Near Fatal Pulmonary Embolism

A El-Dawlatly, S AlMajid, M Elsherbeny, W Alotaiby, F Shakeel

Citation

A El-Dawlatly, S AlMajid, M Elsherbeny, W Alotaiby, F Shakeel. *Near Fatal Pulmonary Embolism*. The Internet Journal of Anesthesiology. 2003 Volume 8 Number 2.

Abstract

A 37-year-old female patient was admitted for elective caesarean section (CS). Her laboratory and biochemical investigations were normal. She underwent CS due to large size of the baby under spinal analgesia. On the first postoperative day, she developed acute shortness of breath, cyanosis, sweating and cardiac arrest. She was resuscitated successfully with oxygen via facemask, cardiac massage and i.v fluids. ECG showed supraventricular tachycardia and incomplete right bundle branch block. Echocardiography revealed a mildly dilated left and right ventricle with 50mmHg mean pulmonary artery pressure (PAP). Spiral CT scan confirmed the diagnosis of saddle pulmonary embolism (PE). Dublex ultrasound was negative for leg and pelvic deep venous thrombosis (DVT). She was admitted to surgical intensive care unit (SICU) and received adenosine and labetalol to control cardiac arrhythmia. Her blood pressure was consistently low where she received intermittent boluses of ephedrine and adrenaline. She was kept on dobutamine i.v infusion drip. After a cardiac surgeon consultation, surgical removal of PE was abandoned due to stable vital signs and high risk of surgery. Full heparinization was started with i.v 5000U then 900-1200U/h. Thrombolytic therapy was not given due to the recent surgery and bleeding complication. Subsequent spiral CT scan revealed slight improvement and partial revascularization of the pulmonary vascular bed. Repeated echocardiography showed mild dilated right ventricle, and 30mmHg PAP. She is now on the 20th postoperative day on warfarin 2-5mg/day. So far she is stable and making slow recovery.

INTRODUCTION

PE is common in pregnancy and occurs in one out of every 2000 pregnant women with high mortality rate ($_1$). This report describes a case of near fatal saddle PE following CS under spinal analgesia.

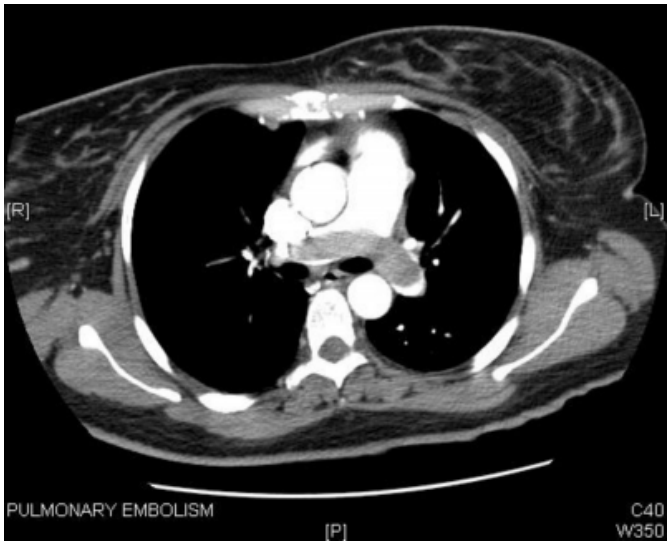
CASE REPORT

A 37-year-old female patient was admitted for elective caesarean section (CS) due to large fetal size. She was para gravida 6. She had a history of gestational diabetes during the previous pregnancies controlled with diet. She had no previous operations. Family history was suggestive of two incidences of cerebral strokes to her mother. Her preoperative laboratory and biochemical investigations were normal. She underwent CS under spinal analgesia. Spinal analgesia was performed at L3-4 and block was achieved with 2.5ml of 0.5% bupivacaine and 25mcg fentanyl. The level of sensory block reached up to D4. The operation was uneventful with minimal blood loss. The baby delivered with Apgar score of 15.

