FOCUS REVIEW

Beware of administration of methylergometrine prior to uterine incision and delivery; venous air embolism during caesarean section

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Keywords:
Venous air embolism
Caesarean section
Placenta previa
Methylergometrine
Oxytocics

SUMMARY

Caesarean section has been associated with venous air embolism. Uterine sinuses are vulnerable to the entrance of air, especially in the presence of placenta previa. We report a 32-year-old, gravida-IV admitted with antepartum haemorrhage and intra-uterine foetal demise at 34-weeks of gestation. She underwent an emergency caesarean section under general anaesthesia and developed venous air embolism intra-operatively which was managed successfully. We speculate, the administration of methylergometrine prior to uterine incision probably lead to ‘iatrogenic’ abruption of the placenta and venous air embolism. We recommend anaesthesiologists to avoid or be cautious while administering oxytocics prior to uterine incision (in case of intra-uterine foetal demise).

1. Introduction

Venous air embolism (VAE) is the entrainment of air from ruptured veins to the central venous system producing embolisation to the right heart or pulmonary artery. Uterine sinuses are theoretically vulnerable to the entrance of air, especially in the presence of placenta previa or during manual extraction of the placenta. The first fatal case of VAE in association with pregnancy was reported in 1850. Caesarean section (CS) has also been associated with VAE. Although the majority of VAE cases during CS are not fatal, many cases of massive VAE during caesarean section with devastating morbidity or mortality have been reported. We report a non-fatal case of VAE which developed as a result of administration of methylergometrine prior to uterine incision and ‘iatrogenic’ abruption of the placenta during CS.

2. Case report

A 32-year-old, gravida-IV, para-III with 3 living children admitted to the Obstetrics and Gynaecology department of our hospital at 34 weeks of gestation with history of vaginal bleeding and loss of foetal movement since 10 h. Her past history was unremarkable except that she had undergone CS (for foetal distress, under subarachnoid block) 5 years ago for the delivery of her 2nd child.

She was conscious and her peripheries were warm. Her heart rate was 140/min and blood pressure-106/74 mmHg. Cardiovascular, respiratory system and airway examination were unremarkable. Foetal cardiac activity was absent on cardiotocography. Laboratory investigations on admission were as follows; haemoglobin-8.8 g/dl (88 g/L), total leucocyte count-17 200 cells/mm³, platelets-387 000/mm³, blood group-A, urea-10 mg/dl (3.57 mmol/L), creatinine-0.6 mg/dl (53 μmol/L), prothrombin time-16.8 s (control-13 s), activated partial-thromboplastin time-43.2 s (control-28 s) and international normalised ratio-1.29. An obstetric ultrasound examination showed no cardiac activity, moulding of foetal head (consistent with foetal demise) with breech presentation and a low-lying anterior placenta partially covering the internal os without any evidence of abruption. One unit fresh whole blood was transfused and 3 more units were arranged. In view of her clinical condition [intra-uterine foetal demise >10 h, risk of disseminated intravascular coagulation (DIC) and shock (compensated)], it was decided to proceed with emergency CS under general anaesthesia.

In the operation theatre, peripheral venous access was secured with two large-bore cannulae, monitors attached [ECG (electrocardiography), NIBP (non-invasive blood pressure) and pulse-oximeter (SpO₂)] and 500 ml of normal saline was rapidly infused. The right radial artery was cannulated under local anaesthesia. Due to some problem in the pressure transducer (unable to ‘zero’), continuous monitoring of invasive pressures could not be initiated. Following pre-oxygenation, ‘modified rapid sequence induction’ (fentanyl-100 μg, thiopentone-175 mg and succinylcholine-100 mg intravenously) was performed and trachea was intubated with 7 mm cuffed endotracheal tube. Following intubation, vecuronium-5 mg and...
morphine-6 mg were administered intravenously and a double lumen central venous catheter was inserted through right internal jugular vein. Manually measured central venous pressure was 5 cm normal saline. Anaesthesia was maintained with \text{O}_2:\text{N}_2\text{O} (50:50) and halothane (0.3–0.6%). Patient developed transient hypotension (BP-63/37 mmHg, for about 3–4 min) following intubation which was treated with ephedrine-6 mg intravenously. Methylergometrine-0.2 mg was administered intramuscularly at the beginning of the surgery and the operating table was tilted to Trendelenburg position at uterine incision as requested by the obstetrician. Oxytocin-2 IU was administered intravenously at uterine incision followed by infusion. Near complete separation of placenta was noted on uterine incision (even before the baby was extracted).

