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CASE REPORT

Survival following amniotic fluid embolism and cardiac arrest complicated by sub-capsular liver haematoma

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SUMMARY. We describe the anaesthetic and intensive care management of a 38-year-old mother with presumed amniotic fluid embolism who suffered cardiorespiratory collapse following delivery of a normal baby by caesarean section. After initial resuscitation, her recovery was complicated by development of disseminated intravascular coagulation and a large sub-capsular hepatic haematoma. We describe the initial resuscitative efforts and subsequent intensive therapy to full neurological recovery and discharge from hospital.

Keywords: Amniotic fluid embolism; Cardiac arrest; Hepatic haematoma; Disseminated intravascular coagulation

INTRODUCTION

Amniotic fluid embolism is an uncommon but usually fatal condition accounting for around 10% of all maternal deaths. It is classically characterised by hypoxia, hypotension, haemodynamic collapse and coagulopathy.

CASE REPORT

A previously healthy 38-year-old female (gravida 3, para 2) presented to the obstetric unit for elective induction of labour at 40 weeks' gestation. Her previous obstetric history included two forceps deliveries for large babies. After labour was established using 1 mg of Dinoprostone E_2 vaginal gel (`Pharmacia & Upjohn) an 18-gauge epidural catheter was sited at the L3/4 interspace in the sitting position, using continuous loss of resistance to saline and a 16-gauge Tuohy needle. A 60-mg test dose of lidocaine was followed by a 10-mL bolus of 0.25% bupivacaine. An infusion of 0.1% bupivacaine containing fentanyl 2.5 μg/mL was given at 10 mL/h. The patient reported effective analgesia and intravenous oxytocin was given to hasten the progress of labour.

After 18 h, labour had failed to progress sufficiently (cervical dilatation 6 cm) and the decision was made to proceed to caesarean section. Ranitidine 50 mg and metoclopramide 10 mg were administered i.v. and the epidural was topped up with 0.5% bupivacaine 20 mL by slow bolus injection. After 15 min the level of sensory block to pin prick was estimated at T6 bilaterally. Sodium citrate (30 mL 0.3 M) was administered orally and the patient was transferred to the operating theatre with a 15° left lateral tilt. She was monitored with pulse oximetry, electrocardiography (ECG) and non-invasive blood pressure (NIBP). The level of block was reassessed (still at T6) but surgery was allowed to begin.

A female baby weighing 3.9 kg was delivered 10 min after the start of surgery, with an Apgar score of 10 at 5 min. After delivery, Syntocinon 10 units followed by cefuroxime 750 mg and metronidazole 500 mg were given i.v. and preservative-free morphine 3 mg was given via the epidural catheter.

During closure of the rectus sheath (approximately 20 min after delivery) there was a sudden drop in arterial pressure to 74/0 mmHg, associated with a heart rate of 70 beats/min and normal QRS complexes on the ECG. The patient appeared well and maintained a normal conscious level. The hypotension was treated with

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intravenous ephedrine but was unresponsive to a total dose of 30 mg. Meanwhile the patient's conscious level deteriorated. Three minutes after the initial hypotensive event, she completely lost consciousness and became pulseless. Cardiac arrest with pulseless electrical activity was diagnosed, the ECG still showing normal QRS complexes and a heart rate of 51 beats/min. Cardiopulmonary resuscitation (CPR) was commenced following European Resuscitation Council guidelines.[1] External cardiac massage was performed at a rate of 100/min, she was intubated and the lungs were ventilated with 100% oxygen. Five cycles of CPR were administered with adrenaline 1 mg given i.v. with each cycle (total 5 mg). Clinical examination ruled out tension pneumothorax and although there was no obvious major haemorrhage, 1500 mL of gelatine solution (Gelofusine) was given to treat possible hypovolaemia. Cardiac output returned approximately 8 min after cardiac arrest was diagnosed and the first recordable blood pressure reading was 78/44 mmHg. The patient began to cough and spontaneous respiration returned. She was sedated with midazolam 4 mg, neuromuscular blockade was achieved with atracurium 50 mg and the lungs were ventilated with 100% oxygen.