On the first postoperative day, she developed acute shortness of breath, cyanosis, sweating and cardiac arrest. She was resuscitated successfully with external cardiac massage, oxygen via facemask and i.v fluids. Following resuscitation, the ECG showed supraventricular tachycardia, incomplete right bundle branch block and inverted T wave in leads V1-V4. Her laboratory and biochemical investigations including LDH, SGOT and CK were normal. Arterial blood gases with FiO_2 0.6 showed, pH 7.4, $PaCO_2$ 27.3mmHg, PaO_2 73.4mmHg, bicarbonate 17.2mmol/l and O_2 saturation 94.8%. Echocardiography revealed a dilated right ventricle, moderate tricuspid regurgitation and PAP 50mmHg.

Spiral CT scan showed a filling defect in the main pulmonary artery and first branches consistent with PE (Fig 1).

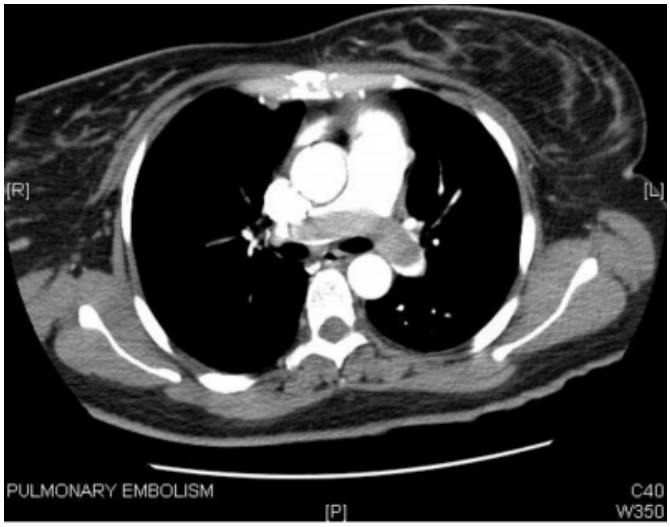
Figure 1



Dublex ultrasound was negative for leg and pelvic deep venous thrombosis (DVT). She was admitted to the SICU. Supraventricular arrhythmia was treated with i.v adenosine 6mg twice and labetalol 5mg incremental dosages. Her blood pressure was low with 70/50mmHg. She received intermittent boluses ephedrine and adrenaline followed by dobutamine continuous infusion at a rate of 3-5mcg/kg/min. Afterward her blood pressure improved. The patient was referred to a cardiac surgeon consultation, where surgical removal of PE was abandoned due to stable vital signs and high surgical risk encountered. The option of thrombolytic therapy was not given due to the recent surgery and bleeding complications. Thereafter, full heparinization was started with i.v 5000U then 900-1200U/h to keep APTT between 60-80sec. She was kept in bed with minimal mobilization and started on antibiotics.

Subsequent spiral CT scan revealed slight improvement and partial revascularization of the pulmonary vascular bed with right-sided pleural effusion (Fig 2).

Figure 3



Repeated echocardiography showed mild dilated left ventricle, and PAP 30mmHg. On the 9 th postoperative day oral warfarin has replaced heparin in a dose of 10mg and then 2-5mg/day to keep INR between 2-3 times its normal value. On the 15 th postoperative day a repeated spiral CT scan revealed partial recovery and more revascularization of the pulmonary bed compared with the previous one (Fig 3).

{image:3}

She is now on the 20 th postoperative day on warfarin therapy, stable hemodynamics and making slow recovery.

DISCUSSION

Thromboembolic disease during pregnancy is common. For many years it was limited to the third trimester, however, many recently published reports indicated that the risk of PE is increased throughout pregnancy ($_{2}$). Age over 30, obesity, multiparity, prolonged bed rest, and CS all predispose to embolism ($_{3}$, $_{4}$). In the present case report, the patient hadtwo risk factors, multiparity and she underwent CS. Patients who have undergonegynecological surgery, may have DVT that start at any location. Massive PE is one of the most common causes of unexpected death, thought fatal, accurate diagnosis and timely treatment can reduce the mortality rate.

