Precordial Doppler diagnosis of haemodynamically compromising air embolism during Caesarean section

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This is a report of a 39-year-old parturient who had a haemodynamically compromising venous air embolism during a repeat Caesarean section under lumbar epidural anaesthesia. The embolism occurred immediately after surgical incision during surgery in the superficial subcutaneous tissues. The diagnosis was made using intraoperative precordial ultrasonic Doppler monitoring which allowed early and successful treatment.

On rapporte l'histoire de cas d'une parturiente de 39 ans ayant eu une embolie gazeuse veineuse ayant des répercussions hémodynamiques lors d'une césarienne sous anesthésie épidurale lombaire. L'embolie est survenue immédiatement après l'incision chirurgicale. Le diagnostic fut fait lors de l'opération par une sonde précordiale à ultrason qui a permis un traitement qui s'est couronné de succès.

Key words
ANAESTHESIA: obstetric;
ANAESTHETIC TECHNIQUES: epidural;
COMPLICATIONS: embolism;
EMBOLISM: air;
SURGERY: Caesarean section.

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Venous air embolism is a potential hazard during any surgical procedure in which the operative site is above the level of the heart.[1] Air embolism during pregnancy, Caesarean section, and vaginal delivery has been detected by various methods. However, the incidence of significant parturient morbidity and mortality from air embolism is rare. We found no other reports in the Caesarean section literature of a haemodynamically compromising venous air embolism occurring immediately after skin incision nor were there any reports of the detection of such an event by precordial ultrasonic Doppler monitoring.

Case report

A 39-year-old patient presented for repeat low flap Caesarean section for a transverse lying fetus. Her pregnancy was remarkable for chronic hypertension and the onset of primary hyperparathyroidism which required no treatment. Her medical history was positive for scoliosis, obesity (height 160 cm, weight 105 kg), a history of renal stones and a previous uncomplicated Caesarean section for failure to progress. Otherwise, her past medical history, physical examination and laboratory values were unremarkable. The patient's vital signs were: blood pressure (BP) 135/85 mmHg, heart rate (HR) 72 beats $\cdot$ min^{-1} and respiratory rate (RR) 16 breaths $\cdot$ min^{-1}. After prehydration with two litres of Ringer's lactate, lumbar epidural anaesthesia was performed without complications using 0.5 per cent bupivicaine. The final level of anaesthesia was the fourth thoracic dermatome.

The patient was placed on a level operating room table in the supine position with left uterine displacement. The intraoperative monitors included an automatic blood pressure monitor (Physio-Control VSM2), electrocardiogram, precordial stethoscope, pulse oximeter, precordial ultrasonic Doppler and a skin temperature monitor. Her vital signs were: BP 124/80 mmHg, HR 82 beats $\cdot$ min^{-1} and RR 16 breaths $\cdot$ min^{-1}. By pulse oximetry, her haemoglobin oxygen saturation was 100 per cent.

Immediately after surgical incision was made, during surgery in the superficial subcutaneous tissues, there was

a characteristic change in the precordial ultrasonic Doppler signal consistent with venous air embolism. Simultaneously, the patient complained of chest pain, shortness of breath and nausea; she was diaphoretic and tachypnoeic with a respiratory rate of 30–35 breaths · min^{-1}, peripheral cyanosis, and a decrease in haemoglobin oxygen saturation from 100 to 74 per cent. She had two 15 sec runs of ventricular tachycardia with a decrease in blood pressure from 140/80 to 95/60 mmHg. The patient was immediately given 100 per cent oxygen (O_2) by face mask and 100 mg lidocaine IV according to standard Advanced Cardiac Life Support protocol. The surgical field was flooded with saline. The patient was placed in the Trendelenburg position with increased left lateral tilt. The Caesarean section was continued with the surgical field flooded with saline. Within the five minutes, the patient was still awake, alert and now mildly dyspnoeic. Her oxygen saturation improved from 74 to 94 per cent; electrocardiogram showed sinus tachycardia at 110 beats · min^{-1}. Her blood pressure was 95/60 mmHg, and RR was 25 breaths · min^{-1}. Because of the continuing hypotension, a total of 15 mg IV ephedrine sulfate was given with a resultant increase in BP to 120/65 mmHg. A male infant weighing 3330 g was delivered. Apgar scores at one and five minutes were nine and nine, respectively. A right peripheral central venous catheter and a left radial artery catheter were placed. Aspiration of air from the central venous catheter was attempted without success, and the central venous pressure was 10 mmHg. Arterial blood gas analysis on a fraction of inspired oxygen of 1.0 via a non-rebreathing face mask revealed: pH 7.29, PCO_2 37 mmHg, PO_2 74 mmHg, HCO_3^- 19 mmol · L^{-1} and a haemoglobin oxygen saturation of 93.1 per cent. Her serum sodium and potassium concentrations were normal. There were no persistent electrocardiographic changes in leads II or V_5.

