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CASE REPORT

Perioperative Acute Pulmonary Embolism in a Trauma Patient: A Case Report

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Abstract

Perioperative acute pulmonary embolism is a rare complication; however, it can be very serious and devastating sometimes. The incidence is higher in major orthopedic procedures like hip fracture repair, hip hemiarthroplasty and knee replacement/ arthroplasty. Here, we report a case of trauma presented with right acetabulum fracture planned for open reduction internal fixation (ORIF), who suffered pulmonary embolism intraoperatively with thrombolysis done in trauma and managed effectively.

Abbreviations

PE: Pulmonary Embolism; GCS: Glass Gowcoma Scale; CECT: Contrast Enhanced Computed Tomography Scan; NCCT: Non Contrast enhanced Computed Tomography Scan; ASA: American Standards of Anaesthesia; AST: Antibiotic Sensitivity Test; $EtCO_2$: End Tidal Carbon di oxide; $PaCO_2$: Arterial Partial Pressure of Carbon di oxide; POCUS: Point Of Care UltraSound; CTPA: CT Pulmonary Angiogram; DVT: Deep Vein Thrombosis; VTE : Venous Thromboembolism; ABG: Arterial Blood Gas analysis; HFNC: High Flow Nasal Cannula

Case Description

A 41-year-old male patient, admitted in the trauma centre after he met with road traffic accident hit by two-wheeler. On primary survey, his airway was patent with GCS of 15/15. On radiological evaluation his chest X-ray showed no abnormality, NCCT head and spine were normal, but CECT torso showed bilateral minimal pneumothorax and right acetabulum fracture, quadrilateral right SPR fracture plate extending to ilium zone 3 and pelvic hematoma. After initial stabilization, the patient shifted to ward and planned for ORIF for right acetabular fracture on the 4th day. A preoperative evaluation workup showed he was known type 2 diabetic from last 5 years (control on Tab. Metformin & Tab Glimepiride), body weight of 100 Kg having BMI of 35 kg/m^2 with STOPBANG of 4/8. Other preoperative investigations were within normal limits.

Patients shifted to operating room for right acetabulum surgery. Standard ASA monitors attached to the patients and general anaesthesia with tracheal intubation was administered along with epidural placed for the pain management. A right internal jugular vein was already in situ due to difficult intravenous access. Perioperative analgesia was maintained intravenous morphine, injection paracetamol and epidural top up. Approximately after 2 hours of surgery, a sudden drop in saturation from 99% to 88% and $EtCO_2$ from 38 mmHg to 26 mmHg noted on ventilator along with ECG showing ST segment depression, tachycardia from 89/min to 110/min with slightly decrease in blood pressure value to 100/54 mmHg.

Immediately FIO_2 was increased to 100% and patients was taken on manual ventilation to check for the compliance (increase in the airway pressure), and circuit connection were checked. On auscultation of chest bilateral breath sound heard and peak airway pressure were normal. An ABG obtained which showed pH-7.31, pCO_2-51, pO_2-101, K-5. In view of ABG findings, respiratory rate was increased from 16 to 20 and PEEP was increased to 8. After, 10 minutes saturation

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improved from 90% to 96% with FIO_2 of 90%, heart rate of 110/min and BP OF 116/68 mmHg. Surgery was allowed to continue for another half an hour.

Patient shifted to ICU with ETT tube in-situ, having HR-120/min, BP-110/69 mmHg, Spo_2- 100% on FiO_2 of 90% in view of high oxygen requirement due to suspicion of thromboembolic event. A cardiac POCUS in done in ICU showed RA enlarged, RV normal, mild TR and LVEF of 50%. An ECG done shows- S1Q3T3 pattern and bedside TROP I tested positive. Patients continued to ventilate overnight with urgent cardiac consultation sought in view of the above findings and low dose noradrenaline was started. All the routine investigations along with d-dimer and urine for microscopy was sent.

