood flow in the lungs[1]. Based on the timing of occurrence, PE can be classified as immediate, early or late. Immediate PE is diagnosed with the initial computed tomography (CT) scan post-trauma. The literature lacks a concrete definition of early or late; however, PE occurring after four days can generally be classified as late. Early PE, occurring before four days, is rare[2].

PE in trauma represents a perplexing and poorly understood phenomenon. Its underlying mechanisms remain largely obscure, compounded by the clinical complexity of polytrauma patients. Commonly associated thoracic injuries, such as rib fracture, pneumohemothorax, lung contusion and myocardial ischaemia/infarction, coupled with haemorrhagic shock, low Glasgow coma scale score and intubation, further complicate an accurate diagnosis. Several studies have proposed that early onset PE following trauma may result from localised vascular inflammation and endothelial injury within the pulmonary circulation, leading to in situ thrombosis rather than embolic migration from peripheral veins[3-5].

CASE DESCRIPTION

A 24-year-old previously healthy non-smoker male with no comorbidities presented to the emergency department as a motorcyclist struck by a car resulting in a car rollover. His main complaint was chest pain with normal Glasgow coma scale score. He had no history of air travel, prolonged immobilisation or previous admission to the hospital. There was no family history of venous thromboembolism. He had received the COVID-19 vaccine two years previously and never had the infection.

His vitals showed heart rate 111 bpm, 20 br/min, blood pressure of 135/82 mmHg. SpO_2 was low at 92%; he had sinus tachycardia and slight tachypnoea. The patient started desaturating after six hours. He was put on a non-rebreather mask with an oxygen flow rate of 10 l/m. The physical examination revealed bilateral chest wall contusions, equal bilateral air entry and a deformity of the right clavicle confirmed by X-ray as a fracture of the right clavicle. An arterial blood gas test showed low oxygen saturation, and an extended focused assessment with a sonography in trauma scan was negative. Doppler ultrasound scanning for lower limbs was negative for deep vein thrombosis (DVT). CT imaging demonstrated bilateral main pulmonary emboli involving the main pulmonary arteries, extending into the proximal lobar branches *(Fig. 1 and 2)*. His laboratory findings were unremarkable apart from elevated d-dimer value *(Table 1)*. The electrocardiogram was unremarkable except for sinus tachycardia *(Fig. 3)*. Echocardiography revealed a mildly dilated right ventricle functioning at the low end of normal. Septal motion suggested right ventricle pressure overload on the septum. Pulmonary artery pressure was moderately increased with a definite thrombus, measuring 1.6 cm by

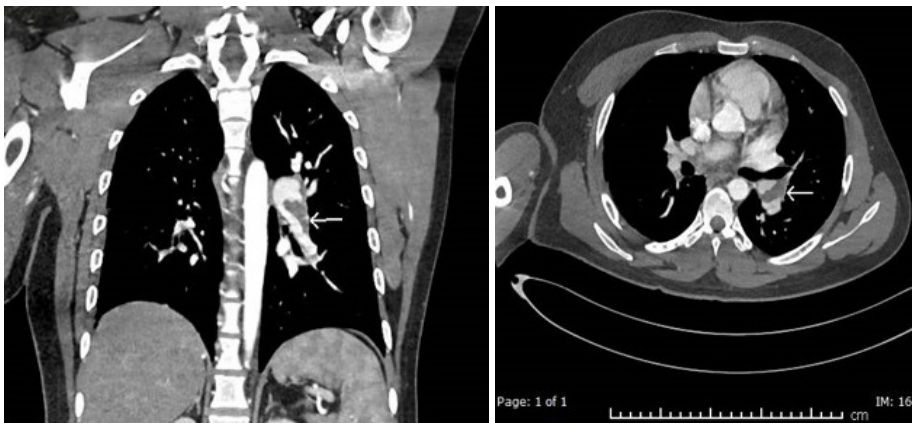


Figure 1. *CT chest with IV contrast showing left main pulmonary artery thrombosis.*