Within 15 min of this episode, the patient was no longer symptomatic. Arterial blood gas analysis breathing 100 per cent O_2 showed a pH 7.31, PCO_2 32 mmHg, PO_2 178 mmHg, HCO_3^- 19 mmol · L^{-1} and haemoglobin oxygen saturation of 99.4 per cent which correlated with pulse oximetry. For the remainder of the Caesarean section, the patient was in the reverse Trendelenburg position with vital signs within five per cent of: BP 120/75 mmHg, HR 85 beats · min^{-1} and RR 15 breaths · min^{-1}. There were no further changes consistent with air emboli on Doppler monitoring.

Postoperatively, her chext *x*-ray was remarkable for severe dextroscoliosis, pulmonary congestion, and a possible right upper lobe infiltrate or atelectatic area. On the third postoperative day, the asymptomatic patient was noted to have bibasilar rales with mild expiratory wheezing on physical examination. Repeat chest *x*-ray was unchanged. The patient continued to receive chest physiotherapy and was begun on an Alupent (metaproterenol) inhaler with resolution of her respiratory rales and wheezes by the fourth postoperative day. At this time, because of right calf tenderness, the patient had a ventilation/perfusion scan and a right leg venogram. These were interpreted as low probability for pulmonary embolus and negative for deep venous thrombosis. On the seventh postoperative day, the patient was discharged home, fully ambulatory and able to care for her baby.

Discussion

In 1947, Durant *et al.* reported that the venous sinuses of the uterus in obstetrical patients were theoretically susceptible to the entrance of air especially in the presence of placenta praevia and/or manual extraction of the placenta.[1] Using the precordial ultrasonic Doppler, Malinow *et al.* reported a 52 per cent incidence of venous embolism without haemodynamic alterations in patients undergoing Caesarean section with spinal or lumbar epidural anaesthesia, with 52 per cent of these patients experiencing chest pain and 20 per cent experiencing dyspnoea. Of these presumptive air emboli, 74 per cent occurred with uterine incision, two per cent with the delivery of the baby, 13 per cent with placenta removal and 11 per cent with uterine repair.[2] Despite the high incidence of venous air embolism during Caesarean section, haemodynamically compromising air embolism during Caesarean section is rare. Because of this low incidence, there are varying opinions about which patients undergoing Caesarean section should be monitored with precordial ultrasonic Doppler.[3–5] In our practice, we decided to monitor Caesarean section patients for venous embolism to see if our incidence concurred with the observations of Malinow *et al.*

This case report illustrates the usefulness of precordial ultrasonic Doppler monitoring for early detection and thus successful treatment of a haemodynamically compromising venous air embolism during Caesarean section under lumbar epidural anaesthesia in a normovolaemic patient without known risk factors for air embolism. Furthermore, it shows that air embolism can occur at any time during the surgical procedure (i.e., when operating in the subcutaneous tissue), not just at the times mentioned by Malinow *et al.*[2] Although obesity may have contributed to this problem, it is not a proven risk factor for venous air embolism. Venous air embolism was the cause of this intraoperative event; pulmonary thromboembolism and amniotic fluid embolism were ruled out by the patient's rapid improvement with the inspiration of 100 per cent O_2, a negative ventilation/perfusion scan, a negative venogram, and the lack of adverse postoperative sequelae.

If a haemodynamically compromising air embolism

occurs during Caesarean section, the treatment consists of flooding the surgical field with saline *before* placing the patient in Trendelenburg position with extreme left lateral tilt. Then, 100 per cent O_2 should be administered, a central venous catheter inserted to aspirate air and to infuse any necessary resuscitative drugs, and additional cardiorespiratory support given as required. As long as the surgery continues and the patient is in the Trendelenburg position with left lateral tilt to prevent paradoxical arterial embolization of air, the surgical field must be completely flooded with saline. When the patient's haemodynamic condition permits, the reverse Trendelenburg position can be used to elevate the patient's heart above the surgical site to minimize further air entrainment.

References

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