

Management of a patient with perioperative saddle embolus

Austin Lee, Jaime Ortiz

Austin Lee, Jaime Ortiz, Department of Anesthesiology, Baylor College of Medicine, Houston, TX 77030, United States

Author contributions: Lee A and Ortiz J participated in the case and wrote entire manuscript.

Correspondence to: Jaime Ortiz, MD, Department of Anesthesiology, Baylor College of Medicine, 1709 Dryden Way, Suite 1700, Houston, TX 77030, United States. jaimeo@bcm.edu
 Telephone: +1-713-8732860 Fax: +1-713-8732867

Received: February 28, 2013 Revised: March 20, 2013

Accepted: March 25, 2013

Published online: March 27, 2013

Abstract

Pulmonary embolism is a major cause of morbidity and mortality. Risk factors include venous stasis, endothelial injury, and hypercoagulability. Prevention centers on the use of sequential compression devices and anticoagulation in the hospital patient. This is the case of a 45-year-old male who presented for open reduction and internal fixation of tibia plateau fracture. He developed a saddle embolus during the perioperative period which was diagnosed in the recovery room after workup for the cause of his poor oxygenation. A chest computed tomographic scan showed an extensive saddle embolus with partial occlusion of the bilateral main pulmonary arteries and all segmental pulmonary artery branches. This case report discusses his diagnosis, management and clinical course. In addition, risk factors, treatment and prevention for pulmonary embolus and described.

© 2013 Baishideng. All rights reserved.

Key words: Venous thromboembolism; Pulmonary embolus; Saddle embolus; Tibia fracture; Anesthesia

Core tip: The case report describes the diagnosis, management and treatment of a 45-year-old male who developed a saddle pulmonary embolus during open reduction and internal fixation of tibia plateau fracture. The incidence of pulmonary embolism, risk factors, and

treatment and prevention choices are discussed.

Lee A, Ortiz J. Management of a patient with perioperative saddle embolus. *World J Anesthesiol* 2013; 2(1): 11-13 Available from: URL: <http://www.wjgnet.com/2218-6182/full/v2/i1/11.htm>
 DOI: <http://dx.doi.org/10.5313/wja.v2.i1.11>

INTRODUCTION

Venous thromboembolism clinically manifested as deep vein thrombosis and pulmonary embolism, is a common cause of increased morbidity and mortality. Pulmonary embolism (PE) can manifest in a variety of clinical settings ranging from acute shortness of breath in the emergency department to intraoperative cardiovascular collapse, hypoxemia in the recovery room, to respiratory distress in the intensive care unit. In the perioperative setting, it is critical for the anesthesiologist to have a high index of suspicion for PE as symptoms and signs are often vague and non-specific. To do so, one must know the medical and surgical risk factors that can increase the probability of PE as well as the prophylactic measures that can be used to diminish the risk. In addition, it is critical to know the management of pulmonary embolism in the perioperative setting.

CASE REPORT

A 45-year-old African American male was admitted to the hospital with a left tibia plateau fracture after a motor vehicle accident. He had no other injuries from the accident and had no previous medical history other than 20 pack-year history of smoking tobacco. The patient was admitted and started on enoxaparin 40 mg subcutaneously once a day for deep venous thrombosis prophylaxis. On hospital day 3 he underwent open reduction and internal fixation of the left tibia plateau fracture. The surgical procedure was uneventful. A laryngeal mask airway was

used initially, but was subsequently switched to an endotracheal tube after difficulty with placement and difficulty maintaining adequate ventilation and oxygen saturation. After placement of the endotracheal tube, ventilation and oxygenation went back to normal and the procedure proceeded as planned.

The patient was subsequently extubated at the end of surgery. Although he was awake and was ventilating well, he continued to have O₂ saturations in the mid 80 to 90 s with 100% non-rebreather mask along with sustained tachycardia in the 110 to 120 s even after adequate pain control. An arterial blood gas was drawn in the post anesthesia care unit and it showed a pH of 7.40, pCO₂ of 41.6 mmHg, pO₂ of 61.0 mmHg, bicarbonate of 25.1 and oxygen saturation of 91%. A chest X-ray was taken showing an interval development of a right upper lobe opacity concerning for pneumonia or aspiration when compared to his admission chest X-ray. A computed tomography scan of the chest was performed and an extensive saddle embolus with partial occlusion of the bilateral main pulmonary arteries and all segmental pulmonary artery branches with significant thrombus burden. In addition, a right upper lobe dense airspace consolidation with peripheral ground glass opacification along the right posterior segmental bronchus and bronchus intermedius which was suspicious of infarction was found. An echocardiogram was then performed showing a severely dilated right atrium and right ventricle. However, he had normal left ventricular function and his ejection fraction was between 55%-60%. Bilateral lower extremity Doppler studies showed occlusive thrombi in the left common femoral and superficial femoral veins.

The patient was started on a heparin infusion and subsequently had an inferior vena cava filter placed. He remained in the intensive care unit for several days until his oxygen demand was reduced from 100% oxygen *via* non-rebreather mask to nasal cannula. He never required intubation to support his oxygenation and ventilation and remained hemodynamically stable throughout his hospital stay. He was transitioned to the regular nursing floor without need for oxygen supplementation and soon discharged on warfarin with follow up at the Pulmonary and Orthopedics clinics. On follow up, he had returned to normal function and recovered from his surgical procedure well.

DISCUSSION

Our patient was able to fully recover from an event that can often be lethal. It is unknown at which point after his tibia fracture the patient developed his blood clot and pulmonary embolism. We were able to begin treatment as soon as the diagnosis was made. It also helped that our patient was healthy and physically fit before this event took place.