It is a clinically challenging diagnosis with no decline in its incidental discovery at autopsy over the past 30 years ($_{5}$). Acute major PE is associate with right ventricular function and shock ($_{6}$). In the present report, the patient had mild to moderate right ventricular dilatation with high PAP. In cases of major PE, patients are at high risk of death due to right ventricular failure within the first hours of onset ($_{7}$). Survival

depends on rapid canalization of the pulmonary arterial occlusion and reduction of right ventricular afterload.

According to the results from a multicenter registry, overall in-hospital mortality rate ranges from 25% for patients presenting with cardiogenic shock to 65% for patients undergoing cardiopulmonary resuscitation ([8]). High resolution helical (spiral) computed tomographic angiography (CTA) has become a standard diagnostic tool for diagnosis of PE. In many patients, helical CT scans with intravenous contrast can resolve third-order pulmonary vessels without the need of invasive pulmonary catheters ([9], [10]). More recently electron beam tomography has been used for diagnosis of an interpulmonary saddle embolus ([11]). The aim of therapy of massive PE is to improve pulmonary circulation and hence right ventricle failure. There are different treatment modalities for massive PE like, thrombolysis, catheter fragmentation and/or emergent pulmonary embolecomy ([12]).

Successful thrombolysis was reported of submassive PE in bariatric surgery using i.v heparin followed by an infusion of i.v. alteplase 100mg over 2 hours ([13]). Thrombolytic therapy is a useful adjunct to heparin in patients who have PE and hemodynamically unstable ([14]). Thrombolytic therapy has been demonstrated to reduce clot burden in patients with PE more rapidly than heparin alone ([15]). There is no advantage of direct infusion of thrombolytic into the pulmonary artery versus intravenous infusion ([16]). The Food and Drug Administration has approved the use of urokinase (4400 IU/kg i.v. over 10 min, then 4400 IU/kg/hr i.v. for 12hr), strepokinase (250 000 IU i.v. over 30 min, then 100 000 IU/hr i.v. for 24hr), and tissue plasminogen activator (100 mg i.v. over 2hr) for the treatment of PE. The American Heart Association recommended the consideration of thrombolysis for major PE in the setting of syncope, hypotension, severe hypoxemia, or heart failure ([17]).

In the present case report, initially the patient's blood pressure was low, however, she responded immediately to resuscitation and inotropic drugs. Therefore, the question of whether to give thrombolytic therapy or not was discouraged. Major hemorrhage has been reported as a major risk of thrombolytic agents. The reported incidence of intracranial hemorrhage following thrombolytic therapy varies from 1.5-3% ([18]). Moreover, major bleeding has been reported in 22% of patients after thrombolysis in a large study ([19]). Therefore and due to lack of randomized controlled trial, the decision of using thrombolytic agents in PE remains controversial. Transvenous catheter embolectomy or open surgical embolectomy should be considered in patients for whom thrombolysis is contra-indicated or unsuccessful ([20], [21]).

CONCLUSIONS

In conclusion, massive PE is fatal and needs urgent treatment. We demonstrated a caseof near fatal PE diagnosed with helical CT scan and treated by anticoagulants with favorable outcome. Anticoagulants is the standard treatment for PE, in addition to thrombolysis or embolectomy.

