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Cardiac arrest due to uterine inversion during caesarean section

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ABSTRACT
We describe the case of a 20-year-old G3P0 woman who was delivered by caesarean section under general anaesthesia, complicated by uterine inversion secondary to undiagnosed placenta accreta and cardiac arrest requiring cardiopulmonary resuscitation. Uterine inversion is a known complication of placenta accreta and is a rare occurrence at caesarean section. Similar cases have been reported, though cardiac arrest is an uncommon feature. The possible causes and management are discussed.

Keywords: Caesarean section; Placenta accreta; Uterine inversion; Cardiac arrest in pregnancy

Introduction

Cardiac arrest during caesarean section is extremely rare. There are multiple causes including haemorrhage, anaphylaxis, embolic events, hypertensive disease of pregnancy, sepsis, high spinal blockade and cardiac disease. Placenta accreta occurs when there is abnormally invasive implantation of the placenta due to a defect of the decidua basalis and increases the chance of uterine inversion during delivery. The presence of placenta accreta represents a significant threat to the mother and poses challenges for the obstetric anaesthetist and surgeon in providing safe delivery of the baby. We describe a case of cardiac arrest in a parturient with undiagnosed placenta accreta and discuss the possible causes and management of the cardiac arrest.

Case report

A healthy 20-year-old woman (G3P0) with no significant past medical history except for two suction terminations of pregnancy under general anaesthesia, had an uneventful pregnancy until 39 weeks of gestation. She had a routine 20-week ultrasound scan, which showed a normal fetus and a posterior placenta. She weighed 65 kg and was 162 cm in height (body mass index 24.8 kg/m^2). At 39 weeks she was found to be hypertensive with a blood pressure of 150/100 mmHg and proteinuria 7.6 g/L. Preeclampsia was diagnosed, and the patient was admitted to hospital for blood pressure control with oral labetolol. Her haemoglobin concentration on admission was 9.9 g/dL and platelets 238×10^9/L. After 24 h in hospital, labour was induced with two vaginal prostaglandin PGE_2 pessaries. When the cervix was 3 cm dilated the membranes were ruptured and an oxytocin infusion started. An epidural catheter was sited at L3-4 without complication; a 3-mL test dose of 0.5% plain bupivacaine was followed by an 8-mL loading dose of 0.25% bupivacaine 5 min later. An epidural infusion of 0.1% bupivacaine with fentanyl 2 μg/mL was started at 10 mL/h. Assessment revealed a patchy epidural block and the midwifery staff requested review of the pain relief. The patient required an extension of her epidural, but this coincided with an obstetric decision that the she needed a caesarean section for failure to progress in the second stage of labour (category 3).

The epidural was extended with a mixture of 2% lidocaine 10 mL, 0.5% bupivacaine 10 mL and fentanyl

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50 μg given in 5-mL aliquots. Antacid prophylaxis with i.v. ranitidine 150 mg metoclopramide 10 mg and oral 0.3 M sodium citrate 30 mL were given. Maternal heart rate, non-invasive blood pressure and oxygen saturation were monitored. When the sensory level extended to T6 to touch and T4 to cold bilaterally surgery started. On opening the peritoneum, however, the patient complained of discomfort and the decision made to convert to general anaesthesia using rapid sequence induction. Anaesthesia was induced with thiopental 375 mg and suxamethonium 100 mg and the trachea intubated, maintenance was continued with isoflurane in an oxygen/nitrous oxide mix. After induction a fall in blood pressure to 90/60 mmHg was treated with a 500-mL i.v. bolus of lactated Ringer's solution and a 6-mg bolus of ephedrine. The caesarean section proceeded with extra monitoring including end-tidal carbon dioxide, inspired oxygen and volatile anaesthetic concentration. A live male infant was delivered with Apgar scores of 8 and 10 at 5 and 10 min, respectively; a 5-IU i.v. bolus of oxytocin was given over 10 s followed by an infusion of 40 IU in 500 mL 0.9% saline over 4 h. Following the second cord traction there was complete uterine inversion through the lower segment incision. This complication was immediately reported to the anaesthetist and both consultant anaesthetist and consultant obstetrician were summoned to theatre. At this time the placenta remained adherent to the uterine wall. The oxytocin was stopped and attempts to reduce the uterus manually were unsuccessful. At this point there was no obvious bleeding and blood pressure, heart rate and oxygen saturations were within normal limits, but one litre of intravenous gelatin (Gelofusine) was administered in view of the considerable risk of haemorrhage.

