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Case Report

A pulmonary embolism secondary to fracture tibia:a clinical case report

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ABSTRACT

Pulmonary thromboembolism (PTE) occurs when a thrombus, typically originating from venous thromboembolism, obstructs the pulmonary arteries, disrupting blood flow to the lungs. The thrombus often becomes lodged at the bifurcation of the main pulmonary artery or within the lobar branches, causing hemodynamic instability. Although the thrombus most commonly originates in the lower extremities as deep vein thrombosis (DVT), it can occasionally arise from the pelvic veins, renal veins, upper extremity veins, or the right side of the heart. Various factors can contribute to the development of deep vein thrombosis, but in the context of long bone fractures, it Deep vein thrombosis (DVT) typically develops 7 to 10 days after an injury. However, in rare cases, pulmonary thromboembolism (PTE) has been observed within 72 hours of the trauma. Several theories have been proposed to explain the mechanisms behind the early onset of PTE in such instances. Presenting a case which involves a 34-year-old male who developed pulmonary thromboembolism (PTE) following a roadside accident, and had segmental closed fracture of the right tibial shaft.

Keywords: Deep vein thrombosis, Tibial fracture, Thromboembolism, Virchows triad

INTRODUCTION

Pulmonary embolism (PE) occurs when a thrombus blocks blood flow in a pulmonary artery, usually after it travels from a vein in the lower limb. The annual incidence of PE is estimated to range from 60 to 120 cases per 100,000 people.

Patients with long-bone fractures have a higher risk of developing PE early after the injury.[1] In 1856, Virchow described the classic triad of venous stasis, endothelial vascular damage, and hyper coagulability as key contributors to the development of deep vein thrombosis (DVT).

Given that venous stasis is considered the primary factor in DVT formation, and subsequently pulmonary embolism (PE) in trauma patients, it has traditionally been believed that these events typically occur five to seven days following the injury. Pulmonary embolism (PE) can be classified into three types: acute, subacute, and chronic.

Acute PE

Symptoms appear suddenly and can be challenging to diagnose. Acute PE typically requires urgent treatment, including thrombolytic therapy (clot-busting drugs) and anticoagulants.

Subacute PE

Symptoms develop gradually over a period of 2 to 12 weeks and are often more subtle, making diagnosis more difficult. Sub-acute PE carries a higher risk of mortality compared to acute PE.

Chronic PE

Symptoms progressively worsen over time, potentially leading to complications like heart failure. Chronic PE occurs when a small obstruction persists after an initial acute PE.

This case present to you show that even performing a simple nailing in fracture shaft tibia fracture one should be careful, pre op. Consideration of PTE and DVT should be done, necessary steps like venous colour Doppler imaging, echocardiography, D-dimer testing, and serum troponin I should be performed.

CASE REPORT

A patient age 34 years male adult came to our emergency department with complaint of pain and swelling rt leg since, 1 day. He had met a road side accident 1 day back. On examination there was gross swelling over rt leg with multiple blisters and there was gross deformity of the limb.

X ray was done. X-ray confirmed fracture both bone of right leg. Below knee slab was applied and was admitted. Next day further investigation was done and patient was posted for surgery. External fixator was applied and he was advised for definitive surgery after 15 days.

After 15 days he came back for definitive surgery. During surgery while we were inserting the nail patient felt breathlessness, chest pain and suddenly there was drop in pulse and after some time no vitals could be assessed.

He was revived by CPR. The surgery was continued. During closure he again developed same features. He was pulse less and his could not be monitored for more than 10 minutes. He was revived by CPR again then he was intubated and shifted to ICU.

He was advised for CT pulmonary angiography, HRCT Thorax, and NCCT HEAD And report showed "cute pulmonary thromboembolism of segmental branches of B/L pulmonary arteries with? pulmonary infarct", NCCT head was normal. He was in ICU for 5 days and was in ventilator for 2 days. He was discharged after 7 of surgery.

Figure 1: (a) Pre operative skin condition of leg (right); (b) pre operative X-ray of leg (right).

