Thrombolysis in a Case of Cardiac Arrest due to Massive Pulmonary Embolism with Ongoing Cardio-Pulmonary Resuscitation: a Case Report

Dr Shweta Tyagi[1], Dr Prashant Mishra[2], Dr Mehul S. Shah[3]

Abstract

Pulmonary embolism (PE) is a life-threatening condition which often gets missed due to non-specific initial presentation in the Emergency Department (ED). We report a case of a young male who presented to the ED with sudden onset breathlessness and diaphoresis. Appropriate resuscitation was initiated. Soon after patient started gasping and had a cardiac arrest with first monitored rhythm being pulseless electrical activity (PEA). Cardio-pulmonary resuscitation (CPR) was initiated and continued as per ACLS protocol. Portable Echocardiograph revealed severe pulmonary hypertension with right ventricular dysfunction. Provisional diagnosis of PE was made and patient was managed with immediate thrombolysis with ongoing CPR. Brief episodes of cardiac arrest occurred which were successfully revived following which he remained hemodynamically stable. Diagnosis was confirmed by Colour Doppler of bilateral lower limbs and pulmonary angiogram. Patient was weaned off ventilator within 24 hours and discharged home after a week with full neurological recovery. Timely diagnosis and management were key in this patient's survival.

Keywords: Pulmonary embolism, cardiac arrest, thrombolysis

Introduction

PE is an acute and critical presentation in the ED which requires high index of suspicion for accurate diagnosis and prompt initiation of management. Literature reports a mortality of around 30% in cases of massive PE and when PE is associated with cardiac arrest then the mortality rises to up to 95%.[1]

Case Report

Our patient was a 32-year-old male with no significant past medical history, who presented to the ED with sudden onset breathlessness and diaphoresis. Initial evaluation revealed tachycardia (Heart Rate: 120/min), tachypnea (Respiratory Rate: 40/min), hypoxemia (Saturations: 88% on room air) and hypotension (Blood Pressure: 88/60 mmHg). He was diaphoretic and responding to verbal commands. Appropriate resuscitation was initiated with high flow oxygen and intravenous crystalloid bolus and vasopressors.

ECG revealed sinus tachycardia with no significant ST-T changes ruling out any probable acute coronary event (Figure 1).

Chest radiograph was within normal limits (Figure 2).

Arterial blood gas had type I respiratory failure and uncompensated metabolic acidosis (pH 7.24, Po2 80, pCO2 32.1, Bicarbonate 13.9, Lactate 3.2). The above ABG findings along with normal respiratory system examination suggested the diagnosis of PE more likely. Intravenous Heparin 5000 IU was administered. Soon after, patient started gasping and had a witnessed car-

[1] Consultant Emergency Medicine, Paras Hospital, Gurugram, India
[2] Class 1 Specialist medical officer, Dept of medical health and family welfare, Uttar Pradesh
[3] Chief Intensivist Critical Care Department, Sir H N Reliance Foundation Hospital, Mumbai, India
Corresponding author: Dr Shweta Tyagi, Consultant Emergency Medicine
Paras Hospital, Gurugram, India. **Address:** M-1, Green Park Extension, New Delhi 110016. Email:shwrah@gmail.com

