

Metatarsal Fracture Leading To Massive Pulmonary Embolism

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Abstract

Immobilization and bed rest after fracture and orthopedic surgery are routinely advised protocol. Period of bed rest usually depends on the type of injury and orthopedic procedure, ranging from few days to weeks. The trauma, surgery, and immobilization with other contributing factors can lead to deep vein thrombosis and pulmonary embolism (PE) in these patients. Although there is high incidence of PE in such patients, it is difficult to diagnose, primarily because of the variety of nonspecific signs and symptoms. Here, we discuss a case of a 30-year-old female, who had suffered a trivial roadside accident leading to metatarsal bone fracture and later on presented in emergency with seizures, pulmonary edema, and cardiac arrest, after immobilization of just 5 days which was diagnosed to be result of massive PE. Here, we will discuss the pathophysiology, risk factors, and management of massive PE.

Keywords: Fracture, metatarsal fracture, pulmonary embolism, thrombolysis

INTRODUCTION

Venous thromboembolism (VTE) is common after traumatic injuries. However, life-threatening massive pulmonary embolism (PE) after a metatarsal fracture is quite rare.^[1] Massive PE is defined as acute PE with sustained hypotension, pulselessness, or persistent profound bradycardia.^[2] It is treated with thrombolysis given no other risk factors are present. We discuss a case of massive PE culminating in cardiac arrest which was successfully treated with thrombolysis.

CASE REPORT

A 30-year-old female presented to the emergency department with fits, frothing from mouth, and loss of consciousness. On examination, patient's radial pulse was feeble, blood pressure, and oxygen saturation were not recordable. The extremities were cold and clammy. Due to sudden cardiovascular collapse in the emergency room, patient was intubated after cardiopulmonary resuscitation. Vasopressors were started immediately, following a fluid bolus. After patient's resuscitation, her cardiac examination revealed tachycardia with presence of third heart sound and right ventricular (RV) heave. Pulmonary examination revealed bilateral basal crepitation. Her internal jugular veins were also dilated. The

initial electrocardiogram (ECG) presented sinus tachycardia at a rate of 180/min, with right bundle branch block and wide QRS complex of 151 ms, along with right axis deviation of 110 degrees. Diffuse ST segment depression and T wave inversion were also present. After patient's stabilization, she was shifted to intensive care unit on vasopressor and ventilator support. Early echocardiogram of the patient revealed severe RV dilatation with severe hypokinesia of RV free wall and the ventricular septum, but RV apical contraction was preserved. The left ventricular cavity was obliterated, but there was no evidence of wall motion abnormality. Pulmonary artery pressure was also increased, estimated to be 50 mmHg (assuming the right atrial pressure of 15 mmHg), and a minimal pericardial effusion was also present. At this time, patient was diagnosed as a case of acute PE. Detailed history revealed that patient was being conservatively managed at home for fracture of the first metatarsal, after meeting with a roadside accident. Although the patient kept on complaining of a sick feeling and had severe bouts of sweating in the following days, for

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which she was severely depressed, she did not visit hospital until late. Further history revealed that she was also using oral contraceptive pills. A computed tomogram (CT) of the chest confirmed large PE with radiological evidence of RV strain. A noncontrast CT head was done to rule out any intracranial bleeding. All other investigations were completely normal, including complete blood count, coagulation profile, and basic biochemistry panel except CT pulmonary angiography which was suggestive of massive PE causing 90%–95% luminal compromise in lower lobe and segmental branches of the left pulmonary artery [Figure 1]. The patient was then thrombolysed with injection tenecteplase 30 mg iv bolus over 5 s along with injection reteplase 10 mg iv over 2 min followed by 10 mg after 30 min to a total dose of 20 mg with continuous hemodynamic monitoring. The patient was maintained on mechanical ventilation for next 24 h and extubated after improvement of the symptoms.

On 7th day, patient was discharged for home, on oral anticoagulants.

