International Journal of Obstetric Anesthesia (2007) **16**, 269–273

doi:10.1016/j.ijoa.2006.10.008

CASE REPORT

Rapid reversal of critical haemodynamic compromise with nitric oxide in a parturient with amniotic fluid embolism

N. J. McDonnell, B. O. Chan, R. W. Frengley

Departments of Anaesthesia and Intensive Care, Waikato Hospital, Hamilton, New Zealand

SUMMARY. We describe a case of amniotic fluid embolism presenting as cardiovascular collapse during labour. After initial resuscitation and emergency caesarean section, the patient was transferred to the intensive care unit with profound hypoxaemia, a high inotropic drug requirement and severe coagulopathy. A transoesophageal echocardiogram demonstrated acute right ventricular overload, severe pulmonary artery hypertension and marked diastolic dysfunction of the left ventricle secondary to a dilated right ventricle. The introduction of nitric oxide at 40 ppm produced a dramatic improvement in her cardiorespiratory status. Mother and baby both survived with no apparent long term sequelae.

Keywords: Amniotic fluid embolism; Nitric oxide; Transoesophageal echocardiography; Pulmonary hypertension; Anaphylactoid syndrome of pregnancy

INTRODUCTION

Amniotic fluid embolism (AFE) is a rare but catastrophic complication of pregnancy. In 1995 Clark suggested that the term "anaphylactoid syndrome of pregnancy" was a more accurate term for the condition.[1] The underlying pathophysiology is thought to be an initial intense vasoconstriction of the pulmonary vasculature, which results in severe hypoxia and acute right heart failure.[2] Clinically this presents as acute shortness of breath, fetal distress, and rapid progression to cardiorespiratory collapse.[3] Severe coagulopathy develops rapidly in most cases.[4] It has been postulated that nitric oxide has potential therapeutic benefit if the patient survives the initial insult, but its use has not been previously described in this setting.[5–7] We report the successful use of nitric oxide guided by transoesophageal echocardiography in a parturient with AFE.

Accepted October 2006

Nolan J McDonnell BHB MB ChB FANZCA, Department of Anaesthesia and Pain Medicine, King Edward Memorial Hospital for Women, Subiaco, Western Australia; **Brian O Chan** BHB MB ChB FANZCA, **Robert W Frengley** MB ChB FANZCA FJFICM, Departments of Anaesthesia and Intensive Care, Waikato Hospital, Hamilton, New Zealand.

Correspondence to: Dr Nolan J. McDonnell, Department of Anaesthesia and Pain Medicine, King Edward Memorial Hospital for Women, 374 Bagot Road, Subiaco, Western Australia 6008, Australia. Tel.: +61 8 9340 2222; fax: +61 8 9340 2260.
E-mail: nolanvini@mac.com.

CASE REPORT

A 35-year-old woman (G2, P0) presented to the delivery suite of a tertiary teaching hospital in spontaneous labour at 41 weeks and 6 days' gestation. She was otherwise in good health. Meconium-stained amniotic fluid was present on the initial vaginal examination performed by the obstetric team. The mother subsequently requested epidural analgesia.

An 18-gauge Tuohy needle and loss of resistance to saline were used to place an epidural catheter, with the parturient in the sitting position. An initial attempt at the L3-4 interspace was complicated by the presence of blood on advancement of the catheter, necessitating reinsertion at the L2-3 level. A 3-mL test dose of 2% lidocaine resulted in no adverse effects and effective epidural analgesia was established over the next 10 min using 0.375% ropivacaine 10 mL and fentanyl 100 μg. The anaesthetist remained in the birthing room for a further 10 min, completing documentation. At the end of this period the epidural block was assessed and was found to extend to T10 bilaterally. The parturient was comfortable and had been placed head up in a semi left lateral position.

Approximately 20 min after the epidural block had been assessed, sudden cardiovascular collapse occurred. The attending midwife had just completed a set of routine observations which were recorded as a blood pressure of 140/66 mmHg and a heart rate of 114 beats/min. Over a period of 30 s the patient complained of an unusual taste in her mouth (which she later described as very sweet) and difficulty breathing. This was followed by loss of consciousness and what was initially described to the arrest team as a seizure. An emergency was immediately declared by the attending midwife and a response team consisting of obstetric and anaesthetic staff was present within 2 min.

