



Intraoperative acute pulmonary thromboembolism with rare presentation of recurrent bradycardia-A Rare Case

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Abstract

Acute pulmonary thromboembolism (APTE) can be life-threatening. Early detection is more difficult for patients who lack of pathognomonic signs and symptoms of pulmonary embolism (PE). We report a case of orthopedic surgery under combined spinal epidural anesthesia (CSE). A 47 year-old man underwent surgery for fracture femur under CSE. No arterial and deep vein thrombosis (DVT) was found prior to the surgery, presented with the hypotension, recurrent bradycardia, and final diagnosis of PE made during intraoperative period. In the absence of drug reaction, acute MI and AV blocks, sudden onset of recurrent bradycardia and shock followed by sudden decrease in Pet CO₂ and increase in PACO₂, we should pay more attention to PE, which is an important differential diagnosis of refractory bradycardia with shock. In this context, rapid ECHO and USG of deep veins should be done to rule out APTE and underlying DVT respectively in operation theatre. Multidisciplinary consultation was started immediately. After discussion with the consultation team anticoagulation therapy was started and surgery was withheld and patient shifted to AICU but patient succumbed to death due to refractory bradycardia and shock inspite of treatment for pulmonary thromboembolism as per guideline. Pulmonary embolism is a rare and potentially high-risk perioperative situation, with a difficult diagnosis when presented with bradycardia and hypotension. The separation phenomenon of decrease in Pet CO₂ and increase in PaCO₂ might be a useful and suggestive sign along with ECHO and USG if ECG is not showing typical changes of PTE.

Keywords: *Interoperative, acute pulmonary thrombo embolism, bradycardia, hypotension, combined spinal epidural anesthesia.*

Introduction

Pulmonary thromboembolism (PTE) is a relatively common cardio-vascular emergency that causes obstruction of the pulmonary vascular bed and can leads severe acute failure of the right ventricle (RV) which is potentially and rapidly lethal.¹

Prompt diagnosis is essential since immediate therapy is very effective. Depending on the clinical presentation, initial therapy is primarily directed to restore circulation to the obstructed pulmonary arteries.² The diagnosis is difficult to reach and may be complicated by an atypical

clinical presentation. Common signs and symptoms of pulmonary embolism include dyspnea, pleuritic chest pain, tachycardia and tachypnea. Recurrent short episodes of bradycardia and hypotension are rarely reported as clinical manifestations of PTE. We describe an intra-operative case of acute massive PTE presenting with dyspnea, burning chest pain and recurrent bradycardia and hypotension when patient was in combined spinal anesthesia. Due to refractory bradycardia and hypotension and fall in spo2 patient was converted from CSE to GA. But before completion of surgery patient developed sudden cardiac arrest and patient revived with CPR. Surgery was stopped and patient shifted to AICU where thrombolysis started after diagnosing acute PTE on the basis of ECHO finding, D-dimer value. CT angiography thorax could not be got done due to hemodynamic instability of our patient. Before completion of thrombolysis patient succumbed to death due to refractory severe bradycardia and hypotension which leads to cardiac arrest.

Case Report

A 47 years old male with fracture shaft of femur, fracture distal end of radius and fracture of proximal phalanx right thumb. Pt. was admitted in MOW in Dr. RPGMC Kangra at Tanda H.P. After preanesthetic check he was posted for elective surgery. Patient was not known case of diabetes mellitus, hypertension, cardiac disease, thyroid dysfunction and cancer. On examination airways-Mets>4, MPS-11, TMD >6.5, MO> 3FB, NM-adequate, No loose teeth, chest B/1 VBS, CVS-S1& S2 (N), HB-11.2 gm.%,RBS -98mg/dl , urea-22 mg and creatine 0.8mg. Serum electrolytes-sodium 140meq/l and potassium 5.3 meq/l respectively. Platlet-163000mm³, TFT within normal, PTI/INR 14/1.2 respectively. CRX-grossly normal, ECG-normal, USG FAST-normal, D-Dimer 250 microgram per liter. NCCT head suggestive of normal study.

Patient was taken to operating room and all routine monitor viz ECG, NIBP, SPO2 are

attached. Intravenous lines are secured. Pre op. vital- BP 120/74mm hg, PR-100/min SPO2-93%on room air. CSE anesthesia was administered to the patient.

During intraoperative period pt. suddenly destaurated & sudden hypotension and bradycardia occurred for which atropine 0.6 mg was given. Patient was taken on Bain circuit with 100% oxygen@ 6-8 l/min. Patient remained unresponsive. Patient was intubated with 7.5 mm id ETT and fixed at 20 cm. B/L air entry equal. BP was 74/36 mmhg. Infusion of dopamine started @10micr/kg/min. Patient still having bradycardia HR-50/min and injection atropine was repeated and patient still having bradycardia HR -36-25/min. CPR started and injection adrenaline 1mg was given. HR increased to 108/min (sinus tachycardia).

Patient again having episode of bradycardia. HR-30, BP-50/30. CPR started, injection adrenaline given and infusion of nor-adrenaline started @ 1 micro/kg/min and HR returned 128/ min, BP-76/36. Surgery was abandoned and patient was shifted to AICU. Post operative HB, S. electrolytes, RFT, CK-MB, TROP-T, D-dimer, ECHO was done. Echo was showing- RA/RV dilated, TAPSE<17MM, RV SYST DYSFUNCTION AND NORMAL LV SYST FUNCTION, CK-MB was 68 u/l, troponin-T was detected, coagulation profile was normal and d-dimer was >2500 ng/dl. Thrombolysis with streptokinase started after consultation of physician and as per guideline. After two hours patient had an episode cardiac arrest and CPR done but pt. could not be revived. Autopsy was done and massive pulmonary embolism was found. Probable cause of PE in our case might be due to prolonged bed rest for > 5 days. No thromboprophylaxis was given at the time of admission. Long bone fracture itself may be another cause of thromboembolism, dislodgement of clot during reaming of intramedullary cavity may be the reason.

