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CASE REPORT

A Fatal Journey of a Clot: Pulmonary Thromboembolism Case Report

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ABSTRACT

Pulmonary thromboembolism (PTE) following deep vein thrombosis (DVT) is associated with high mortality, particularly in trauma patients with long bone fractures. Due to its nonspecific clinical presentation, PTE is often not suspected until autopsy, which serves as a gold standard for diagnosis in such cases. We report the case of a 42-year-old male who died from an acute PTE after sustaining multiple injuries in a road traffic accident. He presented to the emergency department unconscious, with head and leg injuries, and was intubated for a low Glasgow Coma Scale score. Despite intensive care including resuscitation from recurrent cardiac arrests, the patient died on the fourth day of hospitalization. Autopsy revealed extensive deep vein thrombosis in the injured limb and a saddle pulmonary embolus as the cause of death. This case underscores the need for early screening and prophylaxis for PTE in patients with multiple fractures, and it highlights the value of medicolegal autopsy and histopathological examination in confirming the diagnosis and guiding future clinical practice.

KEYWORDS

• Pulmonary thromboembolism • Road traffic accident • Forensic pathology • Autopsy

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INTRODUCTION

Pulmonary thromboembolism (PTE) following DVT carries a high mortality rate, especially in patients immobilized by long bone fractures.[1] The clinical presentation in the emergency setting is often nonspecific, resulting in a broad range of differential diagnoses.[2] According to one report, the mortality rate of untreated PTE ranges from 30 – 35 %.[3] Indian data from a tertiary healthcare center documented an incidence of 22 PTE cases per 10,000 hospital admissions, with a mortality rate of 49.5% among those patients.[4] The rapid progression of this disease and delayed clinical suspicion make autopsy a crucial quality control measure as well as the gold standard for diagnosing PTE. Although an autopsy cannot benefit the deceased, it can help prevent similar deaths in the future by alerting clinicians to this complication. Here, we report a case of PTE in a patient who developed DVT due to multiple lower-limb fractures sustained in a road traffic accident. This case emphasizes the importance of autopsy and histopathological examination in prompting early suspicion of PTE in such trauma cases and in guiding modifications to treatment protocols.

Case Report

A 42-year-old man was involved in a high-speed road traffic accident and succumbed to his injuries four days later in the hospital. He had been admitted unconscious to the emergency department with severe head trauma, a visibly shortened right leg (suggestive of a femur fracture), and bleeding from the nose. The injuries occurred when he was struck by a fast-moving two-wheeler while crossing the road, and he was immediately transported to the hospital. On arrival, the patient was assessed by the trauma team, intubated, and managed with broad-spectrum antibiotics, intravenous fluids, blood transfusions, and inotropic support. He had no significant prior medical or family history. During his hospital course, he suffered multiple episodes of cardiac arrest, each successfully resuscitated with high-quality cardiopulmonary resuscitation (CPR). In the early morning of the fourth day post-admission, he sustained another cardiac arrest and could not be revived. According to the death summary, the final diagnoses included traumatic brain injury; bilateral lung contusions; fractures of the right femur, tibia, and fibula; fracture of the left maxilla; fracture of the base of the right temporal bone; and sepsis with shock. A medicolegal autopsy was then conducted to determine the exact cause of death.

Autopsy Findings

A complete autopsy was conducted approximately nine hours after death. The body was moderately built and nourished. Rigor mortis was in the developing stage in the upper body. The conjunctivae and oral mucosa were congested, and the fingernail and toenail beds were cyanotic. Greenish fluid stains around the mouth and nostrils and on the clothing indicated postmortem purge of gastric contents. Multiple reddish-brown abrasions with scabs were present on the face, involving both sides of the forehead, the nose (alae and tip), the chin (symphysis menti), and the area lateral to the left eye. Palpation of the face revealed crepitus over the right maxillary region, indicating an underlying facial bone fracture. Several other abrasions were present on the body. Two sutured lacerated wounds were noted: one on the left frontal scalp (frontal eminence) and one on the right index finger at the middle phalanx. The right thigh was markedly swollen at an oblique fracture of the right femur (~23 cm below the hip). The right lower leg showed abnormal mobility and localized swelling about 34 cm below the knee, consistent with fractures of the tibia and fibula. Internal examination of the head revealed a subscalp contusion and pericranial hemorrhage over the left temporoparietal region, with underlying contusion of the temporalis muscle. An undisplaced comminuted fracture (3 × 1cm) was present in the left squamous temporal bone. The brain weighed 1480 g and was congested and edematous. A thin film of diffuse subarachnoid hemorrhage with patchy subdural hemorrhage covered the bilateral cerebral hemispheres and cerebellum. On sectioning the brain, multiple petechial hemorrhages were noted in the left frontal lobe.