On initial assessment the patient appeared markedly cyanosed with no respiratory effort and no pulse detectable. Cardiopulmonary resuscitation was initiated, consisting of face mask ventilation with 100% oxygen and intermittent chest compression. Ephedrine was available and hence 6 mg was administered immediately. A palpable pulse was restored less than 2 min after the response team arrived, which appeared as a sinus tachycardia of between 140–160 beats/min on the defibrillator monitor. Despite restoration of cardiac output, the fetus was severely compromised with a heart rate of 60 beats/min. Spontaneous maternal respiratory effort rapidly returned. The decision was made to proceed to emergency caesarean section and the patient was transferred immediately to the operating theatre located on the delivery suite. At this stage the working diagnosis was of local anaesthetic toxicity with resulting neurological and cardiovascular compromise.

General anaesthesia was provided using a rapid sequence induction and a live male infant (4220 g) was delivered 17 min after the collapse. The initial Apgar scores were 1 and 5 at 1 and 5 min respectively. The infant was intubated, large amounts of thick meconium were suctioned from his trachea and he was then transferred to the neonatal intensive care unit.

Intraoperatively, after a short period of relative stability, the maternal haemodynamic state deteriorated requiring escalating doses of phenylephrine and noradrenaline. Progressive hypoxaemia necessitated increasing oxygen requirement and the application of positive end-expiratory pressure.

After abdominal closure marked fresh vaginal bleeding necessitated a repeat laparotomy. This showed a well contracted uterus with no cause for the vaginal bleeding being found. Multiple sites of microvascular bleeding were evident in the abdomen, signifying a rapidly developing coagulopathy. By this stage the differential diagnosis of the collapse and subsequent events had been expanded to include anaphylaxis, eclampsia, pulmonary embolism and AFE. After a thorough examination of the abdomen and birth canal no definitive source of bleeding was evident. The bleeding subsequently slowed down and her abdomen was closed; a hysterectomy was not performed.

Her coagulation profile at the time of reopening of the abdomen (approximately one hour after the initial cardiac arrest) showed a prothrombin ratio (PR) of 1.7 (normal 0.8–1.2), activated partial thromboplastin time (APTT) of 78 s (normal 24–40) and serum fibrinogen of 0.9 g/L (normal 1.5–4.0). Her haemoglobin was 12.2 g/dL and platelet count was 169×10^9/L. At the time of performing the coagulation studies she had received approximately 2 L of crystalloid solution. In total during her operative course she received 5 L of crystalloid, 2 units of packed red cells and 2 units of fresh frozen plasma.

Postoperatively she was transferred to the intensive care unit (ICU) where ventilation was continued. She remained hypoxaemic and a chest X-ray demonstrated widespread pulmonary infiltrates. She required large doses of inotropes, peaking with noradrenaline at 2.2 $\mu g\ kg^{-1}min^{-1}$ and adrenaline at 0.17 $\mu g\ kg^{-1}min^{-1}$. During the relatively short time period of reopening her abdomen and transferring her to the ICU, the coagulopathy worsened significantly. On admission to the ICU her coagulation studies showed a PR 2.8, APTT > 250 s, and a fibrinogen of 0.3 g/L. Her haemoglobin was 7.3 g/L and platelet count 51×10^9/L.

Urgent transoesophageal echocardiogram (TOE) was requested. As this case occurred in the early hours of a Sunday morning it was not immediately available on site and was performed approximately 4 h after the initial collapse (approximately 45 min after her arrival in the ICU). The TOE showed severe right ventricular dysfunction, indicated by right ventricular dilatation and hypokinesis, diastolic interventricular septal flattening, severe tricuspid regurgitation and elevated pulmonary artery pressures (peak 68 mmHg) (Figs. 1 & 2). No thrombogenic material was seen in the pulmonary circulation. It was felt at this stage that AFE was the most likely reason for her problem. This was consistent with her sudden cardiovascular collapse, seizure, hypoxaemia, coagulopathy and acute right ventricular failure.

On the basis of the acute right ventricular failure and pulmonary hypertension, nitric oxide was introduced at 40 ppm. This resulted in a dramatic improvement of her haemodynamic state, monitored in real time on the TOE. During the following hour her inotropic drug requirements rapidly decreased, such that 2 h after introduction of nitric oxide therapy she only needed adrenaline 0.07 $\mu g\ kg^{-1}min^{-1}$. Over the course of the day her FiO_2 was weaned from 100% to 40%. She was successfully extubated on the second day and discharged from the ICU on the fourth day. The epidural catheter was removed when the coagulation parameters had returned to normal; there was no adverse neurological outcome from the epidural catheter placement.