Clinical Reports

Anaesthetic management of emergency Caesarean section followed by pulmonary embolectomy

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This report discusses the anaesthetic management of a patient undergoing emergency Caesarean section followed by an open pulmonary embolectomy with cardiopulmonary bypass. This case was complicated by the major physiological alterations associated with pregnancy and delivery, massive blood loss, cardiac dysrhythmias, hypotension, and cardiac arrest. Both patients, mother and child, made a full recovery.

Key words:
ANAESTHESIA: obstetrical; COMPLICATIONS: pulmonary embolism, cardiac arrest, EMBOLISM: pulmonary; HEART: arrest, arrhythmia, bradycardia, cardiac massage; PREGNANCY: Caesarean section; SURGERY: cardiovascular, cardiopulmonary bypass.

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Patients with a massive pulmonary embolus have numerous, severe physiological derangements. These deviations from normal, which include hypoxia, low cardiac output, acidosis, and pulmonary hypertension, often respond poorly to medical management and are not tolerated well by many patients. The patient in this report was stressed not only by a massive pulmonary embolus, but also by the physiological changes associated with pregnancy. Successful pulmonary embolectomy during pregnancy has been reported on only two occasions. Unfortunately, neither report considered the anaesthetic implications.[1,2] Two reports did discuss the anaesthetic management of patients with a pulmonary embolus, but neither discussed open pulmonary embolectomy.[3,4]

Two-thirds of the patients who die from a pulmonary embolus do so within 30 minutes of the acute event.[5] Patient management includes diagnostic tests (ventilation/perfusion ($\dot{V}/\dot{Q}$) scan and/or pulmonary angiogram), cardiovascular and respiratory support, anticoagulation and, very occasionally, surgical intervention (vena cava ligation, placement of a Greenfield filter, or pulmonary embolectomy).[6–9]

Case report

A 27-year-old, 70 kg woman, at 32 weeks gestation, presented to the emergency department (ER) because of a syncopal event at work. During the previous month she had received antibiotic treatment for a chronic cough and dyspnoea and in the preceding week she had developed mild haemoptysis. When examined in the ER she was cyanotic and awake but inattentive. She had a heart rate of 120 min^{-1}, a blood pressure (BP) of 102/70 mmHg, and a respiratory rate of 28 min^{-1}. Her initial arterial blood gas analysis (ABGs) on room air was pH 7.42, PaO_2 48 mmHg, $PaCO_2$ 32 mmHg and HCO_3^{-1} 21 $mmol \cdot L^{-1}$. Breathing 40 per cent O_2 improved her ABGs (PaO_2 70 mmHg). Other laboratory results were normal except for a

platelet count of 49×10^9 per litre. The fetal heart rate, which could not be heard on admission, was detected with an external doppler fetal heart monitor. A haematologist, respirologist and obstetrician were immediately consulted.

During the next four hours the patient's BP ranged from 90/70 to 60/40 mmHg, heart rate increased to 130 min^{-1} and her total urine output was only 25 ml, in spite of an infusion of 5 L Ringer's lactate. A $\dot{V}/\dot{Q}$ scan demonstrated areas of considerable $\dot{V}/\dot{Q}$ mismatch.

On arrival in the intensive care unit (ICU), a heparin infusion was started, her systolic BP had stabilized to 90 to 100 mmHg, and her PaO_2 was 60 mmHg while breathing 100 per cent O_2 by mask. Central venous access was established, but a pulmonary artery catheter was not inserted. The central venous pressure was 21 mmHg.

One hour after admission to the ICU, persistent fetal bradycardia necessitated emergency Caesarean section. Upon arrival in the operating room the patient was placed in the left lateral tilt position. Monitoring consisted of an ECG (Lead II), pulse oximeter, radial artery cannula and blood pressure cuff. After skin preparation, the patient's trachea was intubated whilst she was awake. The patient was administered 100 per cent O_2, and anaesthesia was induced with ketamine, 100 mg, and succinylcholine, 100 mg. Three minutes later atracurium, 30 mg, was administered. Approximately two minutes later a 1.86 kg infant was delivered and transferred to a neonatalogist (Apgar score was one at one and three minutes, and six at five minutes). The patient was given oxytocin 5 IU bolus, IV, and 10 IU in one litre of normal saline by continuous infusion. Uterine tone was adequate.