References

1. Demers C, Ginsberg JS. Deep venous thrombosis and pulmonary embolism in pregnancy. Clin Chest Med 1992; 13:645-656.
2. Bergqvist D, Hender U. Pregnancy and venous thromboembolism. Acta Obstet Gynecol Scand 1983; 62:449-453.
3. Jeffries WS, Bochner F. Thromboembolism and its management in pregnancy. Med J Aust 1991; 155:253-258.
4. Rutherford SE, Phelan JP. Clinical management of thromboembolic disorders in pregnancy. Crit Care Clin 1991; 7:809-828.
5. Stein PD, Henry JW. Prevalence of acute pulmonary embolism among patients in a general hospital and autopsy. Chest 1995; 108:978-81.
6. Nass N, McConnell MV, Goldhaber SZ, Chyu S, et al. Recovery of regional right ventricular function after thrombolysis for pulmonary embolism. Am J Cardiol 1999; 83: 804-806.
7. Dalen JE, Albert JS. Natural history of pulmonary embolism. Prog Cardiovasc Dis 1975; 17:259-270.
8. Kasper W, Konstantinides S, Geibel A, et al. Management strategies and determinants of outcome in acute major pulmonary embolism: results of a multicentre registry. J Am Coll Cardiol 1997; 30:1165-1171.
9. Tapson VF, Witty L.A. Massive pulmonary embolism: diagnosis and therapeutic strategies. Clin Chest Med 1995; 16:329-40.
10. Grifioni S, Olivotto I, Cecchini P et al. Short-term clinical outcome of patients with acute pulmonary embolism, normal blood pressure and echocardiographic right ventricular dysfunction. Circulation 2000; 101:2817-22.
11. Enzweiler CN, Lambcke AE and Hamm B. Electron beam tomography of an interpulmonary saddle embolus. Heart 2004; 90(1): 36.
12. Cooper J.M and Beckman J.A. Massive pulmonary embolism: a remarkable case and review of treatment. Vascular Medicine 2002; 7: 181-185.
13. Pulipati RC, Lazzaro RS, Macura J et al. Successful thrombolysis of submassive pulmonary embolism after bariatric surgery: expanding the indications and addressing the controversies. Obes Surg 2003; 13(5):

792-6.
14. Goldhaber SZ. Thrombolysis for pulmonary embolism. N Engl J Med 2002; 347:
1131-1132.
15. Konstantinides S, Geibel A, Olschewski M et al. Association between thrombolytic
treatment and the prognosis of hemodynamically stable patients with major
pulmonary embolism: results of a multicentre registry. Circulation 1997; 96: 882-88.
16. Verstraete M, Miller GAH, Bouranmeaux H et al. Intravenous and intrapulmonary
recombinant tissue-type plasminogen activator in the treatment of acute massive
pulmonary embolism. Circulation 1988; 77: 353-60.
17. Hirsh J, Hoak J. Management of deep venous thrombosis and pulmonary embolism: a
statement for healthcare professionals from the council on thrombosis (in consultation
with the council on cardiovascular radiology), American Heart Association.
Circulation 1996; 93: 2212-45.
18. Goldhaber SZ. Thrombolysis in pulmonary embolism: a debatable indication.
Thromb Haemost 2001; 86: 444-51.
19. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary artery
embolism: clinical outcomes in the International Cooperative Pulmonary Embolism
Registry (ICOPER). Lancet 1999; 353: 1386-89.
20. SchmitzRode T, Janssens U, Duda SH et al. Massive Pulmonary embolism:
percutaneous emergency treatment by pigtail rotation catheter. J Am Coll Cardiol
2000; 36: 375-380.
21. Georghiou GP, Brauner R, Berman M et al. Successful resuscitation of a patient with
acute massive pulmonary embolism using emergency embolectomy. Ann Thorac
Surg 2004; 77(2):697-9.

Author Information

Abdelazeem El-Dawlatly, M.D.
Associate Professor, Department of Anesthesia, College of Medicine, King Saud University

Sulaiman AlMajid, MRCP
Professor of Pulmonology, Department of Medicine, College of Medicine, King Saud University

Medhat Elsherbeny, MBBCh, MsC
Registrar, Department of Anesthesia, King Khalid University Hospital

Wadha Alotaiby, KSUF, Arab Board, Saudi Board
Demonstrator & Senior Registrar, Department of Anesthesia, College of Medicine, King Saud University

Faiz A. Shakeel, MFFP, MRCOG
Consultant, Department of Obstetric & Gynecology, King Khalid University Hospital