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Bomb Blast and Its Consequences: Successful Intensive Care Management of Massive Pulmonary Embolsim.

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CASE REPORT

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ABSTRACT

A suicide bomb blast in 2013 at a distant city of Pakistan killed 84 and wounded more than 150 people. Some patients were transferred to our tertiary care hospital because of extreme load on medical services there. This patient arrived at the Aga Khan Hospital, 2 days after the bomb blast injury and underwent an orthopedic procedure. Next day, he developed sudden tachypnea, desaturation, and circulatory collapse. After initial cardiopulmonary resuscitation, he was immediately transferred to surgical intensive care unit. Based on history, echocardiography findings and patient parameters, a clinical diagnosis of massive pulmonary embolism was made and immediate thrombolytic therapy with alteplase was started. The immediate improvement in hemodynamic status was evident following 2 hours of alteplase infusion. This case also highlights the aggressiveness of resuscitation, decision making in initiating thrombolytic therapy on clinical grounds, importance of deep venous thrombosis prophylaxis, and exhaustion of health resources due to blast related mass destruction.

Key Words: *Pulmonary embolism. Resuscitation. Intensive care. Alteplase.*

INTRODUCTION

The clinical picture of acute pulmonary embolism (PE) is not similar from various aspect of its pathophysiological course presenting from minimal symptoms to variety of severity of disease like severe hypoxia, hypotension, right heart failure and death.[1] Massive pulmonary embolism is defined as obstruction of blood flow to a lobe or multiple segments of the lung, or for unstable hemodynamics, i.e. failure to maintain blood pressure without supportive measures. This case highlights about aggressiveness of treatment and role of thrombolytic therapy for a severely hemodynamic unstable patient.[2]

CASE REPORT

In February 2013, a 48-year male with no prior co-morbidities transferred from Quetta to the Aga Khan University Hospital, Karachi, after sustaining multiple lacerations and left tibia bone closed fracture in a bomb blast. After initial management in the hospital, he underwent an orthopedic procedure (application of Illiazarov over left tibia) under general anaesthesia. The surgery went uneventful, and he was then shifted to the ward. On the next morning, he suddenly developed dyspnea, became hypotensive and developed tachycardia (respiratory rate about 40/minute, pulse 140 beats per minute and blood pressure around 60/30 mmHg). He was barely maintaining oxygen saturation 90% on 10 liters of O_2 via facemask. Initially, he was given 2 liters of fluid bolus but hemodynamics did not respond. A few minutes later, he went into pulseless electrical activity (PEA) and cardiopulmonary resuscitation (CPR) had to be started. During CPR, he was intubated, epinephrine 1 mg given and return of spontaneous circulation (ROSC) occurred after 12 minutes of CPR. Dopamine infusion was given to support the blood pressure but it could not raise more than 60/30 mmHg. He was immediately transferred to surgical intensive care unit (SICU).

On arrival in SICU, the GCS of patient was 2/10; pupils were fixed and dilated (6 mm in size). He was put on mechanical ventilation with assist control mode (settings: R/R 26 minutes, TV 500 ml, PEEP 7 and 100% FiO_2). An arterial and central venous catheter inserted, quickly switched over to norepinephrine and epinephrine infusions in escalating doses and fluid resuscitation was continued. Despite all these efforts, blood pressures were still unresponsive. The 12-lead ECG showed S1Q3T3 with right ventricular strain pattern (Figure 1). The first ABG's and serum electrolytes were indicative of severe metabolic acidosis and also lactic acid was 11 meq/l, which suggested hypo-perfusion. A bedside echocardiogram revealed moderate right ventricle (RV) dysfunction.

Looking at clinical parameters, investigations, and history of distant transfer, it was suggestive of massive pulmonary embolism (PE). Because of severe hemodynamic instability, he was unable for shifting to radiological suit for helical CT scan. Immediate thrombolysis with tissue plasminogen activator (alteplase) planned and high-risk consent was taken.

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Then 100 mg alteplase intravenously infused over 2 hours; and immediately following infusion, his blood pressures were getting better and vasopressors requirement decreased. The acidosis slightly got better than previous (pH 7.09/$PaCo_2$ 38/PaO_2 180/BE - 12/SaO_2 99% and pH 7.12/$PaCo_2$ 31/PaO_2 150/BE 10/SaO_2 98%). Despite this adequate resuscitation, the patient became anuric with increased creatinine. Anticipating acidosis aggravation, sodium bicarbonate infusion at 20 meq/hr was also started and meanwhile continuous renal replacement therapy (CRRT) initiated next morning. The most encouraging sign at this moment was improvement in patient's consciousness as GCS was 10/10.

Next evening, there was profuse per oral bleeding and he went into moderate hemorrhagic shock. The hemoglobin dropped to (8.4 g/dl) and the coagulation profile became markedly deranged (INR - 6.5, APTT 69/30 seconds, fibrinogen 40 mg/dl). The other significant biochemical abnormalities were BUN 58 mg/dl, Cr 3.7 mg/dl, HCO_3 9.5 mg/dl, and Trop I - 64 IU. Three units pack cells, 8 FFPs, 8 cryoprecipitate, and 6 PLTs were transfused, besides vitamin K 10 mg IV, desmopressin 0.4 mcg/kg, and 4 gm of calcium gluconate over 4 hours was also given. Gradually, he got out of this hemorrhagic shock and CRRT continued to facilitate ultra-filtrate, hemofiltration and removal of toxic metabolite.

