Case Report

Pneumothorax complicating pulmonary embolism after combined spinal epidural anesthesia in a chronic smoker with open femur fracture

Shivendu Bansal, Sohan Lal Solanki[1], Neena Jain, V K Vijayvergia

Department of Anaesthesia and Intensive Care, JLN Medical College and Hospitals, Ajmer, Rajasthan, [1]Post Graduate Institute of Medical Education and Research, Chandigarh, India

Abstract

Pulmonary embolism during or after regional anaesthesia is although very rare, it has been reported in cases undergoing lower limb orthopedic procedures. We presenting a 48 years old male, a known smoker since 25 years, with history of road traffic accident and open fracture right femur for external fixation. Combined spinal epidural anaesthesia was given. After 35 minutes patient complained dyspnea and chest pain. SpO2 decreased to 82% from 100%. Continuous positive airway pressure with 100% oxygen was given. SpO2 increased from 82% to 96%. Suddenly he had bouts of cough and SpO2 became 79-80% with unstable haemodynamics. On chest auscultation there was decreased breath sounds on right side with limited expansion. Trachea was intubated after inducing anaesthesia with fentanyl 70 μg and thiopental 300 mg. Chest radiograph showed right sided pneumothorax. Intercostal drain with a water seal was put. After 5 minutes HR was 80/min, BP was 110/69 mmHg and SpO2 was 97%. Pulmonary thromboembolism secondary to deep vein thrombosis was suspected and was confirmed by D-dimer Elisa and color Doppler of lower limbs. Patient was shifted to intensive care unit after completion of surgery. Anticoagulant therapy was started. He was weaned from the ventilator on 3rd day and trachea was extubated. Chest drain was removed after 9 days and he was discharged from hospital on 15th post operative day

Key words:Pneumothorax, Pulmonary Embolism, Smoker

Introduction

Pulmonary thromboembolism (PTE) during or after regional anesthesia, although very rare, has been reported in cases undergoing lower limb orthopedic procedures.[1,2] Patients with fractures of the hip, pelvis, or femur are at high risk of developing deep vein thrombosis and PTE.[3] Chronic smokers are known to be at risk of developing venous thromboembolism[4] and spontaneous pneumothorax.[5] We present a rare case of spontaneous tension pneumothorax complicating PTE in a chronic smoker with traumatic fracture of the shaft of right femur who was administered combined spinal epidural anesthesia.

Address for correspondence: Dr. Shivendu Bansal,
Department of Anaesthesia and Intensive Care, JLN Medical College and Hospitals, Ajmer, Rajasthan, India.
E-mail: shivendubansal@yahoo.co.in

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Case Report

A 48-years-old man weighing 68 kg, and of height 170 cm, was brought to the emergency unit after sustaining an open fracture right femur in a road traffic accident. He was scheduled for external fixation. On examination, the patient was conscious and well oriented. There was no history of loss of consciousness, vomiting or bleeding from oral and nasal cavities. The patient was a smoker since last 25 years (10 cigarettes/day). He denied any other medical comorbidity or surgical treatment in the past. Non-contrast computed tomography (CT) of head was normal, blunt trauma chest and abdomen was ruled out, no cervical spine injury was observed and hemodynamic parameters were also stable. Airway examination revealed mouth opening of 3 cm, thyromental distance of 8 cm, and full range of neck movements. Airway was classified as Mallampatti class II. Preoperative chest radiograph was normal and electrocardiography (ECG) showed normal sinus rhythm. Hemogram, coagulogram

and renal and liver function tests were within normal limits. Functional capacity was more than 4 metabolic equivalents (METS).

After explaining the procedure and risk involved to the patient, written informed consent was taken. In the operating room, ECG, noninvasive blood pressure (NIBP), and pulse oximetry were attached and baseline parameters noted. An 18-G intravenous cannula was secured. Preoperative heart rate (HR) was 90/min, blood pressure (BP) 130/80 mm Hg, respiratory rate 16/min, and SpO_2 (on room air) was 97%. Arterial blood gases (ABG) showed pH 7.35, pO_2 98, pCO_2 26, HCO_3 15, BE −5, and SaO_2 97%.

An 18-G epidural catheter was inserted via the L_3-L_4 space with the patient in a sitting position, using midline approach, and fixed at 11 cm. At the same level, 15 mg of 0.5% bupivacaine heavy was administered in the subarachnoid with a 25-G Quincke's spinal needle. Supplemented oxygen was administered by a venturi facemask (FiO_2 0.5). A sensory loss up to T10 level was achieved with adequate motor paralysis after 9 min and surgery was then started. ECG, NIBP, and SpO_2 were monitored. About 35 minutes after the administration of the subarachnoid block, the patient complained of dyspnea and chest pain. There was a sudden fall in SpO_2 to 82% from 100%. The patient became tachypneic, started coughing and his HR rose to 126/min. However, BP was within normal limits and ECG showed no marked changes. ABG showed pH 7.36, pO_2 65.6, pCO_2 45, HCO_3 17.2, BE −6.1, and SaO_2 82%. The patient was given continuous positive airway pressure of 100% oxygen with a facemask. SpO_2 increased from 82% to 96%. A sample for D-dimer test was sent with the suspicion of pulmonary embolism. The patient had another bout of cough and SpO_2 fell to 79-80%, BP to 81/54 mm Hg, and HR was 125/min. Dopamine infusion was started. On chest auscultation, there was reduced breath sounds on the right side. ABG values were pH 7.32, pO_2 55.5, pCO_2 54.2, HCO_3 17, BE −6.9, and SaO_2 80%. Anesthesia was induced with intravenous fentanyl 70 μg and thiopental 300 mg. Intravenous vecuronium 7 mg was administered and trachea secured with 8.5 mm endotracheal tube. The position of the tracheal tube was confirmed by capnography. Anesthesia was maintained with isoflurane in 100% oxygen. A chest radiograph was taken immediately, which showed a right-sided pneumothorax. Cardiothoracic consultation was taken and an underwater seal intercostal drain placed. After 5 minutes, HR improved to 80/min, BP 110/69 mm Hg, and SpO_2 97%. The surgery lasted for 130 minutes and was completed without any further complication. Postoperatively, SpO_2 was 97%, BP 105/62 mm Hg, and HR 113/min. The patient was shifted to the intensive care unit (ICU) and mechanically ventilated on Synchronized Intermittent Mandatory Ventilation (SIMV) mode with pressure support.