The incidence of mortality after pulmonary embolism has been reported to be as high as 300000 per year^[1]. This number understates the significant morbidity including chronic pulmonary hypertension, disability, and im-

paired quality of life that affects survivors. It is estimated that the economic burden of pulmonary embolism is greater than \$1.5 billion a year in healthcare costs with estimates stating that each pulmonary embolism results in additional healthcare costs in excess of \$30000^[1]. The three-month mortality of pulmonary embolism has been stated as high as 15%-18%^[1]. While most patients with acute pulmonary embolism survive, possible long term sequelae include chronic thromboembolic pulmonary hypertension and chronic leg pain and swelling.

It is important for all medical personnel to be aware of patients at higher risk for developing venous thromboembolic disease. Risk factors that acutely increase the risk of pulmonary embolism include orthopedic surgeries, especially total hip and knee replacement, surgery for hip fractures, as well as trauma and spinal cord injuries. Acute medical morbidity, especially malignancy is also a major risk factor.

A good approach to understanding all the risk factors that predispose patients to pulmonary embolism is to understand Virchow's triad^[2]. Virchow's triad states that venous stasis, endothelial injury, and hypercoagulability will increase the risk of thrombosis^[2]. Venous stasis can occur when patients are immobile (*i.e.*, spinal cord injury, trauma, orthopedic fractures) or when there is a problem with the pump (*i.e.*, heart failure). Surgical procedures are a major culprit to endothelial injury, as are invasive catheter-based procedures including angiograms and placement of transvenous pacemakers. Hypercoagulability can be hereditary such as deficiency or mutation of certain factors (prothrombin, protein C, protein S) or from pro-inflammatory states (malignancy, myeloproliferative syndromes, antiphospholipid antibodies, hyperhomocysteinemia, heparin-induced thrombocytopenia, acquired immunodeficiency syndrome (AIDS), burns, lupus, oral contraceptives)^[1].

Orthopedic patients have a high risk from development of deep vein thrombosis and pulmonary embolism. A study by Geerts *et al*^[3] showed an incidence of deep venous thrombosis (DVT) of 69% in patient with lower extremity fractures. This made our patient with a tibia fracture at high risk for development of DVT. As is customary with most trauma patients at our hospital, he was on enoxaparin for DVT prophylaxis up until the morning of his surgery. However, it did not prevent him from developing a saddle pulmonary embolus.

When acute pulmonary embolism is present, parenteral anticoagulation with either low molecular weight heparin or unfractionated heparin should be administered, unless contraindicated^[4,5]. The primary indications for placement of an inferior vena cava filter are contraindications to anticoagulation, risk of major bleeding during anticoagulation, and recurrent embolism while receiving adequate therapy^[4]. Filters are also considered in cases of massive pulmonary embolism, with the thought being that additional thrombotic burden may be life threatening. In cases of massive pulmonary embolism with cardiovascular collapse, requiring cardiopulmonary

resuscitation and blood pressure support, mortality is much higher. A retrospective review performed at Texas Heart Institute by Konstantinov *et al*^[6] showed that emergency treatment using cardiopulmonary bypass may be beneficial. Rapid recognition and treatment are very important to prevent severe morbidity and mortality after pulmonary embolism.

Current methods used to prevent DVT and PE have significantly reduced the incidence of fatal PE^[7]. Treatments that combine mechanical prophylaxis such as, sequential compression devices or inferior vena cava filters, with low molecular weight heparin appear to be most effective^[7]. However, even patients on appropriate prophylaxis are still developing DVT and PE. Future research will hopefully help with better prevention and treatment of pulmonary embolism.

In conclusion, we presented the case of a 45-year-old male who developed a saddle pulmonary embolism after tibia fracture during the perioperative period. Prompt diagnosis and treatment prevented morbidity and mortality in this patient.

REFERENCES

- 1 MacDougall DA, Feliu AL, Boccuzzi SJ, Lin J. Economic burden of deep-vein thrombosis, pulmonary embolism, and post-thrombotic syndrome. *Am J Health Syst Pharm* 2006; **63**: S5-S15 [PMID: 17032933 DOI: 10.2146/ajhp060388]
- 2 Virchow RLK. Collected essays on scientific medicine [in German]. Frankfurt Am Main, 1856
- 3 Geerts WH, Code KI, Jay RM, Chen E, Szalai JP. A prospective study of venous thromboembolism after major trauma. *N Engl J Med* 1994; **331**: 1601-1606 [PMID: 7969340 DOI: 10.1056/NEJM199412153312401]
- 4 Torbicki A, Perrier A, Konstantinides S, Agnelli G, Galiè N, Pruszczyk P, Bengel F, Brady AJ, Ferreira D, Janssens U, Klepetko W, Mayer E, Remy-Jardin M, Bassand JP. Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). *Eur Heart J* 2008; **29**: 2276-2315 [PMID: 18757870 DOI: 10.1093/eurheartj/ehn310]
- 5 Prandoni P. Anticoagulant treatment of pulmonary embolism: impact and implications of the EINSTEIN PE study. *Eur J Haematol* 2012; **89**: 281-287 [PMID: 22834998 DOI: 10.1111/ejh.12002]
- 6 Konstantinov IE, Saxena P, Koniuszko MD, Alvarez J, Newman MA. Acute massive pulmonary embolism with cardiopulmonary resuscitation: management and results. *Tex Heart Inst J* 2007; **34**: 41-45; discussion 45-46 [PMID: 17420792]
- 7 Agudelo JF, Morgan SJ, Smith WR. Venous thromboembolism in orthopedic trauma patients. *Orthopedics* 2005; **28**: 1164-1171; quiz 1164-1171 [PMID: 16237881]

P- Reviewers Mert T, Zhang RX, Konstantinou EA
S- Editor Gou SX **L- Editor** A **E- Editor** Lu YJ