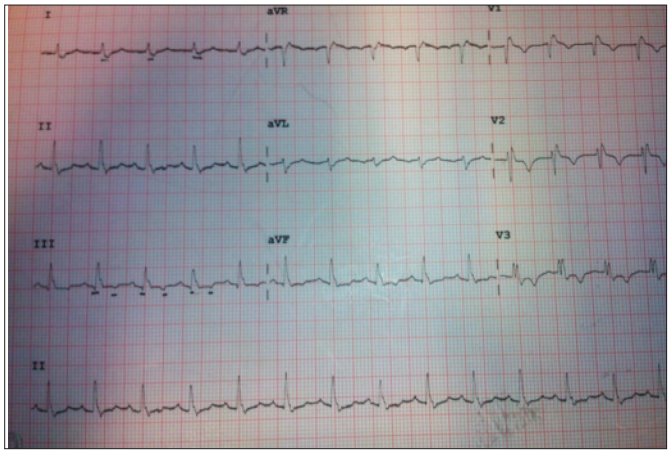

Figure 1: Electrocardiogram showing typical PE changes (S1 Q3 T3).

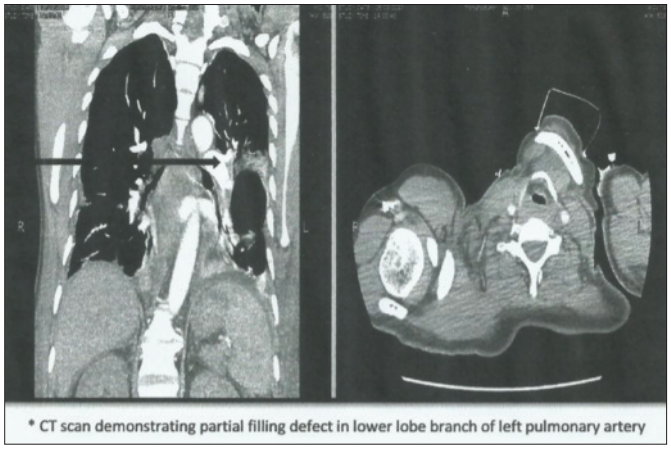

Figure 2: Partial filling defect in lower lobe branch of left pulmonary artery representing pulmonary embolism.

On second day, a repeat echocardiography demonstrated severely dilated right atrium and ventricle, moderately reduced RV function (Mc Connell's sign present), severe tricuspid regurgitation and dilated IVC with loss of inspiratory collapse. CT scan chest was also done which demonstrated filling defect in lower lobe branch of left pulmonary artery (Figure 2). Over the course in ICU, he remained hemodynamically stable, renal functions were supported with renal replacement therapy, and he was taken to interventional radiology suit for inferior vena caval filter placement for prevention of further embolism. He was successfully extubated on 6th day.

He was put on regular hemodialysis sessions in the ward and after staying for 6 weeks in the hospital when his renal function gradually improved, he was discharged from hospital.

DISCUSSION

Terror has radically changed in the last 6 years with terrorist attacks making alarming headlines.[3] In Pakistan, bomb blasts are causing much destruction, mostly being sectarian killing with suicide model. The existing trauma care systems have basic facilities, predominantly found in major cities and without integration at regional or national levels.

The importance of this case can be summarized with multiple reasons. First, the importance of DVT prophylaxis because patient was shifted here after 2 days. Second, the aggressiveness of treatment for a patient with fixed dilated pupils, with very low pressure not perfusing vital organs; and third, the clinical decision to give alteplase (tissue plasminogen activator [tPA]) for high suspicion of pulmonary embolism.

Acute and massive PE causes hypotension, defined as a systolic blood pressure < 90 mmHg or a drop in systolic blood pressure of ≥ 40 mmHg from baseline for a period > 15 minutes.[4] It should be suspected anytime when there is hypotension accompanied by an elevated central venous pressure (or neck vein distension), which is not otherwise explained by acute myocardial infarction, tension pneumothorax, pericardial tamponade, or a new arrhythmia.[5] It is a catastrophic entity that frequently results in acute right ventricular failure and death.

In addition to resuscitative efforts, parenteral anti-coagulant or fibrinolytic therapy should be initiated in all patients in whom acute PE has been confirmed or suspected.[6] Although systemic fibrinolysis is not worth the risk in all patients with acute PE, it is recommended as standard, first-line treatment in patients with massive

PE.[7] In an overview of 11 randomized controlled trials of fibrinolysis versus heparin among 748 un-selected PE patients, major bleeding complications occurred in 9.1% of fibrinolysis-treated and in 6.1% of heparin-treated patients.[8] The preferred fibrinolytic agent was alteplase (tPA) 100 mg continuous 2-hour infusion. In 2010, the FDA label for alteplase (Activase, Genentech, San Francisco, CA) explicitly stated that the agent is indicated for massive PE.[9] Alteplase was chosen as the first choice to manage such catastrophic PE and was found to be extremely useful in this situation. This was the first experience of the authors about alteplase.

In conclusion, management of massive pulmonary embolism requires a rapid and accurate assessment of risk; and a decisive treatment plan should be established, including aggressive resuscitation and anti-coagulant / fibrinolytic therapy.

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