In ICU, the patient was monitored closely with invasive blood pressure, central venous pressure, oxygen saturation, airway pressure, and serial ABG. The D-dimer test report showed elevated values and color Doppler of lower limbs showed deep vein thrombosis of the right lower limb. Low-molecular-weight heparin therapy (enoxaparin 1 mg/kg subcutaneously 12 hourly) was started. Bed-side two-dimensional echocardiography in the ICU showed mild tricuspid regurgitation, mild pulmonary hypertension [RV systolic pressure (RVSP) = right atrial pressure (RAP) + 31 mm Hg], and hypokinesia of the right ventricular wall.

Postoperative analgesia was achieved with epidural injection of 8 ml of 0.125% bupivacaine 6 hourly, and epidural catheter was removed after 48 hours. The patient was weaned off ventilator support after three days and the trachea was extubated. Oxygen supplementation with venturi facemask (FiO_2 0.5) was given for another two days. Inotropic support was removed on the fourth day and the patient shifted to the ward on the fifth day. Chest drain was removed after 9 days, and he was discharged to home on the 15th postoperative day.

Discussion

In the case reported, development of severe hypoxia, tachypnea, dyspnea, chest pain, and tachycardia without hypotension following combined spinal epidural anesthesia in a 48-year-old chronic smoker with traumatic fracture shaft of femur raised the suspicion of pulmonary embolism. This was confirmed by raised D-dimer values[6] and color Doppler scan of lower limbs.[7] Systemic arterial hypoxemia is the earliest and most sensitive manifestation of pulmonary embolism and hypotension is less frequent and transient in patients with no prior cardiopulmonary disease.[8] Michael *et al.*[9] reported a case of intraoperative pulmonary embolism who developed a sudden, marked reduction in arterial oxygen saturation, detected by a pulse oximeter, without hemodynamic change. Diagnosis of acute PTE should be done and based on recommendations of Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) II investigators.[10] Clinical assessment of low, moderate, and high probability should be done prior to imaging. PIOPED II recommends different imaging modalities according to clinical assessment. In the present case we suspected this as a moderate probability case of acute PTE, and thus D-dimer Elisa test was carried out. Further imaging modalities (CT Angiography) was not done due to unavailability.

In our patient with history of chronic smoking, bouts of severe coughing induced due to the development of acute PTE might have resulted in the development of spontaneous tension pneumothorax. The hypothesis is supported by the

sudden worsening after initial improvement in the severity of hypoxemia and dyspnea, along with sudden onset of hemodynamic instability and reduced breath sounds on the right side of the chest. This was further confirmed by chest X-ray.

Chronic smokers are at a higher risk of developing deep vein thrombosis, which can lead to PTE following venodilation due to spinal anesthesia.[11] Chronic smokers are also known be at risk of developing spontaneous pneumothorax following episodes of exertion such as coughing. A definite history of exertion at the onset of pneumothorax in four cases was found in a study by Gupta *et al.*[12] Smoking may lead to inflammation and obstruction of small airways, accounting for the markedly increased risk of spontaneous pneumothorax.[13] According to Bense *et al.*,[14] the lifetime risk of developing a pneumothorax in healthy male smokers may be as much as 12% compared with 0.1% in non-smoker males. Rupture of subpleural blebs or bullae is associated with spontaneous pneumothorax. The etiology of such bullous changes in otherwise apparently healthy lungs is unclear. Undoubtedly, smoking plays a role.[14,15]

Fulminant fat embolism syndrome (FES) should always be considered in the differential diagnosis of respiratory distress in patients with fracture of long bones. Acute onset of FES with worsening hypoxemia has been reported in trauma patients undergoing orthopedic surgeries for long bone fractures.[16] Surgical techniques such as intramedullary nailing and reaming of the long bone have been implicated for massive fat embolism resulting in a sudden onset of pulmonary or cerebral symptoms.[17] Our patient became symptomatic soon after surgery started and was fully conscious until general anesthesia was delivered. He had no cognitive dysfunction following recovery from anesthesia and during the course of illness. There was no petechiae, which are a characteristic feature of FES.[16] Diagnostic criteria such as progressive anemia, thrombocytopenia, and/or leucopenia were absent.

In conclusion, the clinical symptoms like hypoxemia, desaturation, and chest discomfort in patients with traumatic long bone fracture undergoing surgery should be suspected as acute PTE. Further, severe coughing in these patients, especially chronic smokers, can precipitate pneumothorax. Therefore, a cautious vigilance is always necessary even in regional anesthesia to prevent critical events.

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