On Day 1 of ICU, CTPA study done which revealed filling defects in distal LPA, RPA extending into lobar, segmental and subsegmental bronchus of pulmonary artery with right atrium and right ventricle mildly dilated and prominent MPA with contrast reflux into IVC. (Figure 1) In the evening patients was extubated, and put on HFNC at 40L/min flow and 40% FIO_2. An echo by cardiologists shows, RA and RV are dilated. However, b/l DVT scan was negative. His d-dimer level found to be raised 11.8 mg/L. Considering the above findings, a clinical diagnosis of massive pulmonary embolism was made. As advised by cardiologist, injection Tenecteplase 50mg was started and monitoring with aPTT was done. Patients continued on minimum noradrenaline support. Later on, patients started on injection Unfractionated Heparin with 7200 U bolus over 1hr then 1800 U/hr infusion. Patient shifted from HFNC to Nasal Prongs @ 2L/min flow in subsequent 1-2 days but not able to taper off vasopressors. In view of persisting requirement of low dose vasopressor and decide further course of treatment, a repeat CTPA study was done after 3 days which revealed multiple filling defects in distal LPA extending into lobar, segmental and subsegmental branches with left lower lobe infarct. (Figure 2) So, it was advised to continue with Heparin infusion and monitor aPTT. Over next 5 days, the vasopressors got tapered, and patient got hemodynamically stable. Finally on 10th day of ICU stay, patient put on inj. clexane 0.6 ml BD after stopping of UFH infusion and shifted to ward.

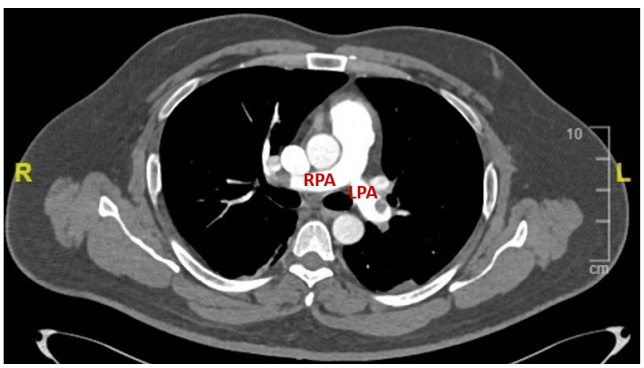

Figure 1: Multiple filling defects in distal RPA, distal RPA extending into lobar, segmental and subsegmental bronchus of pulmonary artery.

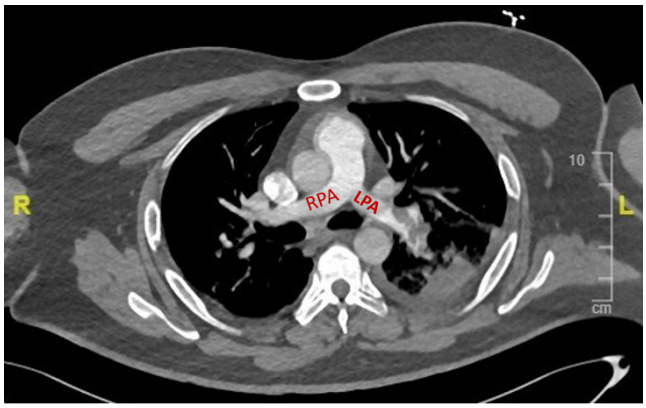

Figure 2: Multiple filling defect in distal LPA extending into lobar, segmental and subsegmental bronchus of pulmonary artery with left lower lobe infarct (Second CTPA).

Discussion

Perioperative acute pulmonary embolism is relatively rare complication but it is very severe and life-threatening situation. Further, diagnosing intraoperatively is very difficult as it can be obscured by the anesthesia. The most common cause of PE remains deep vein thrombosis [1].

Surgical patients have multiple risk factors for PE, which include inflammation caused by tissue injury, activation of the clotting cascade, and venous stasis due to immobilization. The incidence ranges from 0.3% to 1.6%. There is five-fold increased risk of developing VTE, especially during perioperative and post -operative period [2]. But incidence of PE after all orthopedic surgical procedures has been reported as 0.7% to 30% and 4.3% to 24% following hip fracture repair [3].