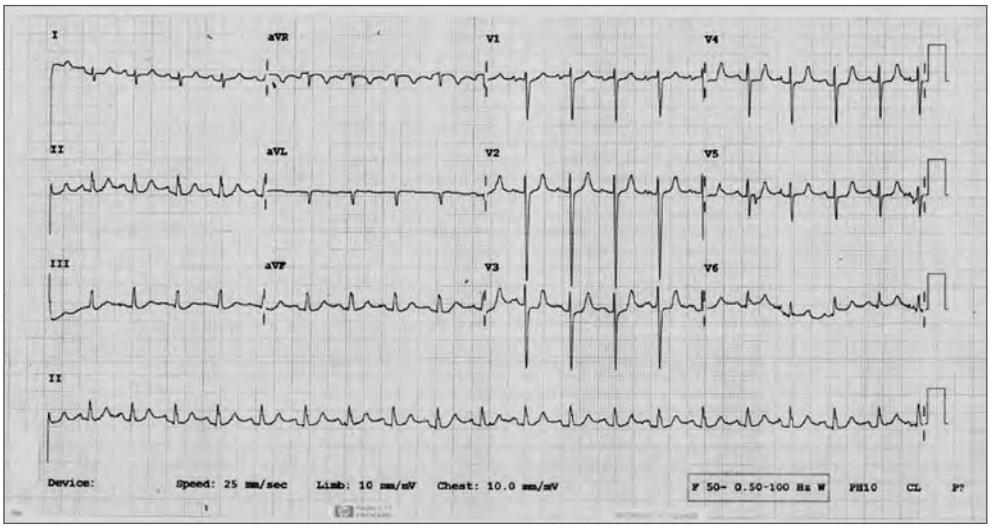

Figure 1: Electrocardiogram

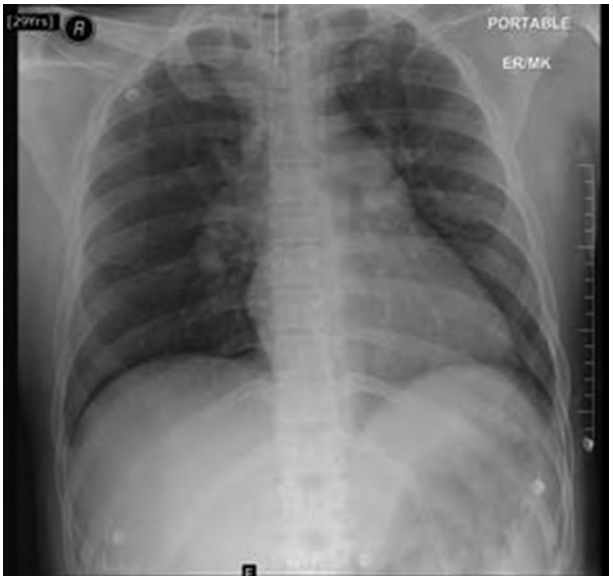

Figure 2: Chest Radiograph

diac arrest with first monitored rhythm being Pulseless Electrical Activity (PEA). Cardio-pulmonary resuscitation (CPR) was initiated and continued as per advanced cardiac life support protocol. Patient was intubated and mechanically ventilated. Return of Spontaneous Circulation (ROSC) was achieved within five minutes. Post-ROSC portable echocardiogram revealed severe pulmonary hypertension with right ventricular dilatation (Figure 3A) and plethoric inferior venecava (Figure 3B). Provisional diagnosis of PE was made.

Meanwhile patient again went into cardiac arrest and CPR was initiated as per ACLS protocol and immediate intravenous thrombolysis was planned. Family was counselled and consent was obtained for thrombolysis. The contraindications were ruled out and intravenous bolus of 50 mg of Alteplase was administered which was followed by infusion of 50 mg over one hour. ROSC was achieved within five minutes. Brief multiple episodes of cardiac arrests kept occurring for the next half an hour which were successfully revived following which the patient remained hemodynamically stable. Portable colour Doppler of bilateral lower limbs revealed near complete thrombosis of right popliteal vein and superficial femoral vein (Figure 4A & Figure 4B).

Post thrombolysis, he was observed in the critical care unit. The blood work, including hemogram, renal, liver and coagulation profile was within normal limits. However, the Troponin I and BNP were elevated. CT pulmonary angiogram eventually was suggestive of acute pulmonary thromboembolism with nearly completely obstructing distal

