A case of massive pulmonary embolism causing cardiac arrest managed with successful systemic thrombolytic in the emergency department

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Abstract

Pulmonary Embolism (PE), when complicated by cardiac arrest, is almost always fatal despite all resuscitative efforts. However, a more favorable is possible when PE is rapidly identified as the cause of cardiac arrest and pulmonary circulation is quickly re-established by specific therapy. A 54-year-old woman was brought to the Emergency Department (ED) by 112 emergency ambulance service with the complaint of shortness of breath that had started 2 hours ago. She developed cardiac arrest while being physical examined 2 minutes after admission, and Cardiopulmonary Resuscitation (CPR) was immediately begun. Massive PE was considered the most likely diagnosis in the light of her history, physical examined, and bedside ultrasonography findings; thus, recombinant tissue Plasminogen Activator (r-tPA) was administered during CPR. The second CPR attempt achieved return of spontaneous circulation within 5 minutes. She was treated at intensive care unit for 32 days and discharged from the hospital with complete recovery.

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Introduction

Acute Pulmonary Embolism (PE) is a common type of venous thromboembolism, which may lead to death in some cases. It exhibits a range of clinical manifestations that are mostly nonspecific, which complicates diagnostic processes. When suspected, it should be meticulously sought to make the diagnosis and start the treatment in a timely manner to reduce patient morbidity and mortality.[1]

Hemodynamically unstable PE, also known as "high-risk" PE, defined as need for Cardiopulmonary Resuscitation (CPR) or a systolic Blood Pressure (BP) that is less than 90 mmHg or vasopressor required to achieve to a blood pressure greater than 90 mmHg despite adequate filling status or persistent hypotension not caused by new onset arrhythmia, hypovolemia or sepsis.[2]

The goal of emergency treatment of high-risk PE cases is to restore blood flow in pulmonary circulation by eliminating pulmonary artery obstruction and to prevent recurrent embolism.[3] Anticoagulation using heparin or thrombolytic drugs, removal of embolus by catheter-based interventions, and surgical embolectomy are the treatment options used for this purpose.[4] Although thrombolytic administration is regarded as the treatment of choice for high-risk PE, CPR constitutes a relative contraindication for thrombolytic therapy, due largely to the concern of potential bleeding after traumatic chest compressions. Nevertheless, it has been repeatedly shown by both case reports and clinical trials that thrombolytics can achieve Return of Spontaneous Circulation (ROSC), and consequently a more favorable neurological function, in survivors when administered during or after CPR.[5]

Herein, we present a patient with high-risk PE causing in-hospital cardiac arrest, who eventually fully recovered after receiving systemic thrombolytic therapy while being resuscitated.

Case Report

A 54-year-old woman was brought to our Emergency Department (ED) with the complaint of shortness of breath that had started 2 hours prior to her presentation. Her past history was

not remarkable for any disease or medication use. Her vital signs at admission were as follows: BP: 95/68 mmHg; pulse rate 114 bpm; respiratory rate 28 breaths/min; SO2 87%. Two minutes after admission, the patient underwent cardiac arrest while her physical examination was performed, and manual chest compression was immediately started according to the Advanced Cardiovascular Life Support (ACLS) algorithm. The patient gained ROSC in a period of less than 20 min after the initiation of CPR. An orotracheal intubation was performed and an Electrocardiography (ECG) was obtained demonstrating right bundle branch block, S1Q3T3 pattern and regular rhytm without p waves with ventricular rate 136 beats per minute (Figure 1). At the beginning of CPR, arterial blood gas analysis showed the following: pH 6.9, PCO2 75 mmHg, PO2 45 mmHg, lactate 10.5 mgdL, glucose 147 mgdL, BE -15.5, HCO3 13.9 mmol.

