Case Report

Pulmonary Embolism Following Induction of Anesthesia

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Introduction

Pulmonary embolism is a potentially lethal complication and accounts for many cases of post operative deaths each year. The signs and symptoms of pulmonary embolism are not specific; it is difficult to be diagnosed in elderly population. One-thirds of the deaths due to pulmonary embolism occur within an onset of 20 minutes, and if untreated embolic episode occurs in most of the patients[1]. Although the primary cause of pulmonary embolism is deep vein thrombosis and emboli originating from the leg or pelvic veins , it may also be caused by amniotic fluid , fat, arthroplasty cement, air, and bone marrow[2]. Patients with trauma, particularly to the long bones and pelvis, who are bed ridden and immobile for a prolonged period of time are at greatest risk of developing venous thromboembolism. The following case report describes the anaesthetic events and management of a patient posted for posterior stabilization of burst fracture lumbar vertebrae (L1) who developed pulmonary embolism immediately following induction of anaesthesia and endotracheal intubation.

Case Report

A thirty-four year-old male with burst fracture L1 following road traffic accident was scheduled for posterior stabilization. The patient presented with complaints of neck pain, inability to move both lower limbs, bowel and bladder incontinence with no other comorbid illness. Airway examination showed Mallampati class I with a thyromental distance of 7cm, and full range of neck movement. Preoperative hemoglobin was 15.2gm/dl and hematocrit was 46.4%. Electrolytes and coagulation profile were within normal limits. Patient was adequately premedicated and shifted to operating room on the day of surgery.

In the operating room, standard monitoring of heart rate, non invasive blood pressure, oxygen saturation were monitored. His pre operative blood pressure was 110/70 mmHg, heart rate 90/min, respiratory rate 16/min, oxygen saturation on room air was 100%. Anaesthesia was induced with inj.glycopyrrolate 0.2mg, inj.midazolam 1 mg, inj.fentanyl 100 mcg, inj.propofol 140 mg and inj.vecuronium 6 mg. Oral intubation with an 8.0 mm I.D flexometallic endotracheal tube was successful, bilateral air entry equal with end tidal carbondioxide 35 mmHg. He was started on volume control mode of ventilation with tidal volume of 500ml, rate of 10/min, I:E ratio 1:2, airway pressure 14 cmH2O.

One minute after commencing ventilator support, the patient's saturation decreased to 82%, blood pressure 70/50 mmHg, heart rate 120/min. Fluid resuscitation with 500 ml crystalloids and inj.ephedrine 6mg was administered intravenously. The saturation remained 80-86% even with 100% oxygen. After two minutes, heart rate was 100/min, and blood pressure was 100/70mmHg. Arterial blood gases revealed pH of 7.19, PCO2 of 60 mmHg, and PaO2 of 70 mmHg. The end-tidal carbon dioxide showed 17 mmHg on the capnograph.The position of the endotracheal tube was reconfirmed for any inequality in lung ventilation or any added sounds to rule out bronchospasm. ECG on monitor showed ST segment depression with T wave inversion and poor progression of R waves, Echocardiography showed left atrial enlargement, right atrium/right ventricle mildly dilated, normal biventricular function, left ventricular ejection fraction 65%, pulmonary artery pressure 45mmHg. Ventilatory support was continued and patient shifted to ICU for further management. Venous Doppler showed deep vein thrombosis involving left popliteal vein extending into posterior tibial vein. The diagnosis was confirmed by CT pulmonary angiogram (Fig1) which showed intravascular filling defect associated with complete vascular occlusion of both pulmonary vessels.