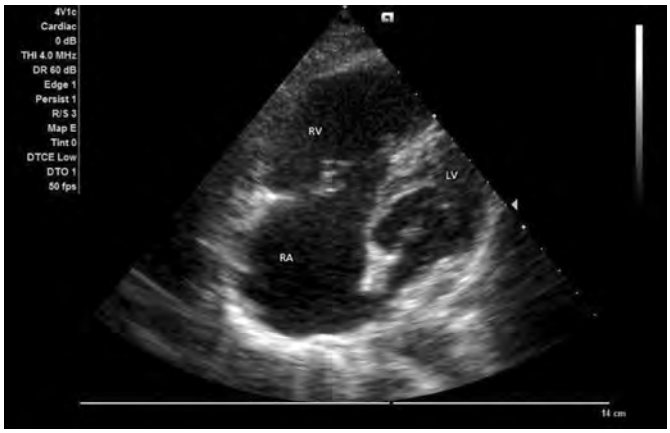

Figure 3A: Portable Echocardiography: Right ventricular Dilatation

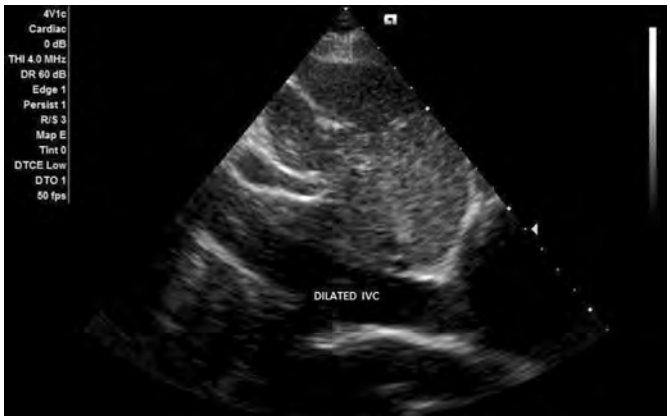

Figure 3B: Portable Echocardiography: Dilated Inferior Venecava

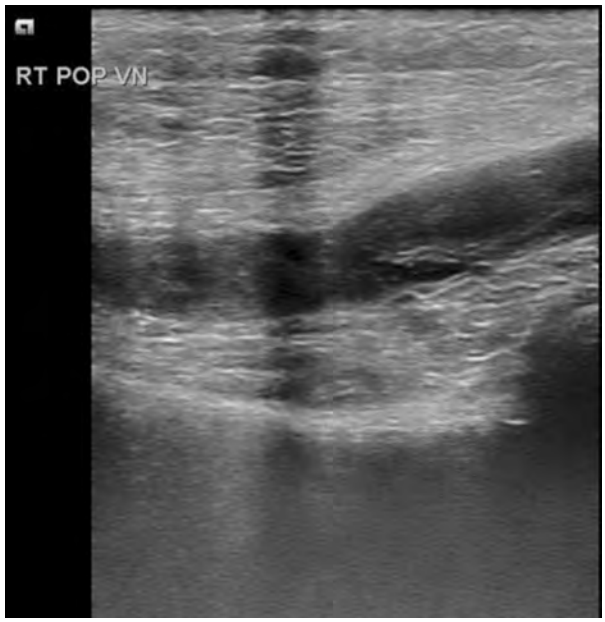

Figure 4A: Venous Doppler: Right Popliteal vein thrombus

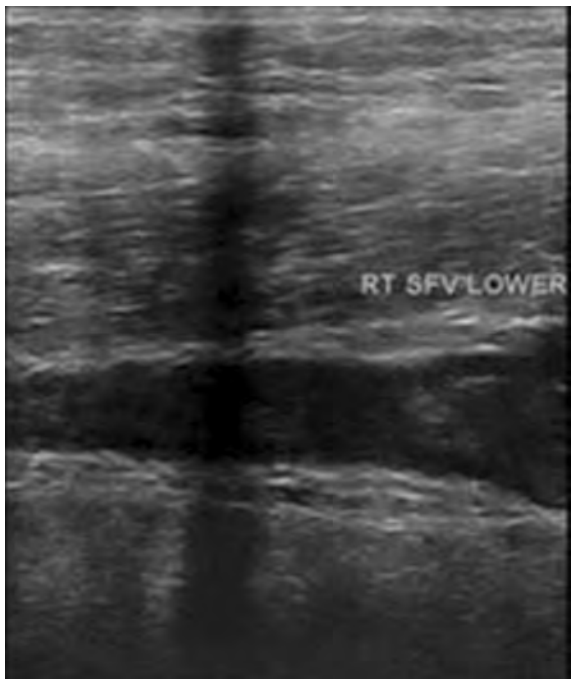

Figure 4B: Venous Doppler: Right Sapheno-femoral vein thrombus

right pulmonary artery with complete thrombotic occlusion of lobar arteries. On the left side, the thrombus was partially obstructing the distal left pulmonary artery (Figure 5).

Subsequently patient was continued on anticoagulation, weaned off the ventilator and given vasopressors for the next 24 hours and discharged home after a week on oral anticoagulants without any neurological deficit.