Examination of the thoracic cavity revealed a massive pulmonary thromboembolism. Opening the heart (using Ghon's en bloc removal method) exposed multiple coiled blood clots filling the right atrium and ventricle, including a saddle-shaped thrombus lodged across the pulmonary artery and extending into both pulmonary branches (*Figure* 1A). This thrombus effectively obstructed blood flow to the lungs. The heart valves and coronary arteries were intact, and the heart weighed 325 g. Numerous petechial hemorrhages were present on the pleural surfaces of both lungs. On slicing the lungs from the hilum to the periphery, multiple clots were found adherent within the pulmonary arteries. The lung parenchyma was markedly congested on cut surface, and the lungs weighed 725 g (right) and 650 g (left). The deep veins of the lower extremities were also dissected. A continuous thrombus was found in the inferior vena cava, extending through the right common and external iliac veins into the right femoral vein (*Figure* 1B). Histopathological examination was performed on tissue sections from the lungs and the retrieved thrombi. Hematoxylin and eosin staining of the lung tissue showed acute bronchopneumonia with extensive pulmonary edema (*Figure* 2B). Microscopic examination of the thrombi revealed alternating dark-red and pale layers (lines of Zahn) in clots from the pulmonary arteries, hilar vessels (*Figure* 3A), right ventricle, inferior vena cava, and external iliac vein confirming that these thrombi formed antemortem. These microscopic features, illustrated by the layered appearance of one clot (*Figure* 3B), indicate organization of the thrombus prior to death. No significant pathology was found in the remaining organs. Based on the autopsy findings, the cause of death was attributed to multiple injuries to the head and right lower limb and their complications chiefly a fatal pulmonary thromboembolism consequent to blunt force trauma.