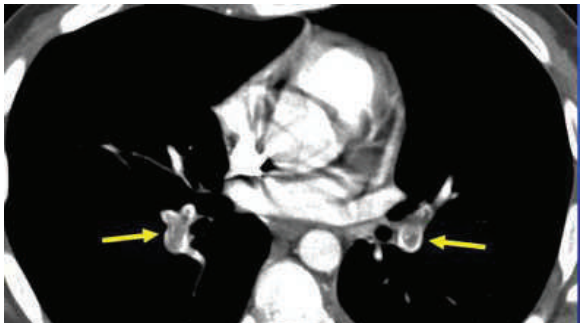

Fig1 - CT pulmonary angiogram shows pulmonary embolism

An inferior vena caval filter was inserted. Surgical procedure was postponed in view of pulmonary embolism. After 6 hours postoperative ABG showed severe respiratory acidosis with metabolic alkalosis. A therapeutic dose of inj. enoxaparin 80mg IV every 12 hours was started. On first post op day ABG showed respiratory acidosis with metabolic alkalosis, fraction of inspired oxygen was increased to 80%.On the second postoperative day, arterial blood gases revealed pH of 7.45, PCO_2 of 37. 1mmHg, PO_2 of 93.6mmHg, and oxygen saturation of 98% on FIO_2 0.4. The trachea was extubated on third post op day. On fourth post op day patient was shifted to ward , low molecular weight heparin was continued and leg compression stocking were placed to prevent clot formation.Subsequently the fracture was treated conservatively.

Discussion

Diagnosing pulmonary embolism is clinically challenging during intra operative period and is more often missed, being an incidental autopsy finding. The survival rate of pulmonary embolism is very poor with three month mortality being 17.1%. The outcome depends upon degree of vascular obstruction and associated right ventricular strain. In severe pulmonary obstruction, the right ventricle needs to generate a systolic pressure over 50 mmHg to maintain adequate cardiac output. About 85% of deaths due to massive pulmonary embolism occurs within the first 6 hours[3]. The main goal of treatment in massive pulmonary embolism is to improve the forward flow and to reduce the afterload in right ventricle, so as to avoid right ventricular failure.

The intravascular thrombus occludes the pulmonary vasculature and increases pulmonary humoral vasoconstrictors release which further increase pulmonary vascular resistance. Increasing the patency of pulmonary artery cross sectional area is the main aim of treatment in massive pulmonary embolism[4]. The measures to prevent the pulmonary obstruction are through thrombolysis, clot fragmentation and clot removal. The modality of treatment selection depends upon, hemodynamic stability of patient, risk of bleeding and the characteristic of thrombus. Traumatic patients who are immobilized are more prone to thromboembolitic events including pulmonary artery embolism secondary to deep vein thrombosis[5]. The predisposing factors are prolonged immobility, obesity, fracture involving pelvic bone or long bone fracture and prolonged surgery. The clots mainly get dislodged on mobilizing the patient and cause pulmonary embolism. Intraoperative features of pulmonary embolism are sudden unexplained hypotension, hypoxaemia, bronchospasm and a decrease in end tidal carbondioxide levels. The patient in our case report had paraplegia secondary to lumbar fracture and prolonged immobility. Since the patient was not started on deep vein thrombosis prophylaxis the embolic event occurred immediately after induction and intubation of the patient. The patient had tachycardia, hypotension, decreased saturation and end tidal carbondioxide levels.

Deep vein thrombosis is a well recognized problem following trauma to long bone fracture and pelvic fracture. The accepted modalities to prevent deep vein thrombosis are unfractioned heparin, low molecular weight heparin, or fondaparineux[6]. In addition anticoagulants are started in patients suspected to have deep vein thrombosis[7]. Deep vein thrombosis prophylaxis reduces the mortality rate from 30% to 10%. Non pharmacological methods like leg compression stockings may be used in patients with low risk of deep vein thrombosis[8]. Inferior venacaval filter is used in patients with high risk of recurrent pulmonary embolism[9]. Post operatively this patient was started on low molecular weight heparin for one week, leg compression stocking were placed while the patient was on bed rest to prevent further clot formation.

Conclusion

Immobilized patients with long bone fracture are a population at high risk for pulmonary emboli than other patients. The mortality following a pulmonary embolism depends on several factors, including the size of the embolus, the number of arteries blocked, and the overall health of the patient. Thorough screening and prophylactic actions to prevent deep venous thrombosis and consequent pulmonary emboli should be directed in all patients.

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