Several minutes after delivery the patient's O_2 saturation decreased to 90 per cent and her BP to 80 mmHg. Intravenous fluids, dopamine infusion, $4\,\mu g \cdot kg^{-1} \cdot min^{-1}$, and isoproterenol infusion, $0.1\,\mu g \cdot kg^{-1} \cdot min^{-1}$, were of no benefit. A 7Fr IV catheter was established in the left internal jugular vein. Pulmonary artery catheter placement was terminated after a brief episode of ventricular tachycardia. Two minutes later, the patient became very hypotensive, BP 40/20 mmHg, with a sinus bradycardia refractory to isoproterenol and atropine. Resuscitation was initiated with closed chest cardiac compressions and adrenaline, 1 mg IV. The patient's chest was opened by the cardiac surgeons. Cardiac massage was begun. Cardiopulmonary bypass (CPB) was promptly instituted. The pulmonary artery was opened and both old and new emboli were removed. The first haematocrit during the embolectomy was 0.1. During CPB the patient was given four units of packed red blood cells, ketamine, 100 mg, and sufentanil, 50 μg.

The patient was weaned from CPB with a left atrial pressure of 10 mmHg and inotropic support (isoproterenol, $0.3\,\mu g \cdot kg^{-1} \cdot min^{-1}$, adrenaline, $0.8\,\mu g \cdot kg^{-1} \cdot min^{-1}$, and dopamine, $5\,\mu g \cdot kg^{-1} \cdot min^{-1}$). After weaning from CPB, anticoagulation was fully reversed with protamine, and the patient was given 20 units of platelets and four more units of blood. Her systolic BP, 100 mmHg, was stable. She was given sufentanil, 25 μg, because of a gradual increase in HR to 145 min^{-1}. The subsequent marked hypotension responded to naloxone, 0.4 mg, IV. After removal of the drapes we discovered the patient had lost about 2 L of blood per vagina, in spite of adequate uterine tone.

The patient was transferred to the ICU. Both mother and child had uneventful recoveries and were discharged soon after.

Discussion

The pregnant patient is at increased risk of developing thromboembolic disease[10] because of venous stasis, vascular damage at delivery and a hypercoagulable state. The incidence of deep-venous thrombosis is 0.36 per cent antepartum and 3 per cent postpartum.[10] Pulmonary emboli occur in 1 in 2000 pregnancies.[10] Thromboembolic disease may account for up to 50 per cent of all obstetric-related morbidity and mortality.[11]

The initial management of the patient with massive pulmonary embolism may include oxygen, fluids, haemodynamic monitoring, ECG, chest x-ray, $\dot{V}/\dot{Q}$ scan, anticoagulation, thrombolytic agents, CPB and closed cardiac massage. Cardiopulmonary bypass has been used to stabilize and resuscitate patients. It is indicated when hypotension persists or right ventricular failure develops. The risk of dislodging emboli while establishing femoral CPB can be decreased by prior Fogarty® catheter embolectomy. Ideally, femoral-femoral CPB would have been established before induction of anaesthesia, but the cardiac team was not available until after the Caesarean section. Closed cardiac massage can be life saving because it may dislodge saddle emboli. Pulmonary angiography should be performed before embolectomy, if time and circumstances permit.[6]

Pulmonary embolectomy is controversial.[7,8,12,13] The reported mortality rate of pulmonary embolectomy ranges from 23 to 100 per cent.[14,15] Sasahara *et al.*[7] recommend embolectomy in patients with angiographically proved emboli and who have no response to medical therapy as shown by (1) signs of persisting peripheral vasomotor collapse, (2) systolic BP less than 90 mmHg, (3) urine output less than 20 $ml \cdot hr^{-1}$ and (4) PaO_2 less than 60 mmHg. Many recommend embolectomy only after complete cardiovascular collapse,[8] because most patients will either die immediately or improve due to clot lysis.