It was then noted that there was profuse haemorrhage from the open abdominal wound and per vaginam. The wound was closed surgically and pressure dressings were applied. Further Gelofusine 1000 mL and cross-matched blood (4 units) were given. A 20-gauge radial arterial and a right internal jugular triple-lumen cannula were inserted. Amniotic fluid embolism was suggested as a diagnosis and peripheral maternal blood was taken for fetal squamous cells. The patient was transferred to the intensive care unit (ICU) for further management.

On admission to the ICU she continued to bleed heavily from the abdominal wound site and per vaginam. She remained hypotensive, and along with continued fluid and blood resuscitation, she was given an infusion of epinephrine (up to 0.25 $\mu g \cdot kg^{-1} min^{-1}$). A 9 French gauge percutaneous sheath introducer (Arrow International Inc, Reading, USA) was inserted into the left internal jugular vein and a pulmonary artery catheter was floated to optimise fluid therapy and guide inotropic support. Her initial haemodynamic readings showed a high cardiac output, low systemic vascular resistance and low pulmonary artery occlusion pressure. Fetal epithelial squamous cells were demonstrated in maternal peripheral blood and haematological studies showed anaemia and disseminated intravascular coagulation (DIC) with a haemoglobin concentration of 6.7 g/dL, fibrin degradation products 16 mg/L, activated partial thromboplastin time 45.4 s, prothrombin time 14 s and platelet count 48×10^9/L. The coagulopathy was treated with a total of 12 units of fresh frozen plasma, 8 units of cryoprecipitate, 4 units of platelets, 10 units of packed red cells, calcium chloride 20 mmol and tranexamic acid 1 g. This treatment started in the operating theatre and continued in ICU.

Within 1 h of admission to ICU, the vaginal bleeding had subsided. She became more haemodynamically stable and inotropic support was reduced. An echocardiogram showed right ventricular dilatation and tricuspid regurgitation consistent with an embolic phenomenon. Although her blood pressure was stable, over the next 2-3 h her abdominal girth increased and haemoglobin concentration continued to fall, despite a further 3-unit transfusion of packed red cells. She underwent a further laparotomy (via the existing Pfannenstiel incision) 15 h after delivery which revealed approximately 2000 mL of clotted blood in the abdominal cavity and generalised venous ooze but no major bleeding points. She returned to the ICU where she was sedated with propofol (1.5-2 $mg \cdot kg^{-1} h^{-1}$) and alfentanil (0.1-0.2 $\mu g \cdot kg^{-1} min^{-1}$) and neuromuscular blockade was continued with atracurium (1 $mg \cdot kg^{-1} h^{-1}$) as intermittent positive pressure ventilation was difficult due to abdominal distension. She was placed on pressure controlled ventilation (inspired pressure 20 cmH_2O, I/E ratio 1:2, PEEP 7.5 cm H_2O and FiO_2 0.5). She remained on an epinephrine infusion (up to 0.2 $\mu g \cdot kg^{-1} min^{-1}$) and was given dopexamine (0.5 $\mu g \cdot kg^{-1} min^{-1}$) to improve renal and splanchnic perfusion. Arterial blood gas analysis showed PaO_2 of 13.0 kPa and overnight the FiO_2 was reduced to 0.4.

The following day her abdominal girth was further increased and an ultrasound scan showed a large subcapsular hepatic haematoma with bleeding into the hepatic parenchyma. She had continued to require blood and colloid transfusions despite complete correction of coagulation tests to normal values. Her abdominal girth continued to increase and she again became difficult to ventilate. The regional liver unit was contacted and advised re-laparotomy and insertion of a large sump drain into the right paracolic gutter to monitor any further blood loss. This was performed uneventfully and after returning to the ICU she remained haemodynamically stable and inotropic support was reduced. She continued to lose more than 100 mL/h via the sump drain, however, and it was decided to transfer her to the regional liver unit. Transfer was uneventful.

On the liver unit the bleeding ceased spontaneously without need for further surgery. She was weaned from mechanical ventilation over the next 24 h. Her neurological status improved over the subsequent 7 days until she was assessed as completely normal. Unfortunately the hepatic haematoma became infected and she underwent further laparotomy for its removal 17 days after the delivery. Following this procedure she was ventilated on the ICU overnight and rapidly weaned from mechanical ventilation the next day. She made a full recovery and was discharged from hospital 6 weeks after the