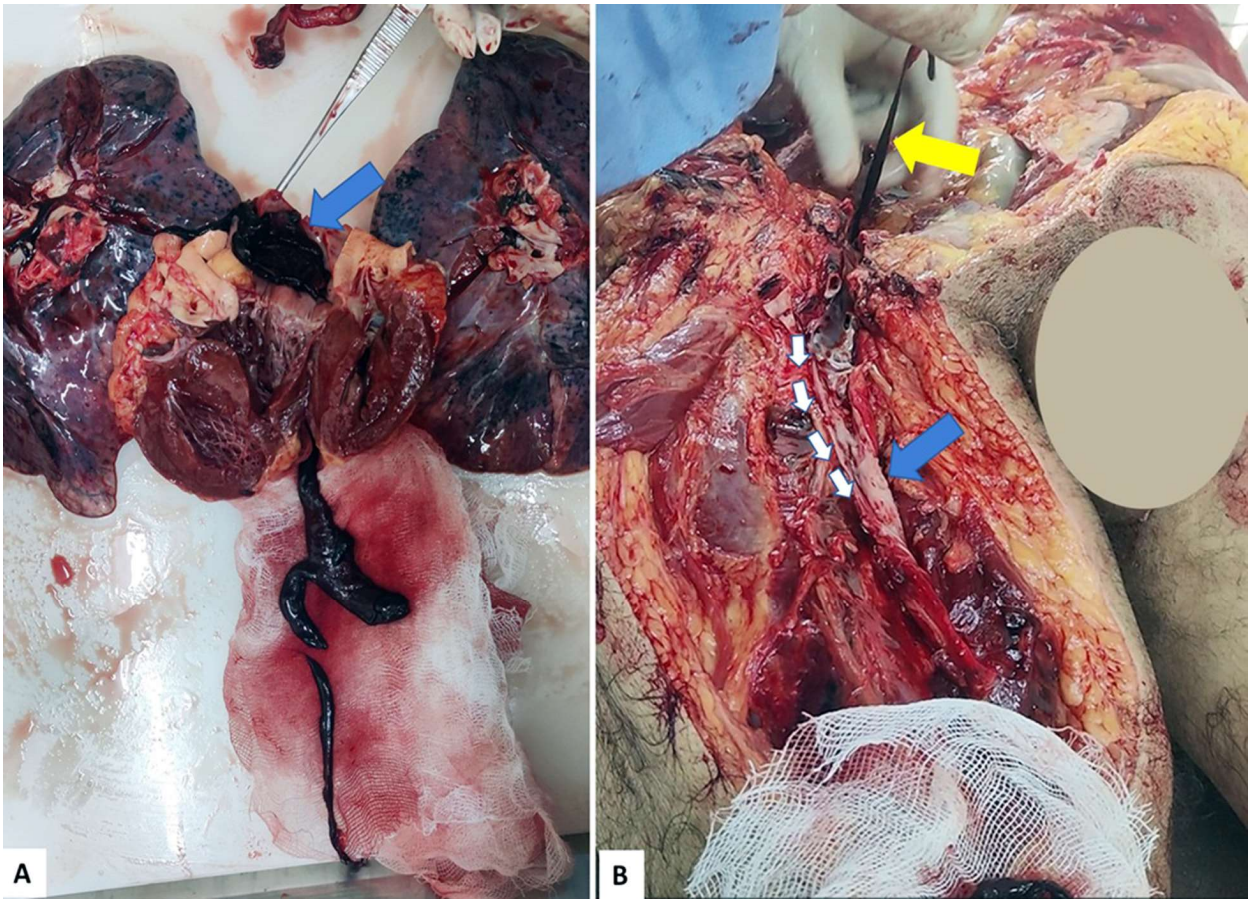

Figure 1: (A) Coiled clots filling the right ventricle and extending into the pulmonary arteries of both lungs (saddle embolus), along with clots retrieved from the inferior vena cava and common iliac vein, **(B)** Dissected right femoral vein (blue arrow) with adherent clots being lifted (yellow arrow) from the vessel during autopsy.