Intraoperative monitoring should include intra-arterial

catheter, PA catheter, ECG, pulse oximeter and capnograph in addition to fetal heart rate monitor. This monitoring guides the management of right ventricular dysfunction, intraoperative pulmonary oedema, postembolectomy pulmonary haemorrhage and hypotension, increased pulmonary vascular resistance (PVR), low cardiac output, blood loss and fluid shifts. The high risk of massive blood loss and fluid shifts necessitates the insertion of at least two large-bore peripheral IV lines. Fluid shifts are associated with IVC ligation and lung reperfusion. Our patient unfortunately did not tolerate PA catheter insertion.

Cardiac arrest on induction of anaesthesia is common.[14] Typically, these patients are acidotic and hypoxic; they have markedly elevated PVR, right ventricular dysfunction, a fixed cardiac output so that decreases in systemic vascular resistance are poorly tolerated, and their cardiovascular function is supported by massive release of endogenous catecholamines. Ketamine was preferred because it is a proven obstetrical anaesthetic, it increases sympathetic tone and it is tolerated well by cyanotic patients. However, ketamine may increase PVR and PA pressure and depress the myocardium. Although narcotics provide stability in patients with a compromised myocardium, pure narcotic induction of anaesthesia in patients with a compromised cardiovascular system, e.g. hypovolaemia, is not without risk. The muscle relaxant used was atracurium because at the time it was the agent available to us that had the least haemodynamic effect. The anaesthetic induction agent and muscle relaxant of choice in this situation is unknown.

The proper use of vasoactive agents in patients with pulmonary embolism is unknown. Isoproterenol did not help this patient. In an animal model of pulmonary emboli all isoproterenol-treated dogs died.[16,17] Noradrenaline improved dog survival and increased mean arterial pressure, cardiac output, stroke volume, and right ventricular performance.[16,18] Dobutamine-treated patients with massive pulmonary embolism have an increased cardiac index and mixed venous oxygen content and a decreased PVR,[19] whereas dopamine treatment increased cardiac output and PA pressure.[20] Based on the limited current literature, either noradrenaline or dobutamine would be the inotropic agent of choice but this ignores the concerns of the utero-placental circulation. Vasopressors, such as noradrenaline, adrenaline, dopamine and dobutamine decrease utero-placental blood flow.[21,22] The inotropic agent of choice during pregnancy would seem to be dobutamine because it has the least amount of alpha-adrenergic activity.[22] Pulmonary vasodilators are unlikely to be helpful because they inhibit hypoxic vasoconstriction and cause systemic hypotension.

Further increases in PVR should be minimised by treating and avoiding acidosis, hypercarbia and hypoxaemia and avoiding agents that increase PVR.

Numerous life-threatening complications may occur after embolectomy. These include right or left ventricular failure, high-pressure pulmonary oedema, pneumothorax, pulmonary infection, phrenic nerve injury, haemothorax and pulmonary infarction, and massive haemorrhagic pulmonary oedema secondary to reperfusion of the ischaemic and infarcted lung.[14,23–25] For example, 1500 ml of blood has been suctioned from the bronchial tree within 30 minutes of pulmonary recirculation.[23] Our major complication was blood loss. In spite of adequate uterine tone, the patient lost 2 L of blood per vagina, presumably secondary to her coagulapathy.

The anaesthetic management of a patient undergoing an emergency Caesarean section followed by pulmonary embolectomy has been presented and critically discussed. This case was complicated by pregnancy, massive blood loss, hypotension, cardiac dysrhythmias, and cardiac arrest. The optimal management of this patient is not known. Our patient tolerated ketamine, succinylcholine, and atracurium, while sufentanil and isoproterenol were not tolerated. The administration of anaesthetic agents to this patient would have been simplified if a PA catheter and CPB had been available earlier. Animal studies are required (1) to assess the effects of anaesthetic agents in such circumstances and (2) to determine the best vasoactive agent to administer to a patient with a massive pulmonary embolism. Additional reports of the anaesthetic management of these cases need to be reported.

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Résumé

Ce papier discute de la conduite anesthésique chez une patiente subissant une césarienne d'urgence suivie d'embolectomie pulmonaire et circulation extra corporelle. Ce cas fut compliqué par des altérations physiologiques majeures associées à la grossesse, l'accouchement, la perte massive de sang, arythmie cardiaque, hypotension et arrêt cardiaque. La mère et l'infant ont eu une recouvrance totale satisfaisante.