Postoperative Acute Pulmonary Embolism: A Case Report

S Mishra, P Kundra, B Hemavathi, A Badhe, M Ravishankar

Citation

Abstract

Deep venous thrombosis and pulmonary embolism are some of the early postoperative complications following prostatectomy1. If the embolism is massive and not recognized and treated specifically, it can be rapidly fatal. Here we give a case report of a patient who presented with early postoperative cardiac arrest, and was diagnosed as acute pulmonary thromboembolism and managed. The diagnosis and management of perioperative pulmonary embolism is discussed.

CASE REPORT

A 65 year male who had transurethral resection of prostate(TURP) under spinal anesthesia developed dyspnoea, tachypnoea and tachycardia, with progressive fall in blood pressure leading to cardiac arrest at 24 hours after surgery. The patient was successfully revived with CPR, and shifted to the critical care for ventilatory support and circulatory support with parenteral fluids and Dobutamine under hemodynamic monitoring.

A 12 lead Electrocardiogram revealed new onset RAD, incomplete RBBB, T inversion in V1-4, and bed side Echo showed RA,RV dilated, with RV hypokinesis.While patient was being stabilized on support a diagnosis of acute pulmonary embolism was made.

Pulmonary artery catheterization revealed, High RA pressure (25 mm hg), RV pressure (60/30) and PAP (75/35), low PCWP (12mm Hg). D-dimer was positive. The patient was anticoagulated with IV heparin and taken up for pulmonary angiography. Pulmonary angiogram confirmed the diagnosis; a filling defect was noted both in Right and Left Pulmonary Arteries. Aspiration of clot was attempted and thrombolysis started with intrapulmonary streptokinase and anticoagulation continued with IV Heparin.

The patient developed bleeding from surgical site requiring surgical exploration and massive blood transfusion with blood products. Heparin had to be discontinued, and consideration given for deploying IVC filter as in spite of initial improvement, A-aDo$_2$ and PAP pressure were still high and recurring pulmonary embolism suspected. A Greenfield IVC filter was deployed on 5 th day and the

patient weaned from inotropic and ventilatory support, as PAP started normalising. X-ray chest showed marked improvement of the earlier pulmonary oligaemia. Oxygenation and room air saturation returned to normal and by 1 week the patient was discharged from the ICU and sent home after another one week. Patient has been doing well on follow up with no functional limitation at 6 months of follow-up.

DISCUSSION

Maintaining a high degree of clinical suspicion for pulmonary embolism is of paramount importance. The onset of symptoms may be sudden as in this patient or gradual. The most common signs and symptoms are nonspecific: dyspnoea, chest pain, tachypnoea and tachycardia. Usually, pulmonary embolism patients with severe chest pain or haemoptyis have anatomically small emboli near the periphery of the lung. Ironically, patients with life-threatening pulmonary embolism often have a painless presentation characterized by dyspnoea, syncope or cyanosis. By clinical presentation this patient could be considered to have had a massive embolism presenting with dyspnoea, tachypnoea, tachycardia and hypotension leading to cardiac arrest requiring CPR. The patient was revived successfully from the arrest. External cardiac massage may force the embolus onwards in the pulmonary arterial tree, minimizing the extent of obstruction [2] .

Pulmonary embolism should be suspected in hypotensive patients when there is evidence of predisposing factors and there is clinical evidence of acute cor pulmonale especially if there is electrocardiographic evidence of acute cor pulmonale manifested by a new S1-Q3-T3 pattern, new

incomplete right bundle branch block or right ventricular ischemia. Under such circumstances it is useful to follow-up with a bedside echocardiogram.

The first confirmative evidence of pulmonary embolism in this patient was seen in the electrocardiogram (ECG).The finding of T-wave inversion in leads V1-4 is surprisingly common in pulmonary embolism [3] . The ECG helps to exclude acute myocardial infarction and to identify electrocardiographic manifestations of right heart strain. The patient had sinus tachycardia with right axis deviation and new onset incomplete RBBB. The differential diagnosis of new right heart strain includes acute pulmonary embolism, acute asthma or exacerbation of chronic bronchitis in patients with chronic obstructive pulmonary disease.

ELISA-determined plasma D-dimer (>500 ng/ml) has more than 90 percent sensitivity for identifying patients with pulmonary embolism proven by lung scan [4] or by angiogram [5] . This test relies on the the principle that most patients with pulmonary embolism have ongoing endogenous fibrinolysis that is not effective enough to prevent pulmonary embolism, but breaks down some of the fibrin clot to D-dimers which can be assayed by monoclonal antibodies that are commercially available. Although elevated plasma concentrations of D-dimers are sensitive for the presence of pulmonary embolism, they are not specific [6] . Levels will be elevated in patients for at least 1 week postoperatively and will also be abnormally high in patients with myocardial infarction, sepsis or almost any other systemic illness. This patient had evidence of fibrinolysis and had massive bleeding requiring re-exploration. Systemic fibrinolysis is particularly known to occur after prostate surgery leading to bleeding [7] .Therefore the plasma D-dimer ELISA is best used in patients without coexisting systemic illness. A normal plasma D-dimer ELISA has a greater than 90 percent probability of excluding PE.

Echocardiography is most useful among haemodynamically unstable patients. Often bedside echo will suggest pulmonary embolism if a constellation of findings indicates right heart failure, especially with sparing of RV apex. The findings include RV dilatation, RV hypokinesis, bowing of IV septum into left ventricle with preserved left ventricular function. With resuscitation, our patient had attained haemodynamic stability on ventilatory and minimal inotropic support. However at this point, echo showed evidence of RV dilatation and hypokinesis.