Figure 2: (a) Post operative condition of leg external fixator; (b) post operative X-ray.

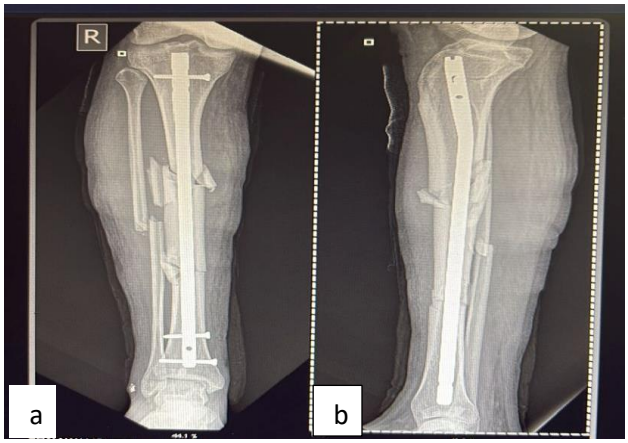

Figure 3 (a and b): Final X-ray after definitive surgery.

DISCUSSION

During the past 3 decades, the incidence of colorectal Pulmonary embolism occurs in 1-5% of patients with tibial fracture. we normally don't practice duplex imaging in our routine for lower leg fractures. But as we show in our case it seems it has its own importance. Performing duplex should be mandatory for the patient with fractures of lower limb specially when there is gross swelling of the limb and you are planning for 2 stage surgery (external fixator-nailing/plating).

Long bone fractures can lead to various complications, including fat embolism, shock, compartment syndrome, deep vein thrombosis (DVT), pulmonary thromboembolism (PTE), disseminated intravascular coagulation (DIC), impaired wound healing, coagulopathy, and infection. Among these, pulmonary thromboembolism is particularly noteworthy as a leading cause of mortality in individuals with long bone fractures.

The majority of complications resulting from long bone fractures, approximately 99%, are typically manageable.

However, in about 1% of cases, these complications can be fatal. Causes of death associated with bone fractures can be grouped into five categories: cardiovascular, infectious, respiratory, malignancy-related, and miscellaneous causes. Cardiovascular causes include myocardial infarction, heart failure, and cerebrovascular accidents. Infectious causes often involve conditions such as sepsis and pneumonia. Respiratory causes include pulmonary thromboembolism and respiratory failure. Other causes involve renal failure and multiple organ failure.

The mortality rate in cases of bone fractures is influenced by factors such as gender, smoking habits, body mass index (BMI), congestive heart failure, dementia, moderate to severe renal disease, and history of a malignant tumor. The incidences vary from 0.5% to 0.7%. This, variability can be attributed to differences in population characteristics, the severity of injuries, and screening protocols. The computed tomography pulmonary angiogram (CTPA) is regarded as the gold standard for diagnosing PTE, offering high predictive value and detailed visualization of the pulmonary arteries.

CTPA is favored for its superior diagnostic accuracy, enabling precise identification of the location and extent of clots, which aids in treatment planning and patient risk stratification. Historically, screening for asymptomatic pulmonary thromboembolism was not feasible. However, recent research, including a study by Schultz, has revealed a higher prevalence of occult PTE than previously recognized.[1] Schultz reported a 24% incidence of asymptomatic PTE in a cohort of 90 moderately-to-severely injured trauma patients using systematic contrast-enhanced helical CT scanning Advances in CT technology, particularly the development of multi-detector scanners, have significantly enhanced the accuracy of PTE detection.[2,6]

Additional investigations for the workup of pulmonary thromboembolism (PTE) include chest X-ray, ECG, arterial blood gas analysis, the 2-level PE Wells score, bedside echocardiography, color Doppler ultrasound of the leg veins, D-dimer testing, and serum troponin I measurement. Assessing Tissue Plasminogen Activator (tPA) 20 resistance can provide early indications of post-traumatic PTE. Factors such as fibrinolysis shutdown and tPA resistance further increase the risk of early PTE. Detecting a hypercoagulable 20 state through tPA resistance testing within 12 hours of admission is critical, offering a window for timely intervention and management to mitigate the risk of PTE.