Soon after the extraction of the baby (about 10 min after the administration of methylergometrine), there was a change in the pitch of the pulse-oximeter tone which attracted the attention of the anaesthesiologist. A drop of SpO\textsubscript{2} from 98% to 87% was noticed along with a reduction in ETCO\textsubscript{2} (end-tidal carbon dioxide) from 39 to 19 mmHg (5.19–2.53 kPa), ST depression (1.8 mm) and a transient drop followed by increase in heart rate (127–113–141/min) (Fig. 1). The obstetrician was alerted immediately (surgical field was still flooded with liquor) and the operation table was adjusted back to neutral position. N\textsubscript{2}O and halothane were switched-off immediately and ventilation continued with 100% O\textsubscript{2}. An attempt was made to aspirate air through the central venous catheter which was not successful. Intravenous crystalloid was infused rapidly. NIBP reading could not be obtained during this period (probably because of hypotension).

Over a period of next 3–5 min, SpO\textsubscript{2}, ETCO\textsubscript{2} and ST changes returned to baseline. Midazolam 2 mg was administered intravenously to ensure amnesia once the patient was haemodynamically stable. Examination of the alarm log of the monitor showed ‘low ETCO\textsubscript{2}’ before ‘low SpO\textsubscript{2}’. Halothane was restarted after 10 min and surgery was completed uneventfully. Intra-operative blood loss was approximately 500 ml and she received crystalloid-0.5 L and fresh whole blood-400 ml. Trachea was extubated at the end of the surgery on return of consciousness and she was shifted to surgical intensive care unit (SICU). She required dopamine infusion (5 \textmu g/kg/min) in the SICU (for about 12 h). Two units of fresh whole-blood were transfused during her SICU stay and repeat investigations were as follows; haemoglobin-8.4 g/dl (84 g/L), platelets-133 000/mm\textsuperscript{3}, prothrombin time-15 s (control-13 s), activated partial-thromboplastin time-44.6 s (control-28 s) and international normalised ratio-1.15. She did not recall any intra-operative event and further course in the hospital was uneventful.

### 3. Discussion

VAE is the entrainment of air from ruptured veins to the central venous system producing embolism at the right heart or pulmonary artery. Prerequisites for VAE are a vascular access and a gradient between the injury site and the right heart. VAE may lead to trapping of air bubbles in the pulmonary vessels producing a broad array of physiological changes ranging from simple gas exchange abnormalities to cellular injury and lung oedema due to the release of vasoactive mediators and increased microvascular permeability, acute cor pulmonale and right ventricular decompenensation secondary to the acute rise in right ventricular afterload. Consequently, the preloading of left ventricle and cardiac output can be severely diminished, followed by cardiac arrest.\textsuperscript{6}

The incidence of VAE during CS ranges from 10 to 97% depending on surgical position or diagnostic tools.\textsuperscript{3,7–9} VAE is responsible for about 1% of all maternal deaths.\textsuperscript{10} Surgery in the Trendelenburg position, abruptio placenta, placenta previa, exteriorisation of the uterus, manual extraction of the placenta, severe preeclampsia, antepartum haemorrhage and hypovolaemia are considered to be the risk factors for VAE during CS.\textsuperscript{11} Factors determining severity of VAE are rate, volume and duration of air entrapment, patient’s position at the time of VAE occurrence, size.
Further entrainment of air was probably prevented as the surgical field was still flooded with liquor and the operating table was neutralised immediately. We were fortunate that she did not develop massive VAE or AFE which could have made the situation worse. We did not put the patient in reverse Trendelenburg position as the effect of a 5–10° reverse Trendelenburg position on the incidence of VAE is controversial\(^\text{18}\) and it can aggravate haemodynamic instability.

We could not obtain an NIBP reading during the critical event. This could presumably be because of the transient hypotension during that period and an invasive blood pressure monitoring could have been extremely useful. We could not initiate continuous monitoring of invasive blood pressure because of technical problem and acknowledge that it was a shortcoming in the management of our patient. We acknowledge that VAE in our patient is a diagnosis of exclusion. End-tidal nitrogen monitoring\(^\text{20}\) is not available for routine clinical use. Precedorial Doppler or trans-oesophageal echocardiography could have been conclusive in diagnosing VAE; unfortunately, they were not available in our institute.

Favourable outcome of our patient could be attributed to many factors; embolism not massive, probably no AFE, timely detection and a vigilant anaesthesiologist. With this experience from our case, we think anaesthesiologists are better equipped to treat postpartum haemorrhage rather than a massive VAE or AFE which do not have definite treatment.

We recommend the anaesthesiologists to avoid or be cautious while administering oxytocsin before uterine incision (in case of intra-uterine foetal demise), lest it create ‘iatrogenic’ abstraction of placenta.

Financial support and conflicts of interest

There are no financial interests and conflicts of interest to declare.

References