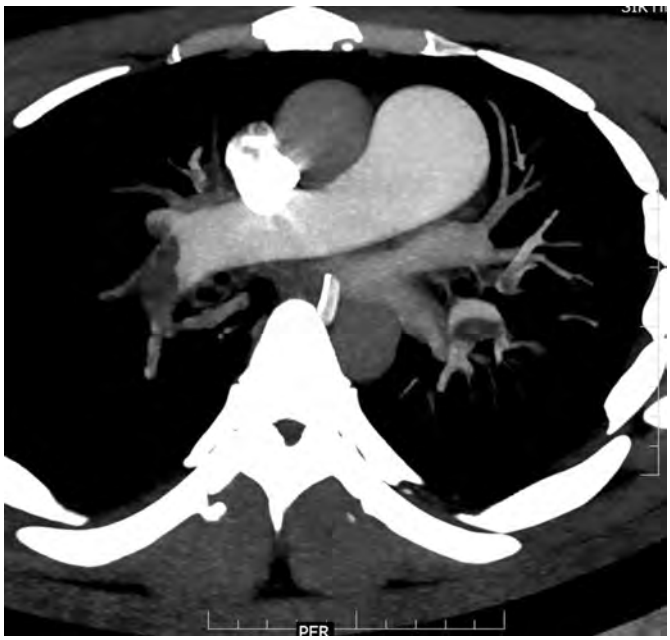

Figure 5: CT Pulmonary Angiogram

Discussion

PE as a cause of cardiac arrest is frequently missed. A post-mortem study by Kürkciyan *et al*. reported that PE as a cause of cardiac arrest was missed in 30% of all PE related cardiac arrest cases.[1] The autopsy study done by Hauch *et al*. that included post-operative patients documented the presence of PE in 62.5% of patients.[2]

Acute PE is one of the reversible causes of cardiac arrest with non-shockable rhythms. Thrombolysis should be considered in such cases and resuscitation should be continued and extended to at least 60-90 minutes once patient is thrombolysed.[3] Echocardiographic evidence of an enlarged right ventricle with a flattened interventricular septum supports the diagnosis of PE.[4] With strong suspicion of PE and when cardiac arrest is eminent immediate thrombolysis with Alteplase 50 mg intravenous (IV) bolus which can be repeated in 15 minutes, or single-dose weight-based intravenous Tenecteplase can be lifesaving.[5] Presence of hemodynamic compromise risk stratifies the suspected PE patient of having high mortality.[6] Systemic thrombolysis helps in faster relieving of obstruction in the pulmonary circulation and improves the right ventricular dysfunction.[7]

Bottiger *et al*. (1994) in a review of three case series concluded that early thrombolysis during cardiopulmonary resuscitation for suspected massive pulmonary embolism reduced mortality and revealed an initial survival rate of 55–100%.[8] Patient's history, clinical findings and portable echocardiography can be useful in reaching the diagnosis and guiding the therapy at

the bedside. However, in a subsequent double- blind, multicentre trial, Böttiger *et al* (2008) reported that no improvement in outcome was detected with the use of Tenecteplase without adjunctive antithrombotic therapy during advanced life support for out-of-hospital cardiac arrest in comparison with placebo.[9] The meta-analysis done by Xin Le *et al*. evaluated the role, efficacy and safety of thrombolysis in CPR and concluded that it significantly improved the rates of achieving ROSC and improved the survival rate to discharge with long term neurological function ($p<0.01$).[10] Karin Janata *et.al*. performed the retrospective cohort study to establish the major bleeding complications while thrombolysis in suspected PE with ongoing CPR and found the benefits outweigh the risk of bleeding.[11]

Er *et al*. retrospectively assessed 104 patients with suspected PE and witnessed cardiac arrest and concluded that rescue- thrombolysis should be considered and started in patients with PE and cardiac arrest, as soon as possible after cardiac arrest onset.[12] This study was quoted in the 2015 American Heart Association guidelines update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, under the statement "Early administration of systemic thrombolysis is associated with improved resuscitation outcomes compared with use after failure of conventional ACLS" in cardiac arrest associated with PE.[13]

In our case, the diagnosis of PE was strongly suspected on the basis of clinical presentation and relevant evaluation in the Emergency Room (ER). Type I respiratory failure, hypotension, sinus tachycardia on ECG, relatively normal chest radiograph and the right ventricular dysfunction on echocardiogram helped us guide the management. Our patient also responded to intravenous thrombolysis during ongoing CPR. High quality CPR, suspecting PE and early fibrinolysis were the key elements of successful resuscitation and complete neurological recovery of this patient.

Conclusion

Though the outcome after cardiac arrest due to massive pulmonary embolism is generally dismal yet fibrinolytic therapy is a potentially life-saving intervention and the Emergency Physician should not hesitate to administer it in the Emergency Room.

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