The management of pulmonary embolism includes anticoagulation therapy, thrombolytic treatment, surgical interventions, and the use of caval filters. Prophylactic measures to prevent PTE involve the administration of anticoagulants such as heparin, low-molecular-weight heparins (LMWH), warfarin, and apixaban.[7] As mentioned earlier, long bone fractures significantly increase the risk of early pulmonary thromboembolism (PTE). Traditionally, PTE was thought to occur most frequently between days 5 and 7 following trauma, with occurrences before day 4 considered rare.[2-5] This belief was based on the conventional understanding that post-traumatic PTE primarily arises from deep vein thrombosis (DVT) in the lower extremities and pelvis stasis, a key factor in DVT formation, was thought to delay the development of PTE until 5 to 7 days after the injury.[2-7]

A review of the literature indicates that lower limb fractures are commonly associated with early pulmonary thromboembolism (PTE).[1,5,6] Several risk factors impact the timing of post-traumatic pulmonary thromboembolism (PTE), including advanced age, obesity severe injury characteristics, compromised hemodynamic status, sepsis, and hypoxemia.[5,10-12,14,15]In this case, pulmonary thromboembolism was identified as the immediate cause of death, with histopathological examination revealing thrombi in the pulmonary arterioles. Post-traumatic pulmonary thromboembolism is a life-threatening condition that typically develops within a week of a long bone fracture, and in some cases, within 72 hours.

However, in this particular instance, pulmonary thromboembolism occurred within 6 hours of the trauma, which is considered rare. Early pulmonary thromboembolism is linked to risk factors such as a history of surgery, hospitalization, pregnancy, cancer, and a family history of blood clots, including Factor V Leiden. The occurrence of early post-traumatic pulmonary embolism (PTE) is influenced by factors related to the fracture's location and the patient's individual characteristics.

Several pathophysiological mechanisms contribute to the early onset of PTE. These include undiagnosed congenital or acquired prothrombotic disorders, hypercoagulable 20 states within the initial four days following trauma, the presence of pulmonary clots, the independent risk posed by long bone fractures, and molecular processes associated with the fracture that led to the formation of new thrombi in the pulmonary circulation. Together, these factors significantly increase the risk of early PTE in patients with long bone fractures.

The adrenergic response to trauma may also contribute to early pulmonary thromboembolism (PTE) by inducing vascular endothelial inflammation and promoting the synthesis of circulating adhesion molecules, which facilitate thrombosis and rapid vascular occlusion. Risk factors for PTE include obesity, advanced age, sepsis, acute renal failure, and congenital prothrombotic disorders. Early PTE is more frequently observed in cases involving a higher incidence of lower extremity fractures and severe extremity trauma.[17-19,22]

In summary, the pathophysiology of PTE is multifaceted. The "lethal triad" of hypothermia, acidosis, and coagulopathy creates a prothrombotic environment. Systemic inflammatory response syndrome (SIRS) and

compensatory anti-inflammatory response syndrome (CARS) contribute to endothelial dysfunction, further promoting thrombosis. Stress mechanisms enhance hypercoagulability by increasing platelet activation and aggregation. The primary hit mechanism initiates clot formation, while the secondary hit exacerbates the prothrombotic state. Disseminated intravascular coagulation (DIC) also plays a role, with widespread activation of coagulation leading to microthrombi formation and subsequent PTE.

CONCLUSION

This case shows that every trauma Patient specially fracture of lower leg should be examined, investigated and treated (surgery) properly, e.g., even simple nailing should not be performed before knowing their vascular status of the limb. Various methods like duplex imaging, echo etc should be done. The acute onset of pulmonary thromboembolism (PTE) may result from a hypercoagulable state triggered by the multi-site fracture, potentially acting as a physiological response to minimize excessive blood loss. However, the imbalance in the coagulation-anticoagulation system proved harmful. So, comprehensive screening for PTE should be prioritized in patients with long bone fractures, along with the implementation of proactive strategies to reduce the risk of its occurrence.

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