A point of care Ultrasonography (USG) examination was attempted to determine the cause of hypoxia and hypotension; however, it could not be performed due to faulty cardiac transducer of the USG device. Instead, bedside USG was taken from the subxiphoid region using the standard ultrasonography transducer, which showed right ventricular enlargement. Massive PE was considered as the most likely diagnosis in the light of sudden onset respiratory distress concomitant with circulatory collapse, physicial examinaton, and USG findings; therefore, a CT Pulmonary Angiography (CTPA) was ordered to diagnose PE. However, the patient suffered a second cardiac arrest and CPR was reinitiated while she was transferred to the tomography room. Since it was strongly considered that the patient was suffering massive PE with poor hemodynamic status precluding imaging, it was contemplated to administer thrombolytic therapy in the form of recombinant tissue plasminogen activator (r-tPA) at a bolus dose of 50 mg during CPR. Fortunately, the patient gained ROSC within 5 minutes after bolus administration of r-tPA and the remainder of 50 mg of r-tPA infusion initiated immediately.

The patient's hemodynamic status was stabilized few minutes after achieving ROSC. Mechanical ventilation at the assist control mode was started. Computed Tomography (CT) angiography was obtained to confirm the diagnosis, and it showed thrombi completely obstructing both pulmonary arteries (Figure 2). After massive PE was confirmed, the patient was admitted to Intensive Care Unit (ICU) for 32 days, and she was discharged after an uneventful hospital course with complete recovery.

Discussion

Pulmonary embolism may manifest with a wide spectrum of clinical scenarios that range from asymptomatic episodes to shock, or even sudden death. Patients most commonly present with dyspnea followed by chest pain, cough, and symptoms of deep vein thrombosis. Hemoptysis is usually not observed. Patients may occasionally suffer circulatory shock, arrhythmia, or syncope.[6]

It is paramount to differentiate hemodynamically unstable PE from hemodynamically stable PE since the former is associated with cardiogenic shock due to severe pulmonary artery obstruction followed by impaired blood flow. Although death secondary to hemodynamically unstable PE is typically observed in the first two hours of the embolic event, its risk is still high by 72 hours after embolic occlusion of pulmonary vasculature.[7] Our patient had dyspnea that had begun 2 hours prior to her ED admission. At admission, she had tachypnea, hypotension, and clinical signs of shock; she developed cardiopulmonary arrest in as little as 2 minutes after admission. Reduced stroke volume and cardiac output are responsible for hypotension secondary to massive PE. PE leads to an increase in pulmonary vascular resistance because embolic occlusion of the pulmonary vasculature and hypoxic vasoconstriction of the pulmonary arterial tree both cause an increase in arterial impedance. This event causes an increased afterload upon the right ventricle, causing the latter to dilate and the interventricular septum to flatten or deviate leftwards. Cardiac output in turn drops precipitously due to reduced right ventricular forward stroke volume and diminished left ventricular filling due to leftward septal shift.[8] In our patient, a bedside USG revealed right ventricular dilation while a CTPA showed thrombi completely obstructing both main pulmonary arteries. We believe that cardiac arrest developed as a result of the aforementioned mechanism.

Massive PE is mostly diagnosed by a CTPA scan or, less commonly, a ventilation perfusion scan or other imaging studies.[9] Computed Tomography (CT) angiography has became the method of preference for suspected PE in routine clinical practice but CT should not be the first-line test for all patients, except suspected

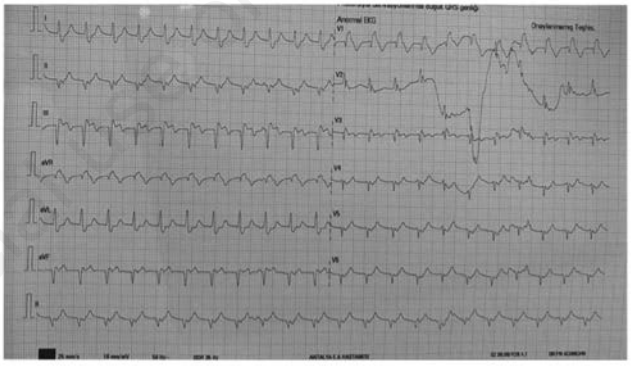

Figure 1. Electrocardiography demonstrating RBBB, S1Q3T3 pattern and regular rhytm without p waves with ventricular rate 136 beats per minute.