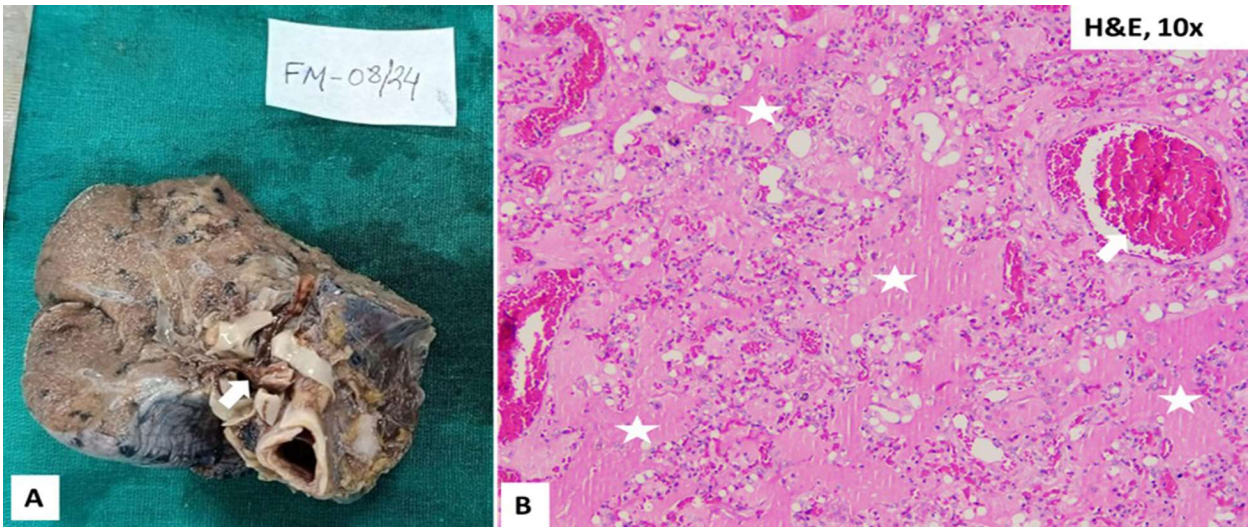

Figure 2: (A) Formalin-fixed lung specimen showing a clot within the pulmonary artery (white arrow indicates a clot extending from the right ventricle), **(B)** Histologic section of lung parenchyma (H&E stain) showing extensive pulmonary edema (white star) and congested blood vessels (white arrow), with areas of interstitial hemorrhage.

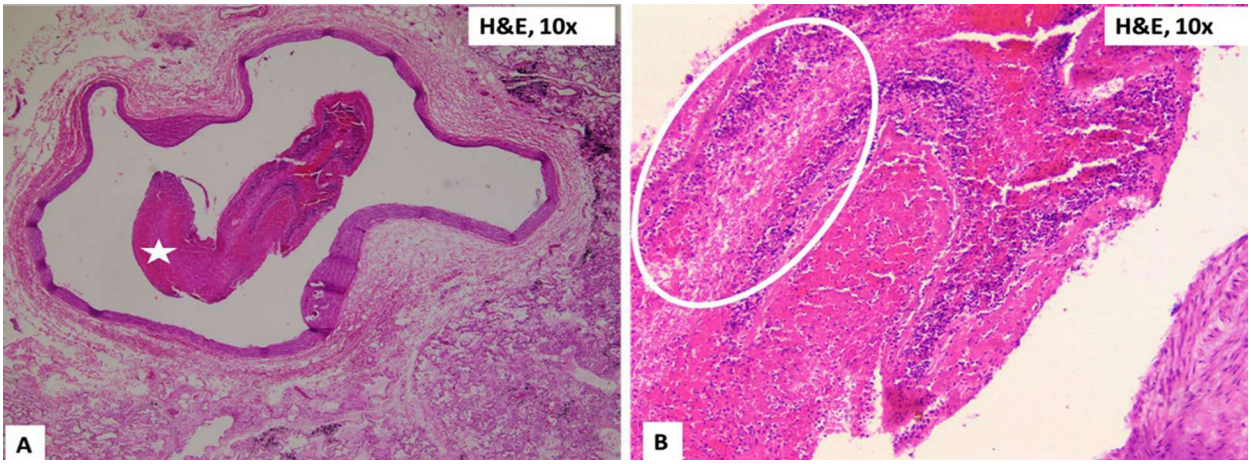

Figure 3: (A) Histologic section of a hilar pulmonary vessel showing an occlusive blood clot (white star), **(B)** Cross-section of a thrombus showing distinct dark-red layers (rich in red blood cells) and pale layers (fibrin and platelets), demonstrating the lines of Zahn indicative of an antemortem thrombus (white oval highlight)

DISCUSSION

PTE is the occlusion of the pulmonary artery by a thrombus that has traveled (embolized) from another site through the bloodstream. The classic risk factors for thrombosis are described by Virchow's triad: stasis of blood flow, endothelial injury, and hypercoagulability. The causes of PTE can be broadly categorized into abnormalities of the vessel wall (e.g., inflammation or atherosclerosis), abnormalities of blood flow (e.g., trauma-related stasis due to immobilization), and coagulation abnormalities.[5] The present case exemplifies an abnormality of blood flow, as prolonged immobilization from multiple fractures in one limb led to venous stasis and thrombosis. In this patient, the episodes of cardiac arrest during hospitalization can be explained by the extensive pulmonary arterial clots found at autopsy, (*Figure* 1A) which critically impeded blood flow through the lungs. Acute PTE increases pulmonary arterial pressure and acutely raises right ventricular afterload. This sudden strain causes dilation of the right ventricle and stretching of the myocardial fibers, triggering neurohumoral activation and arrhythmias. Clinically, cardiac biomarkers such as troponin and N-terminal pro–B-type natriuretic peptide (NT-proBNP) can aid in early identification of PTE-induced right ventricular strain.[6,7] Elevated levels of natriuretic peptides have been emphasized in

the European Society of Cardiology guidelines as objective evidence of acute cardiac dysfunction.[8]