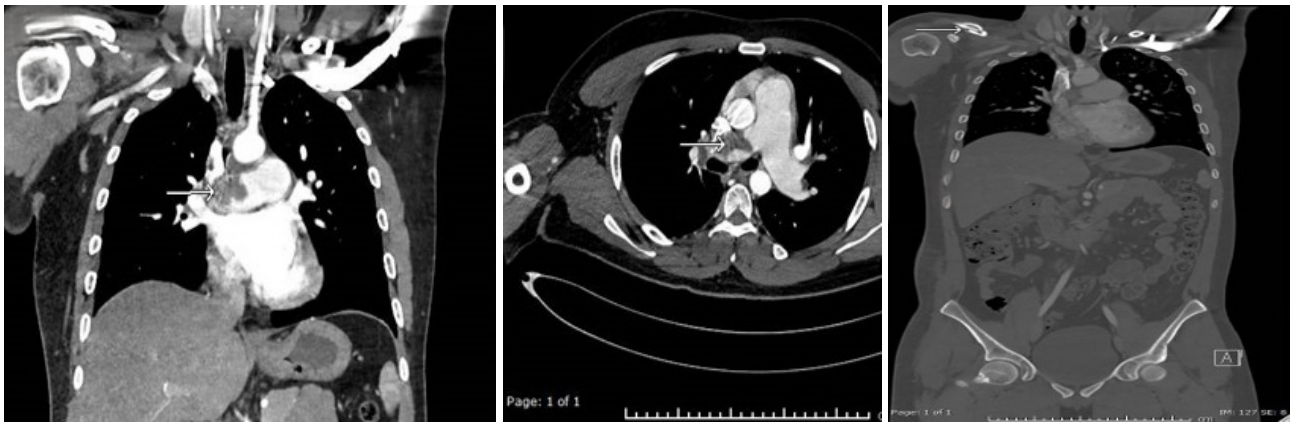


Figure 2. *Chest with IV contrast showing right main pulmonary artery thrombosis, in addition to right clavicle fracture.*

Test	Value
Hs Troponin-T (ng/l)	14
Protein C (%)	91.3
Protein S (%)	75.4
D Dimer (mg/l)	1.02 (normal value: 0.04)
Fibrinogen (gm/l)	1.76
Prothrombin time	15.6
APTT (seconds)	31.5
INR	1.3
Anti cardiolipin Ab IgG Int	Negative
Anti cardiolipin Ab IgM Int	Negative
ATAC (%)	83.7
Anti Xa LWMH (IU/ml)	0.28
ROTEM CFT (seconds)	82
ROTEM MCF (mm)	11
Ph	7.385 (arrival)
PCO2 (mmHg)	39 (arrival)
HCO3 (mmol/l)	21 (arrival)
PO2 (mmHg)	42 (arrival)
SO2 (%)	92 (room air), 87 (on 10 l per minute), 90 (on 4 l per minute)
Base excess	−0.2 (arrival)

Table 1. *Laboratory results.*

Abbreviations: Anti cardiolipin Ab IgG/IgM Int, anti-cardiolipin antibodies immunoglobulin G/immunoglobulin M; Anti Xa, anti-factor Xa; APTT, activated partial thromboplastin time; ATAC, assay for transposase-accessible chromatin; D-dimer, D-domain dimer; HCO_3, hydrogen carbonate (bicarbonate); Hs Troponin-T, high-sensitivity troponin T; INR, international normalised ratio; LWMH, low molecular weight heparin; PCO_2, partial pressure of carbon dioxide; Ph, potential of hydrogen (acidity); PO_2, partial pressure of oxygen; ROTEM CFT, rotational thromboelastometry clot formation time; ROTEM MCF, rotational thromboelastometry maximum clot firmness; SO_2, oxygen saturation.

1.6 cm in the proximal right pulmonary artery. The patient started on a therapeutic dose of low molecular weight heparin (LMWH) enoxaparin 75 mg bid subcutaneously for three days in the intensive care unit (ICU). As his condition improved, he was transferred to the ward. On day 5, the patient was discharged home on rivaroxaban 15 mg oral bid for 21 days, followed by 20 mg once daily, for six months with follow-up in the outpatient clinic. He was followed for one year.