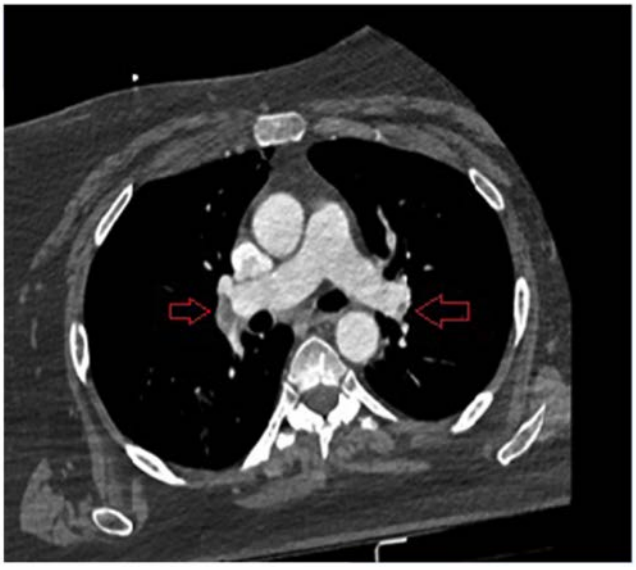

Figure 2. CTPA shows thrombi completely obstructing both pulmonary arteries.

high-risk patients and high clinical probability or "PE likely" patients. In these situations emergency phisician's echographic cardiac study can be specially helpful for a serial patient management.[10] Hemodynamically unstable patients who have no time for definitive diagnostic imaging may gain benefit from bedside echocardiography that may reveal signs of massive PE and allow empiric potentially life-saving therapies.[9] We also attempted to obtain a CTPA to confirm the diagnosis, but we evaluated the patient with a bedside USG because she developed cardiopulmonary arrest. Since the USG showed signs of right ventricular dysfunction, we administered thrombolytic therapy in the light of history, clinical presentation, and USG findings. The diagnosis was confirmed by a CTPA after the patient had ROSC and stable vital signs after the administration of thrombolytic therapy.

Randomized prospective studies demonstrated that systemic thrombolytic therapy is associated with early and rapid hemodynamic benefit. Expert consensus is in favor of administering thrombolytic therapy because the net clinical gain obtained by rapid resolution of hemodynamically important emboli surpasses the theoretical concern of major or life-threatening bleeding in patients with hemodynamically unstable PE.[11] Guidelines for the Diagnosis and Treatment of Acute PE published by the European Society of Cardiology (ESC) made a class 1B recommendation for the administration of thrombolytic therapy in patients with high-risk PE.[2] However, lytic therapy must be weighed against the 9-22% risk of major bleeding, including a 1-5% risk of intracranial hemorrhage.[12] Our patient also developed cardiopulmonary arrest secondary to massive PE. We also gave the patient thrombolytic therapy, taking the risk of major bleeding into account; our patient did not develop any complication of the thrombolytic therapy.

It is known that the earlier thrombolytic therapy is started, the higher its effectiveness. Delays in diagnosis or therapy may result in fatal deterioration; therefore, rapid diagnosis and an appropriate therapeutic approach are needed. Survival has been reported after thrombolytic therapy despite long CPR time among patients who developed cardiac arrest due to massive PE.[13] There are data indicating improvements in cerebral microcirculation at the time of reperfusion following thrombolytic administration for cardiac arrest due to massive PE. In massive PE alteplase is recommended at a dose of 100 mg given Intravenously (IV) over 2 hours.[14] Recent data from a nationwide cohort in Germany showed that thrombolysis was associated with lower in-hospital mortality rates in patients with hemodynamic instability, shock and necessitating CPR.[15] Our patient suffered cardiac arrest 2 minutes after ED admission and underwent CPR for 20 minutes. After the administration of 100 mg IV thrombolytic during CPR, she achieved ROSC in a short time and was discharged from the hospital without neurological sequela after a 32-day ICU treatment.

Conclusions

Massive PE with signs and symptoms of shock or hypotension substantially increases the risk of in-hospital death, and that risk is highest in the first few hours of the primary event. High risk PE is a strong indication for administering reperfusion therapies, in particular systemic thrombolysis, to achieve primary reperfusion.

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