Nine minutes after the uterine inversion, the patient became hypotensive and tachycardic. The lowest recorded blood pressure was 55/30 mmHg with a heart rate of 125 beats/min; oxygen saturations dropped to 80%. Tracheal tube placement was checked by manual ventilation and auscultation confirming that there was good air entry bilaterally. Capnography showed a reduction in end-tidal carbon dioxide to less than 1 kPa, consistent with a decreased cardiac output. Bronchospasm, angio-oedema or rash were not observed. Non-invasive blood pressure became unrecordable but bradycardia did not develop. Both carotid and femoral pulses were impalpable. Pulseless electrical activity (PEA) was diagnosed and cardiopulmonary resuscitation (CPR) instituted. The patient's lungs were ventilated with 100% oxygen, external cardiac massage was started and i.v. epinephrine 1 mg given. After one 3-min cycle of CPR, pulses became palpable and capnography displayed exhaled carbon dioxide. Forcible removal of the adherent placenta then followed from which there was considerable bleeding, blood loss was difficult to estimate but was considered to be in the region of 600 mL. The fundus was replaced through the uterotomy; chemical uterine relaxation was not required. Arterial and central venous pressure lines were then inserted. At this time the patients' blood pressure was 130/80 mmHg, heart rate was 85 beats/min, central venous pressure was 4–5 mmHg and oxygen saturations were 99%. An arterial blood sample revealed: pH 7.29, PCO_2 4.5 kPa, PO_2 21.5 kPa, bicarbonate 15.7 mmol/L and a base excess of −9.6 mEq/L. There was some continued uterine bleeding due to uterine atony, which was treated with carboprost 250 μg i.m. and 250 μg intra-myometrially plus a further oxytocin infusion of 40 IU in 500 mL of 0.9% saline over 4 h. A 1.2-g i.v. dose of co-amoxiclav was given during closure of the abdominal wall. The bleeding was controlled and surgery completed without the need for hysterectomy.

In view of the stable cardiovascular parameters and the control of haemorrhage, the decision was made to extubate the patient on completion of surgery. The patient was observed on the intensive care unit overnight and received a 4-unit blood transfusion due to a postoperative haemoglobin concentration of 6.4 g/dL. She was discharged to the postnatal ward the following day, and mother and baby were discharged home on the fourth postoperative day in good health.

Discussion

Placenta accreta is a rare obstetric complication with an incidence of approximately one in 2500 births and associated with a 7% mortality rate.[1,2] In 2002 the American College of Obstetrics and Gynecology recommended that if there is a high index of suspicion for placenta accreta the patient should be counselled about the possible need for hysterectomy and blood transfusion; blood products and clotting factors should be available; cell saver technology should be considered; appropriate timing and location for delivery should be considered to allow access to adequate surgical personnel and equipment and a preoperative anaesthesia assessment should be made.[3] In a case series of 28 patients with an increased risk of placenta accreta, there were no cases of uterine inversion or cardiac arrest when delivery was via planned caesarean section.[4]

The incidence of acute uterine inversion has been stated to be between one in 2148 and one in 6407 vaginal births.[5,6] Most cases are associated with vaginal delivery. Acute uterine inversion at caesarean section is extremely rare; fewer than 10 cases have been reported in the literature, although many may go unreported.[7–14] Of these case reports only one was complicated by cardiac arrest.[13]

Where uterine inversion has occurred per vaginam the acute management has two objectives. The first is to replace uterus and the second is to treat the associ-