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CASE REPORTS

Type A aortic dissection in pregnancy

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ABSTRACT
Type A aortic dissection is a life-threatening event to both mother and baby, and accounted for 14% of maternal cardiac deaths in the 2006–2008 UK Confidential Enquiries into Maternal Deaths. Difficulty exists in the diagnosis of this rare but potentially curable condition, the mortality of which increases with delay in diagnosis. We present a case of acute type A aortic dissection in a previously well multiparous woman, treated successfully by aortic root repair immediately following caesarean section. The acute presentation of aortic dissection and diagnostic clues that may have expedited the diagnosis are discussed. A brief literature review is presented of the perioperative management of patients undergoing cardiothoracic surgery post-caesarean section and the modifications to standard techniques that are required.

Keywords: Aortic dissection; Cardiopulmonary bypass; Aortic root replacement; Pregnancy; Caesarean section

Introduction

Acute type A aortic dissection (AAD) in pregnancy is a life-threatening event to both mother and baby, and accounts for 14% of maternal cardiac deaths.[1] It is an important consideration for those managing chest pain on the labour ward, as the mortality from untreated proximal aortic dissections increases by 1–3% per hour after presentation and is approximately 25% during the first 24 h, 70% at 1 week and 80% at 2 weeks.[2,3] A total of 50% of dissections in females < 40 years of age occur during pregnancy, typically in the third trimester or early postpartum period.[4] A review of the literature of the last 10 years revealed only case reports[5–15] and limited retrospective case series[16,17] of antenatal aortic dissection in patients without risk factors. We report a case of type A aortic dissection successfully managed with aortic root replacement following caesarean section.

Case report

A 34-year-old woman with a previously uneventful pregnancy presented to the emergency department at 39 weeks of gestation with chest pain and vomiting. Her past medical history included an elective caesarean section (CS) under spinal anaesthesia for breech presentation, followed by a primary postpartum haemorrhage requiring blood transfusion.

She experienced sharp chest, upper abdominal and back pain, worse on deep inspiration and requiring opioid analgesia. She had vomited at least six times, of which one episode was described as resembling dark coffee grounds. On the day of admission, her partner had a gastrointestinal upset following a meal.

Blood pressure and heart rate had been unremarkable at all antenatal checks and on admission. Clinical examination was normal and she was noted to have equal radial pulses. Admission electrocardiogram and chest X-ray (CXR) were reported as normal (Fig. 1). A presumptive diagnosis of gastroenteritis was made. She was kept nil-by-mouth and treated with intravenous fluids and an H2 receptor antagonist. Fetal heart rate was monitored continuously. The cardiotocograph (CTG) was reassuring, with no signs of onset of labour. Hourly observations were recorded on a maternal early warning score (MEWS) chart.

Although observations were initially stable, the patient continued to experience pain and vomiting. After 3 h she became drowsy, and was found to be tachycardic, hypertensive and hypoxic. The CTG showed a fetal tachycardia. Maternal arterial blood gas analysis showed metabolic acidosis and poor oxygenation (pH 7.28, PO_2 11.9 kPa, PCO_2 4.2 kPa, base deficit 10.7 mmol/L, lactate 3.0 mmol/L). An urgent CXR showed diffuse bilateral shadowing in the lower and mid zones, and was reported as suggestive of aspiration of gastric contents (Fig. 2). With a provisional diagnosis

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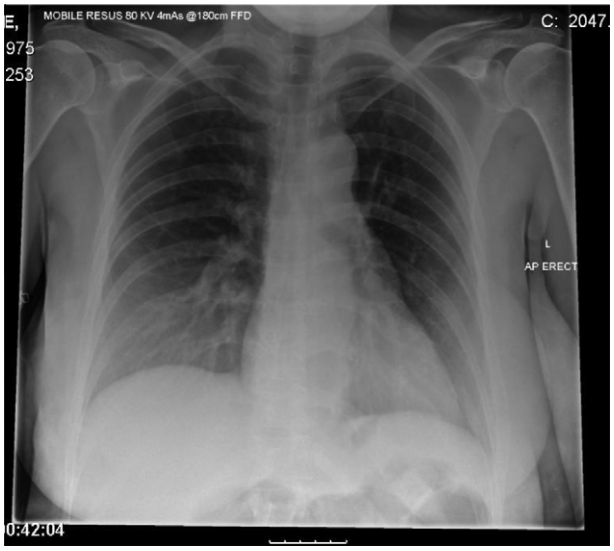