Discussion

Predisposing conditions for PTE are multiple trauma, long bone fractures, pregnancy, previous heart surgeries and chronic deep venous insufficiency/prior venous thrombosis.⁴

Symptoms of PE being non-specific, high index of suspicion is required, especially in presence of risk factors. Massive PE may present as shock with systemic hypotension, poor perfusion of extremities, tachycardia, tachypnea, pallor, sweating, oliguria and impaired mentation.⁵ Our patient was immobilized for 5 days following fracture femur. It is risk factor of pulmonary thromboembolism but no thrombus was found before surgery. The initial hypotensive response observed intraoperative and was thought to be due to antibiotic administration. Hence, Inj. Ephedrine, Inj. Hydrocortisone and Inj. Avil were given as anaphylactic prophylaxis, despite the absence of bronchospasm or urticaria. Since it was a femur surgery, the intraoperative APTE might be caused by embolus released during femoral reaming. The deterioration in hemodynamic status, gasping respiration, fall in SpO₂ and EtCO₂ and extreme pallor led to the suspicion of PE. Intraoperative ECHO, D-dimer value s/o APTE. To confirm the cause APTE need further tests, like CT angiography. It was not conducted in this case due to critical condition of patient. Combining all these findings, the most possible reason for refractory bradycardia and hypotension was APTE. The treatment required accurate and fast risk assessment. Thrombolysis restores pulmonary perfusion more rapidly as compare to anticoagulation alone. It carries risk of major bleeding including intracranial hemorrhage. Surgery itself is relative contraindication to thrombolysis. After consultation thrombolytic treatment was started with injection streptokinase. Instead of initiation of thrombolysis patient went to cardiac arrest again and succumbed to death even after CPR. Despite update guideline, PE remains an important clinical problem with high mortality rate. Even with prophylaxis it could still happen and sometime appear as an emergency.

Early diagnosis of acute massive PE depends on clinical feature. D-dimer concentration (>500ng/ml) has 90% sensitivity in detecting PE and concentration of < 500ng/ml rules out PE in 98-100% of cases.⁶ Trans esophageal echocardiography (TEE) can identify central PE and also serve prognostic function (mortality rate 10% in presence of right ventricle dysfunction. Spiral CT scan of the chest has 94% sensitivity and specificity in evaluating PE.⁷ Ischemia-modified albumin, IMA (93% sensitive and 73% specific) is a potential alternative to D-dimer testing. Diagnosing PTE takes time and is expensive. We did not perform TEE as it is not routinely used in our setup. Thrombolytic therapy should be used in patients with acute PTE associated with hypotension (systolic BP < 90mm Hg), who do not have a high bleeding risk.⁷ Inferior vena cava filters may be used. Emergency pulmonary artery embolectomy is indicated in presence of severe hemodynamic instability or contraindication to thrombolytic therapy, but usually requires the institution of cardiopulmonary bypass which is not available in our rural tertiary care institution. Newer successful techniques include fluoroscopic-guided catheter fragmentation. External chest compressions may also help to fragment a massive PE.

Conclusion

PE is rare an acute fatal perioperative situation with difficult diagnosis when presented with atypical signs and symptoms like bradycardia, syncope and hypotension. Prompt diagnosis is of utmost importance. We conclude that awareness of the possibility of PE, prompt confirmation of diagnosis and therapeutic intervention can decrease mortality. Whenever possible, noninvasive diagnostic tools, such as TEE, USG of peripheral veins and spiral CT of the chest should be used for diagnosis and observing the progress. In our case there was a need of surgical pulmonary embolectomy but this was limitation of our institution. Recurrent bradycardia with

hypotension must be considered as differential diagnosis of pulmonary thromboembolism.

Reference

1. GH Pharo, A Andonakakis, K Chandrasekaran, G Amron, JD Levitt. Survival from cata-strophic intraoperative pulmonary embolism. *AnesthAnalg* 1995, 81:188-90
2. Kenneth Lewis, Adejare Windokun, Peregrina Arciaga, Kiumars Arfai, Monika Soni. Intra-operative pulmonary embolism presenting as a widened PACO₂-ETCO₂ gradient. *Chest*.2006: 130 (4_MeetingAbstracts):295S
3. Dohring DJ, Arens JF. Pulmonary thromboembolism disease recognition and patient management. *Anesthesiology* 1990; 73:146-64.
4. S Mishra, P Kundra, B Hemavathi, A Badhe, M Ravishankar. Postoperative Acute Pulmonary Embolism: A Case Report. *The Internet Journal of Anesthesiology*.2006 Volume 13 Number 2.
5. I G Livaditis, M Paraschos, K Dimopoulos. Massive pulmonary embolism with ST elevation in leads V1–V3 and successful thrombolysis with tenecteplase. *Heart*. 2004 July; 90(7): e41.
6. Nitin Chopra, Pradeep Doddamreddy, Hermanjeet Grewal, and Pratap C Kumar An elevated D-dimer value: a burden on our patients and hospitals. *Int J Gen Med*. 2012; 5: 87–92.
7. Turedi S, Gunduz A, Mentese A, Topbas M, Karahan SC, Yeniocak S, et al. The value of ischemia-modified albumin compared with d-dimer in the diagnosis of pulmonary embolism. *Respir Res*. May 30: 2008;9:49.