Our patient had acetabular trauma. Since it was an acetabulum surgery in trauma patients, the cause of acute pulmonary thromboembolism might be trauma per se. Trauma-induced pulmonary thromboembolism is the second most cause of death in severe trauma patients and if injured patients survive for the first 24 hours, the third most common cause of death is found to be fatal PE [4,5]. The incidence of post-traumatic VTE can be up to 13 times more than that of non-traumatic patients [4]. The incidence of post-traumatic VTE varies considerably from 0.27% to 65% as reported in multiple studies and remains very high after major traumas even with the initiation of prophylactic antithrombotic therapy as early as possible. A study by American College of Surgeons Trauma Quality Improvement Program (TQIP) for patients reported 0.6% PE in trauma patients having Abbreviated Injury Scale (AIS) of at least three in at least one body region [6].

Acute PE interferes with both circulation and gas exchange. The obstruction of pulmonary artery causes pressure overload in the right ventricle (RV), leading to RV expansion and inflammatory reaction. The oxygen demand of RV increases leading to ischemia of

RV, decreasing its contractility and hence the cardiac output. Also, LV preload also decreases leading to hypotension and decreased coronary perfusion. This creates a vicious cycle [7]. With various symptoms of PE like chest pain, syncope, altered mental status, a sudden decrease in $EtCO_2$ is an early sign of potential PE for patients undergoing general anaesthesia as occurred in our case. This is reportedly described as "separation phenomenon" during general anaesthesia, which means a decrease trend in $EtCO_2$ and increase in $PaCO_2$. Hence, we suggest strict monitoring of $EtCO_2$ and arterial blood gas analysis especially when $EtCO_2$ decreases without any other cause during general anesthesia [8].

CECT is known as the gold standard for diagnosing PE but perioperatively difficult to do. Recently, point of care TTE has emerged as a powerful and rapid noninvasive diagnostic tool in acute PE. TTE coupled with $EtCO_2$ changes can be useful during intra-operative period, in high-risk patients. Strict monitoring of $PETCO_2$ and arterial blood analysis especially when PETCO2 decreases without other reasons during general anaesthesia, should be done [8]. In TEE an abnormal echoic area in pulmonary artery (considering thrombus) can be visualised. D-dimer has an important value of excluding VTE but its predictive value is limited in the setting of trauma and postoperative cases. Elevated D-dimer levels can be seen in various conditions, including trauma and postoperative states, which may limit its specificity for PE. Timely investigation of CTPA was done in our case which was beneficial [9]. Thrombolysis of the clot, DVT prophylaxis, anticoagulation, ECMO or embolectomy are various treatment options available. European Society of Cardiology (ESC) strongly recommend intravenous UFH as a treatment of choice in haemodynamically unstable APE. Catheter directed therapy is recommended by the American College of Chest Physicians as second-line therapy for massive pulmonary embolism [10].

Despite the major drawback of bleeding like intracranial haemorrhage, thrombolytic treatment is beneficial as it restores pulmonary perfusion more rapidly than anticoagulation alone. Further, the higher age and presence of comorbidity also increases the risk of major bleeding. Since our patient underwent major surgery, thrombolytic therapy given after 24 hours. Also, after discussion with cardiology team and patient's family members, anticoagulation was started as soon as early possible. Patient was discharged uneventfully after 10 days.

Prevention of PE should be the goal for clinicians as compared to cure. Early starting of ambulation, application of elastic stocking, and intermittent pneumatic compression devices are easy way for prevention.

Conclusion

Perioperative pulmonary embolism is rare but a life-threatening condition. Trauma increases the risk of PE depending upon the severity of injury. Strict monitoring of $PETCO_2$ and arterial blood gas analysis should be done while ruling out other causes of decrease $ETCO_2$. During general anesthesia. Early detection and treatment are vital for a better prognosis.

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