DISCUSSION

We present a rare case of immediate PE diagnosed within seven hours post-trauma. The patient sustained isolated chest trauma without rib fractures, pulmonary contusions or hemopneumothorax, resulting in a low abbreviated injury score. He remained haemodynamically stable and did not require a blood transfusion. Computed tomography pulmonary angiography (CTPA) was performed to rule out chest pathology due to the patient's desaturation. The thrombus was in a major pulmonary vessel rather than in peripheral branches, as is commonly reported. We managed the patient with therapeutic LMWH, eliminating the need for thrombolytic therapy and achieving complete clinical recovery.

Zinoune et al.[3] presented a similar case of immediate PE in a previously healthy male who sustained blunt chest trauma. The patient was diagnosed with PE 20 hours post-trauma. He had no history of prothrombotic conditions or COVID-19. CTPA demonstrated bilateral PE in the segmental branches of the lower lobes, with no evidence of fractures or pneumothorax[3].

PE has mortality rates of up to 50%, making prompt recognition and diagnosis of PE critical to improving patient outcomes[6]. A cohort study demonstrated that not all pulmonary clots detected on the initial chest CT after injury are embolic; they may represent a combination of PE and pulmonary thrombosis[7]. Tissue injury after trauma can contribute to endothelial disruption, which in turn activates coagulation pathways and promotes a hypercoagulable state[8,9]. In a study on early PE, 61% of patients were found to have de novo pulmonary embolism without any evidence of DVT, while 39% exhibited concurrent PE and DVT. Patients with de novo pulmonary embolism were significantly younger and more likely to have rib fractures, pulmonary contusions and peripherally located emboli[9]. Our patient was young and had a lung contusion, but on the contrary had central PE with no rib fracture.

Similarly, Velmahos et al.[4] reported trauma patients who underwent CTPA and CT venography within a week of admission. PE was diagnosed in 19% of the patients, while only 7% showed DVT. The authors concluded that PE may have a unique pathophysiology arising de novo within the pulmonary circulation rather than from peripheral

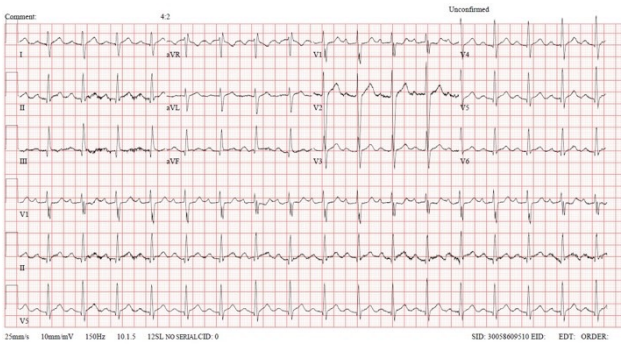


Figure 3. *Electrocardiogram showing sinus tachycardia, SI, QIII and flattening TIII.*

thromboembolism[4]. Knudson et al., after reviewing over 3,700 cases of PE, identified chest trauma as a significant risk for PE that cannot be entirely prevented by mechanical prophylaxis such as leg compression devices or vena cava filters[5].

Literature on PE post-trauma reports that coagulation and fibrinolytic systems are triggered immediately after trauma due to direct tissue injury. The development of in situ pulmonary artery clot formation involves localised inflammation, occult vascular injury and the low-flow state that follows thoracic injury[4]. Pulmonary thrombosis exhibits distinct risk factors compared to those associated with traditional venous thromboembolism. Knudson et al. suggest injury, inflammation and a hypercoagulable state as risk factors of PE post-trauma[7].

Leibowitz stressed the importance of echocardiography in diagnosing pulmonary thromboendarterectomy. In our case, a CT scan and echocardiography both confirmed the diagnosis[1].

CONCLUSION

In conclusion, this case highlights the occurrence of immediate PE after minor blunt chest trauma in the absence of classical risk factors, which has been treated successfully by LMWH.

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