

Case Report:

Intraoperative Pulmonary Embolism in a Hypertensive patient with Pacemaker in Situ - a Case Report

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Abstract

Pulmonary embolism (PE) is a potentially lethal condition. Most patients who succumb to pulmonary embolism do so within the first few hours of the event. Despite diagnostic advances, delay in diagnosing PE is common and represents an important issue. In these patients, recurrent embolism and death can be prevented with prompt diagnosis and therapy. Unfortunately, the diagnosis is often missed because they present with non-specific signs and symptoms. Here we present a case of an elderly female patient who sustained intra-operative massive pulmonary embolism leading to cardiac arrest during bipolar hemiarthroplasty for intertrochantric fracture femur.

Keywords: Pulmonary embolism, bipolar hemiarthroplasty, intra-operative, D-dimer

Introduction

PE is the third most common cardiovascular disease, following myocardial disease and stroke accounting for 15-20% of all in-hospital deaths¹. Intraoperative PE is relatively uncommon, but may occur with specific surgeries, such as total joint arthroplasty, pelvic surgery, caesarean section, fixation of long bone fractures and tumour surgeries. It usually presents as sudden cardiovascular collapse. Acute massive PE causes death of 50% patients within 15 minutes and only 33% survive over two hours².

Pulmonary thromboembolism is not a disease by itself, but rather a complication of underlying venous thrombosis in the deep venous system of the lower extremities and rarely the pelvic, renal, upper extremity veins, or the right heart chambers. Normally, micro-thrombi are continuously being formed and lysed within the venous circulatory system. Larger thrombi may, before travelling to the lungs, lodge at the bifurcation of the main pulmonary artery or at the lobar branches causing hemodynamic compromise.

Case Report

A 75yr old female, weighing 40kgs, was brought to the hospital with left intertrochanteric fracture femur following trauma three days ago. She was on regular treatment for hypertension and ischaemic heart disease since three years and had a permanent pace maker in situ since three months. Patient also had chronic obstructive pulmonary disease. Routine investigations were normal, except haemoglobin 8.8gm%, right bundle branch block with inverted t-waves in all leads on electrocardiogram and echocardiography showed ejection fraction 55% with mild pulmonary hypertension. After correcting anaemia, obtaining fitness from physician and preoperative nebulisation, patient was posted for bipolar hemiarthroplasty

Written informed consent was taken, adequate starvation confirmed. Standard ASA monitors attached in operation theatre and spinal-epidural anaesthesia was administered. Intra-operatively, decision to use a cemented prosthesis was taken. Six minutes into cementing, patient was found unresponsive, with pulse rate 75bpm, feeble; blood pressure (BP) 90/60mmHg, oxygen saturation (SpO₂) 100%. Inj. ephedrine 6mg and Inj. Avil 1amp intravenous stat was given. Within seconds, her respiration became shallow and radial pulse non-palpable. Ventilation was assisted with 100% oxygen with bag and mask and Inj. ephedrine 6mg i.v. was repeated. ECG showed pacemaker rhythm and BP was 70/40mmHg. Surgery was abandoned immediately, patient turned supine and intubated. During this period, she developed ventricular fibrillation. BP and SpO₂ were non-recordable. End-tidal carbon dioxide (ETCO₂) was 5mmHg. Air entry was bilaterally equal. Immediately, DC shock was given and CPR started. After 4 DC shocks with intermittent CPR, Inj. adrenaline(1mg i.v.) three doses at adequate intervals, Inj. Isoprenaline 0.02mg and Inj. Amiodarone 150mg i.v. bolus, patient was revived in ~40 minutes and BP recorded 130/60mmHg. Ten minutes later, her BP dropped again to 98/30mmHg. Femoral line was secured, noradrenaline(2µg/kg/min) and dobutamine(20µg/kg/min) infusions started and patient was shifted to intensive care unit. Post-event, D-Dimer level was 2980, TLC20, 300/cumm.ABG showed respiratory alkalosis. Supportive treatment was instituted. In spite of inotropic support, her BP remained low. Chest auscultation revealed crepitations. Few hours later, she suffered a cardiac arrest again, but could not be revived.

Autopsy revealed massive pulmonary embolism.

Discussion

PE is the implantation of material, chiefly clots dislodged from peripheral veins into the branches of the pulmonary arterial bed, but can also consist of fat emboli, neoplastic cells, amniotic fluid, pieces of catheters or exogenic material that enter the veins. Endothelial injury, stasis/turbulence of blood, hypercoagulability (classical triad of Virchow) predispose to thrombus formation³.PE occurs most commonly in patients undergoing emergency surgery following trauma.

Other predisposing conditions are multiple trauma, long bone fractures, pregnancy, previous heart surgeries and chronic deep venous insufficiency/ prior venous thrombosis⁴. 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, tachypnoea, pallor, sweating, oliguria and impaired mentation⁵. Signs of pulmonary hypertension may be present. Our patient was immobilised for 5 days following fracture femur. Therefore, possibility of deep venous thrombosis was extremely high. The initial hypotensive response observed intra-operatively 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. But the deterioration in hemodynamic status, gasping respiration, fall in SpO₂ and EtCO₂ and extreme pallor led to the suspicion of PE. The patient's circulatory status deteriorated, in spite of aggressive hemodynamic support, eventually proving to be fatal. Though minimally invasive diagnostic strategies are available, early diagnosis of acute massive PE depends on clinical features. D-dimer concentration (>500ng/ml) has 90% sensitivity in detecting PE⁵ and concentration of < 500ng/ml rules out PE in 98–100% of cases³. Trans-oesophageal echocardiography (TEE) can identify central PE and also serve prognostic function (mortality rate~10% in presence of right ventricle dysfunction)⁵. Spiral CT scan of the chest has 94% sensitivity and specificity in evaluating PE⁶. Ischaemia-modified albumin, IMA (93% sensitive and 73% specific) is a potential alternative to D-dimer testing.⁷ Diagnosing PE takes time and is expensive. We did not perform TEE as it is not routinely used in our setup. Further, patient could not be moved for CT scan or angiography. Treatment for massive PE remains controversial. Thrombolytic therapy should be used in patients with acute PE associated with hypotension (systolic BP < 90mm Hg), who do not have a high bleeding risk⁸. 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. Newer successful techniques include fluoroscopic-guided catheter fragmentation. External chest compressions may also help to fragment a massive PE.

Conclusion

PE is an acute fatal condition. Prompt diagnosis is of utmost importance but difficult, because of nonspecific signs and symptoms and because of the unstable haemodynamic condition, making it an acute emergency. We conclude that awareness of the possibility of PE, prompt confirmation of diagnosis and therapeutic intervention can decrease mortality. Whenever possible, non-invasive diagnostic tools, such as TEE and spiral CT of the chest should be used for diagnosis and observing the progress. In this case, a preoperative D-dimer assay would have helped.

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