Fig. 1 Chest X-ray on admission

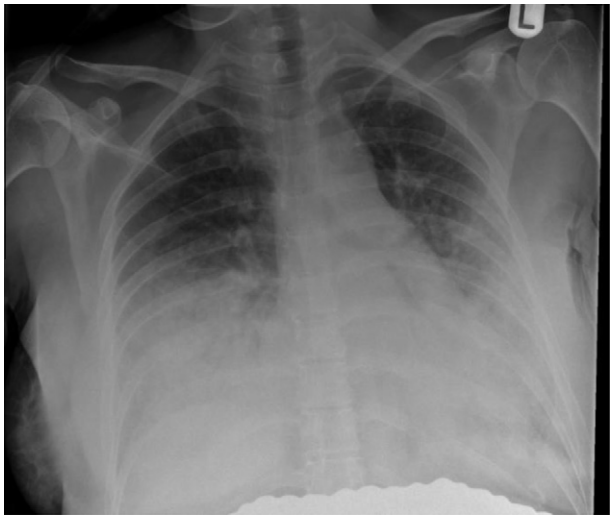

Fig. 2 Chest X-ray before caesarean section

of severe sepsis and non-reassuring CTG, a category-1 CS was made.

Following transfer to the operating theatre, general anaesthesia was induced with thiopental 400 mg and suxamethonium 100 mg. Profuse secretions were encountered on laryngoscopy, and 200 mL of initially brown and later frothy pink fluid was suctioned from the oropharynx and tracheal tube. Hypoxia after intubation required 100% oxygen, high positive end-expiratory pressure and alteration of ventilation ratios to achieve adequate oxygenation. Blood pressure was stable throughout, and the heart rate normalized with intravenous boluses of colloid.

A live female infant was delivered in a flaccid condition. Apgar scores were 4 and 8 at 1 and 5 min, respectively, with acidaemic cord gases (umbilical artery pH 6.96, base deficit 13.6 mEq/L, umbilical vein pH 6.98, base deficit 13.6) and she was transferred to the neonatal unit with a presumed diagnosis of neonatal sepsis, secondary to maternal sepsis.

Maternal central venous and arterial cannulae were inserted. Invasive blood pressure measurements in the right arm were lower than those obtained using a non-invasive cuff on the left arm, which was confirmed by invasive arterial access on the left side. Estimated total blood loss was 1200 mL, attributable to slow contraction of the uterus. An oxytocin bolus of 5 IU was followed by an infusion of 10 IU/h. Maternal blood gas analysis showed severe metabolic acidosis with a lactate of 10.7 mmol/L. She was transferred sedated, intubated and ventilated to the intensive care unit (ICU) for further management.

A bedside transthoracic echocardiogram (TTE) showed aortic root dilatation, moderate to severe aortic valve regurgitation, an intimal dissection flap in the ascending aorta and severely impaired left ventricular function. This was confirmed with transoesophageal echocardiogram (TOE) (Fig. 3). Emergency aortic root replacement was performed, commencing 4 h after her caesarean section. Operative time was 7.5 h, with 2.5 h on cardiopulmonary bypass (CPB) and hypothermia (24°C), but no cardiac arrest. In view of her increased bleeding risk, the oxytocin infusion continued and operative bleeding was limited. She received 6 units of packed red cells, 7 units of fresh frozen plasma, 2 units of platelets and 2 units of cryoprecipitate. Tranexamic acid was given to reduce CPB-associated fibrinolysis. Postoperative international normalized ratio and activated prothrombin time were within normal limits. She returned to the ICU receiving infusions of noradrenaline, adrenaline and milrinone but remained acidaemic (pH 7.1, lactate 24 mmol/L) and received continuous veno-venous haemofiltration. She was quickly weaned from inotropic support. Her CXR continued to show bilateral consolidation consistent with pulmonary oedema or acute respiratory distress syndrome, which de-

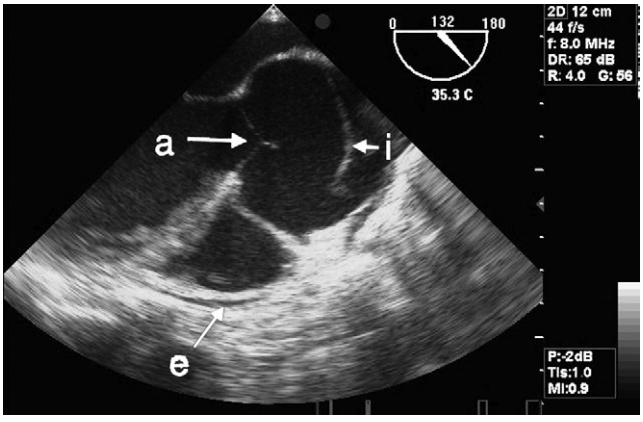

Fig. 3 Transoesophageal echocardiogram preformed before transfer to cardiac theatre: a, aortic valve; e, small pericardial effusion; i, flap of dissected intima in aorta.