DISCUSSION

Pulmonary embolism is a potentially fatal condition which if not managed aggressively, has a grim prognosis. PE is differentiated into massive, submassive, and low-risk PE depending on the hemodynamic instability and associated factors.^[2]

Thrombus formation is influenced by three factors: blood coagulation, venous stasis, and endothelial injury—the Virchows triad.^[3] Factors leading to PE include hypercoagulable states, immobilization, surgery, trauma, pregnancy, oral contraceptives, hereditary factors, and medical illnesses, including cancers.^[4] VTE is usually associated with major trauma and major orthopedic surgery, with massive PE occurring in even fewer cases (0.3% after total hip replacement). Although small, chances of PE are still present with a bone fracture, when it leads to immobilization. Even fracture of metatarsal can lead to PE as is being reported by us.^[5]

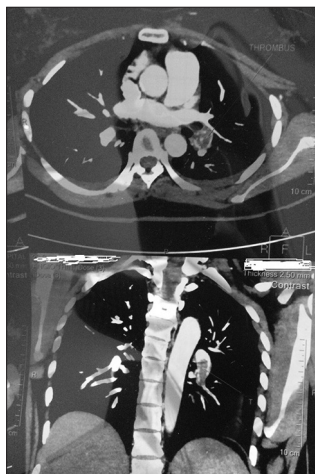


Figure 1: Computed tomogram angiography showing pulmonary thrombus

Undisplaced metatarsal shaft fractures are usually managed conservatively with a soft, padded elastic dressing or immobilization in a posterior splint, with early weight bearing with the help of crutches, followed by usual follow-up in 3–5 days.^[6] In our case, same management was followed, but the patient's prolonged immobilization along with her regular use of oral contraceptive pill's must have led to PE development. Although the American College of Chest Physicians suggests no prophylaxis in patients with isolated lower-leg injuries requiring leg immobilization, NICE recommendations may prove fruitful in such cases (decreased mobility >3 days and use of estrogen-containing contraceptive pills), where they recommend offering pharmacological VTE prophylaxis to patients until the removal of lower limb plaster cast.^[7,8] Throughout the world, especially in India, there is significant underutilization of thromboprophylaxis in orthopedic cases despite being strongly associated with VTE.^[9]

Vague presenting symptoms make PE difficult to diagnose. The symptoms range from shortness of breath, cough, wheeze, seizure, syncope, loss of consciousness, and new onset of atrial fibrillation to sudden hemodynamic collapse.^[10,11] Therefore, a high index of suspicion is required for diagnosing PE, particularly in patients with risk factors, when respiratory symptoms could not be explained by alternative diagnosis.

The physical signs in patients of PE include dyspnea, tachypnea, pleuritic chest pain, rales, tachycardia, accentuated S₂, S₃, or S₄ gallop, cardiac murmur, and cyanosis. Massive PE presents with hypotension, tachycardia, tachypnea, and impaired mentation. Signs of pulmonary hypertension, i.e., palpable impulse over the second left intercostal space, loud P₂, RV S₃ gallop, and a systolic murmur louder on inspiration at the left sternal border may also be present.^[12] Our patient presented with seizure followed by sudden cardiovascular collapse suggestive of massive PE, after which she was resuscitated and treated accordingly.

Computed tomography angiography is the investigation of choice for suspected PE, and same revealed a 90%–95% luminal compromise in the left pulmonary artery.^[13] Other diagnostic modalities include pulmonary angiography, multidetector-row CT angiography, contrast-enhanced CT, ventilation-perfusion scanning, and magnetic resonance imaging. Various laboratory investigations such as D-dimer, antithrombin III, protein C, protein S, lupus anticoagulant, and homocysteine are done.^[14] ECG and chest X-ray though nonspecific, help in excluding other causes of hypoxia and dyspnea. Transesophageal echocardiography is an important prognostic tool with significant correlation being found with regional wall hypokinesia and RV dysfunction.^[15]

Management includes anticoagulation which is done by low-molecular-weight heparin and fondaparinaux starting from the time of diagnosis of PE. Warfarin, direct thrombin inhibitors, and factor Xa inhibitors such as dabigatran are used for prophylaxis and treatment of PE with target international normalized ratio of 2.0. Thrombolysis is considered in massive

or submassive PE with hemodynamic instability through reteplase and alteplase.^{16]} Our patient was treated with injection teneceplase and injection reteplase without any evidence of bleeding.

Anticoagulation is continued for at least 3 months which can be extended in the presence of risk factors.

CONCLUSION

A fracture, however small, compounded with comorbidities may lead to a PE, which if detected late, may have grim prognosis. Hence, thromboprophylaxis should be considered in this scenario.

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Conflicts of interest

There are no conflicts of interest.

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