Rapid bedside imaging can also expedite PTE diagnosis. Thakkar *et al*. highlighted the utility of point-of-care ultrasound (POCUS) in trauma settings to quickly detect signs of pulmonary embolism, enabling prompt intervention to prevent arrhythmias and reduce mortality.[9] In patients with multiple long bone fractures, it is recommended to utilize early diagnostic modalities such as D-dimer assays, NT-pro BNP levels, echocardiography, compression ultrasonography of the limbs, and CT pulmonary angiography to screen for PTE during the initial evaluation. Implementing these tests early could save crucial time in diagnosing a developing thromboembolism.[2] Despite these measures, many PTE cases are only confirmed at autopsy. Autopsy remains the definitive diagnostic method for PTE, especially when clinical suspicion is delayed.[10] A thorough medicolegal autopsy in suspected PTE cases is not limited to finding the lodged thromboembolus; it also involves assessing risk factors, performing meticulous dissection, conducting ancillary investigations (e.g., histology), and correlating findings with the clinical scenario. In the present case, the patient was bedridden for four days with only supportive care. Immobilization is a well-known risk factor for PTE in patients with multiple fractures. Beasley *et al*. reported a fatal PTE associated with prolonged immobility (working at a computer for ~18 hours without moving) and introduced the term "e-thrombosis" for this 21st-century variant of venous thromboembolism. This underscores that prolonged immobilization, even outside of traditional risk settings, can lead to thrombosis (as also seen in our trauma patient).[11]

Identifying the source of fatal emboli is another important aspect of autopsy. Rivers Kennedy *et al*. stressed the need to dissect both lower limbs including the contralateral, uninjured side in cases of fatal PTE. In their series, the majority of patients had deep vein thrombosis in the same limb that had orthopedic injuries, but a significant number had DVT in the opposite or both legs when fully examined.[12] In our case, we explored only the injured (right) leg and found thrombus extending up to the femoral vein at the fracture site. We acknowledge that not examining the contralateral limb is a limitation of this case, as an undetected DVT in the uninjured leg cannot be ruled out. Ancillary studies can provide additional insights into PTE deaths. Histopathological analysis, in particular, plays a pivotal role in determining the chronology of thrombus formation.[13] Fineschi *et al*. described three histological phases of thrombus organization that can aid in estimating the age of a thrombus. In the first phase (days 1–7), fresh thrombi show the lines of Zahn layered deposits of fibrin and platelets alternating with red blood cell layers. The second phase (approximately 1–8 weeks) is characterized by the presence of hemosiderin-laden macrophages and fibrinous transformation of the clot. The third phase (>2 months) features a fully organized thrombus with neovascularization.[14] The microscopic findings in our case (notably the prominent lines of Zahn) correspond to the first phase, consistent with the fact that the fatal thrombosis developed only a few days after the injury.

Advances in postmortem imaging have also been explored for PTE. Burke *et al*. investigated the use of postmortem CT angiography (PMCTA) to diagnose PTE and found that it could reliably detect large saddle emboli. They concluded that PMCTA is a promising adjunct for identifying massive PTE, although smaller, segmental emboli were better detected by traditional autopsy dissection.[15] In summary, the optimal approach to diagnosing PTE at autopsy is multidisciplinary. Careful gross examination and dissection, supported by targeted histopathology and adjunct techniques like postmortem imaging, can together provide a comprehensive understanding of the pathophysiology and timing of fatal PTE events.

CONCLUSION

This case underscores the importance of early screening for pulmonary thromboembolism in severely injured patients, especially those with multiple long bone fractures, during the initial days of hospitalization. PTE in such patients can present with sudden, nonspecific, and unexpected deterioration owing to the rapid progression of thrombosis. We also highlight the critical role of a thorough medicolegal autopsy including detailed histopathological examination in determining the presence and timing of thrombus formation, an aspect

often neglected in routine forensic practice. The lessons learned from this case may help improve acute trauma care by prompting proactive measures against PTE, and also enhance autopsy protocols for investigating similar deaths in the future.

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