A concept of haemodynamic impairment has undergone an important evolution. We used to define haemodynamic instability rather simplistically as persistent systemic arterial hypotension requiring fluid resuscitation and high dose vasopressors. Patients with pulmonary embolism should be judged unstable if they present with right ventricular hypokinesis by echocardiogram, even in the presence of a normal systemic arterial pressure [8,9] . Such patients may initially appear deceptively stable based on the clinical evaluation alone. However, despite adequate heparin anticoagulation, patients with right ventricular hypokinesis are at high risk of recurrent pulmonary embolism and clinical deterioration, even if they are normotensive initially [10,11,12] , or appear to stabilize after initial resuscitation. Such patients therefore are prime candidates for more aggressive treatment with thrombolytic therapy or mechanical intervention. Hence the patient was taken up for pulmonary angiography. Before proceeding with angiography, right heart catheterization was done with careful recording of heart pressures and oximetry. This is vital as it can provide clues to alternate diagnosis not suspected earlier such as left ventricular failure or tamponade. If the pressure tracing "dampens" or "wedges" in the proximal pulmonary artery without balloon expansion, anatomically massive pulmonary embolism should be suspected [6] . In this patient right sided pressure were high and pulmonary angiogram showed intraluminal filling defect. Such defects seen in more than one projection is the most reliable feature to diagnose pulmonary embolism. Secondary signs reflect decreased perfusion and consist of abrupt occlusion of vessels, oligaemia or avascularity of a segment.

Normally, if the diagnosis of PTE is reliably established noninvasively, pulmonary angiography is not necessary, even if thrombolysis is planned. However this patient started bleeding from the surgical site and initiation of thrombolysis along with heparin anticoagulation in a patient having life threatening haemorrhage requires careful consideration. Hence a further step in the confirmation of the diagnosis was undertaken and treatment plan established.

As far as the treatment plan is concerned, Consensus guidelines from the American College of Chest Physicians are summarised as follows [13]

Primary versus secondary therapy: - Primary therapy consists of clot dissolution with thrombolysis or removal of PTE by embolectomy. Anticoagulation with heparin or placement of an IVC filter constitutes secondary prevention of recurrent PTE rather than primary therapy.

Primary therapy should be reserved for patients at high risk of an adverse clinical outcome. When right ventricular function remains normal, patients have good clinical outcome with anticoagulation alone.

Adjunctive therapy: Important adjunctive measures include pain relief with NSAIDS, and supplemental oxygenation and psychological support. Dobutamine may successfully treat right heart failure and cardiogenic shock. Volume loading should be undertaken carefully because increased right ventricular dilatation can lead to even further reduction in left ventricular forward output.

Mechanical ventilatory support with endotracheal intubation may be necessary in some patients. Accepted indications include a) inability to maintain oxygenation on oxygen supplementation by mask (PaO$_2$ < 60mm Hg on FiO$_2$ >0.6), b) hypotension with shock and post cardiac arrest, c) unconscious states with inability to maintain airway and adequate ventilation. As this patient had sustained a cardiac arrest and revived with altered sensorium and haemodynamic instability he was intubated and mechanically ventilated as an initial resuscitative measure and started on inotropic support.

The patient was first anticoagulated with intravenous heparin. Heparin in large doses (15000 units) is given for its serotonin blocking effects, to reduce pulmonary vascular and bronchial constriction [14]. Once embolism was confirmed thrombolysis with streptokinase was started. There is evidence that thrombolytic therapy can hasten the resolution of an embolus. A streptokinase 250,000 unit is given intravenously in the first 30 minutes of therapy followed by 100,000 units per hour for 48-72 hours [15]. Active bleeding is an important contraindication to its use, although it is generally assumed that no increased risk is present beyond 48 hours of surgery. Considering the early postoperative state, this patient was given streptokinase into the pulmonary artery through the PA catheter at a lower dose, hoping to reduce bleeding complications. Recurrent showers of embolism was suspected as, after initial improvement of the pulmonary oligaemia with thrombolysis, the patient continued to be hypoxiemic despite ventilatory support and failed at attempts to wean off ventilatory support. As the patient had massive haemorrhage, heparin was stopped and an alternate method of prevention of recurrence was resorted to and IVC filter used. An IVC filter does not treat an established pulmonary embolism directly, nor does it halt the thrombotic process and it may not be indicated in all patients. Accepted indications for filter insertion include venous thrombosis with active clinically important bleeding that prohibits the use of heparin and evidence of recurrent pulmonary embolism. After the filter was inserted he stabilized and was taken off all supports.

CONCLUSION

To conclude, Acute Pulmonary Embolism should be suspected in case of early postoperative cardiac arrest in susceptible individuals. Even in massive embolism with shock, the outcome can be favorable with timely aggressive management. Patients who appear to be deceptively haemodynamically stable after initial resuscitation are at risk of deterioration if there is evidence of RV hypokinesis by echocardiography and require aggressive primary therapy with thrombolysis.

CORRESPONDENCE TO

DR Sandeep kumar Mishra House no-29, IV- Cross Indira nagar extention Gorimedu Pondicherry -6 e-mail: sandeeplmishra@yahoo.co.in Phone no: - 919344668104

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Author Information

Sandeep Kumar Mishra, M.D.
Senior Resident, Department of Anaesthesiology and Critical Care, JIPMER

P. Kundra, MD, MAMS, FIMSA
Professor, Department of Anaesthesiology and Critical Care, JIPMER

B. Hemavathi, DA, MD
Professor, Department of Anaesthesiology and Critical Care, JIPMER

A. S. Badhe, MD, DA
Professor and Head, Department of Anaesthesiology and Critical Care, JIPMER

M. Ravishankar, DA, MD
Former Professor and Head, Department of Anaesthesiology and Critical Care, JIPMER