

Maternal-fetal Survival Following Amniotic Fluid Embolism: 2 Case Reports

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Citation

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Abstract

We report 2 cases of amniotic fluid embolism occurring during the peripartum period in which both the mother and newborn survived. Despite intraoperative haemodynamic compromise and coagulopathy, expeditious cardiopulmonary resuscitation and supportive therapy led to a salutary outcome in both instances.

INTRODUCTION

Amniotic fluid embolism (AFE) is a rare, catastrophic complication of pregnancy that occurs when the barrier between the amniotic fluid and maternal circulation is disrupted. The onset of signs and symptoms is typically acute and dramatic, classically characterised by dyspnoea, hypoxaemia, hypotension and cardiovascular collapse. Coagulopathy and haemorrhage are common and occur after the diagnosis is made, often clinically. There is a dearth of publications reporting non-fatal episodes of AFE presumably because mortality for both mother and fetus is usually high despite aggressive therapeutic interventions. We describe 2 cases of putative AFE which had typical presentations and complications in previously healthy parturients. Early diagnosis, aggressive resuscitation, correction of coagulopathy and prompt caesarean section resulted in favourable maternal and fetal outcome.

CASE 1

A 35-yr-old gravida 4 para 2 parturient with 2 previous lower segment caesarean sections (LSCS) and 1 missed abortion and a currently uneventful antenatal history was admitted for elective LSCS. She was given a subarachnoid block using 1.8mls of 0.5% hyperbaric bupivacaine, 10µg of fentanyl and 0.1mg of morphine. The level of sensory blockade as assessed by loss of cold sensation was T4 bilaterally. She remained conscious and haemodynamically stable throughout the initial stages of surgery until the delivery of the baby when she experienced sudden loss of consciousness and became cyanosed with the blood pressure (BP) falling to 40/27mmHg from 115/65mmHg and haemoglobin oxygen saturation (SpO₂) decreasing to 88%

from 99%. She responded promptly to intravenous (IV) vasopressors (total of 18mg of ephedrine and 1mg of metaraminol) with her BP improving to 115/60mmHg. She was intubated following IV thiopentone and succinylcholine and the SpO₂ improved to 99% on 100% oxygen. The initial end-tidal carbon dioxide concentration was 25mmHg which increased to 30mmHg subsequently. A provisional diagnosis of AFE was made and insertion of intra-arterial and central venous catheters were undertaken. A dopamine infusion was commenced to maintain her BP. An arterial blood sample was taken which showed that the blood gases, sodium, potassium and calcium were essentially normal. Her haemoglobin (Hb) concentration was 8.0g/dl. Fluid resuscitation was continued totalling 3.5l of crystalloids, 1.5l of colloids, 3.2l of packed cells, 5U of platelets, and 1.6l of fresh frozen plasma (FFP). Her vital signs remained stable throughout the rest of the surgery. Haemostasis of the operative site was secured with 2 peritoneal drains left in-situ and the patient was transferred to the intensive care unit (ICU) for post-operative mechanical ventilation and monitoring. The baby had an Apgar score of 8 at 1 min and 9 at 5 min.

Upon arrival in the ICU, blood investigations revealed that her Hb concentration was 7.9 g/dl and platelets was $128 \times 10^9/l$. Her coagulation profile was abnormal with a prolonged prothrombin time (PT) of 17.8 s (control 13.9 s) and activated partial thromboplastin time (APTT) of 49.2 s (control 32.4s). Peritoneal drainage output of haemoserous fluid increased from 50 to 90 ml/h warranting fluoroscopically-guided arterial embolisation of the uterine vessels to be performed on the first post-operative day.

Thereafter, the output from the drains was reduced significantly and the patient was extubated shortly after. The coagulopathy was treated with cryoprecipitate 10U and FFP 500mls 6 hourly, before normalizing on the second post-operative day. Disseminated intravascular coagulopathy was evident with d-dimer levels $>2 \mu\text{g/ml}$ and soluble fibrinogen monomers in the blood. In addition, the anaemia was corrected with a total of 3l of packed cells. The patient's stay in the ICU remained uneventful till her discharge on the fourth post-operative day. She left the hospital on the eleventh post-operative day with no neurologic sequelae. Her baby was deemed normal on subsequent follow-up at the outpatient clinic.

CASE 2

A 34-yr-old gravida 3 para 2 lady at 40 wk gestation was admitted for spurious labour. She had been well antenatally with no significant past medical and obstetric history. Her previous pregnancies were uneventful and resulted in normal vaginal deliveries.

She underwent induction of labour with misoprostol because of post-dates with an unfavorable cervix at 1cm dilatation. Initially, she remained well with contractions of 1 every 2 min. The cardiotocogram (CTG) showed variable decelerations but with good recovery and variability. Four hours after induction, there was spontaneous rupture of the amniotic membrane emitting clear liquor. The CTG at this point showed a baseline fetal heart rate of 135 to 145 per min with late decelerations. Soon after, the patient suddenly became unconscious, tachypneic and had a thready pulse with unrecordable blood pressure. A provisional diagnosis of AFE was made and the patient was immediately sent to the operating room (OR) across the labour ward for a 'crash' LSCS.

On arrival in the OR, the patient was intubated after succinylcholine 100 mg IV while applying cricoid pressure and subsequently ventilated with 100% oxygen. The electrocardiogram (ECG) showed that there was electromechanical dissociation for which cardiopulmonary resuscitation (CPR) was instituted, whilst the emergency LSCS was being performed. The baby was delivered 5 min after maternal collapse in the delivery suite and 1 min after intubation. Adrenaline 1 mg IV was given to the mother after which she developed transient ventricular tachycardia but this resolved spontaneously. BP which was initially 96/30 mmHg stabilised at about 130/70 mmHg with a continuous IV infusion of dopamine at 3 $\mu\text{g/kg/min}$. The

right radial artery was cannulated for intra-arterial blood pressure monitoring and a central venous catheter was inserted in the right internal jugular vein. The initial central venous pressure was 20 mmHg which later settled to approximately 15 mmHg. Intraoperatively, the patient was clinically coagulopathic for which 1.5l of FFP and 10U of cryoprecipitate were transfused. Blood loss of about 1l was restituted with 800ml of packed cells. The patient was maintained on an FiO_2 of 1.0 and the SpO_2 reading was 100% throughout surgery. Haemostasis was secured and the patient was kept intubated and transferred to the ICU for further management

Blood investigations done in the OR prior to transfusion of blood products revealed that the coagulation profile was abnormal, with a prolonged PT of >120 s (control 13.9 s) and the APTT of 180 s (control 32.4 s). She was also anaemic with a Hb concentration of 7.76 g/dl, a total white count of $18 \times 10^9/l$ and platelet count of $250 \times 10^9/l$. The arterial blood gas revealed a pH of 7.249, PaO_2 of 222 mmHg ($\text{FiO}_2=1$), PaCO_2 of 30.8mmHg, base excess of -12.9mmol/l and standard bicarbonate of 13.2 mmol/l. The fibrinogen concentration was 6.9 mol/l (normal range: 5.3 – 14.1 mol/l), d-dimer concentration $>2 \text{g/ml}$ and there were soluble fibrin monomers in the blood.

Shortly after admission to the ICU, her coagulation profile had normalised with a PT of 14.8 s and APTT of 31 s. The Hb and platelet concentrations were 9.5 g/dl and $123 \times 10^9/l$ respectively. She regained consciousness 2 hr later and recorded a Glasgow coma score of 15. She remained haemodynamically stable and maintained an urine output of $\sim 1\text{ml/kg/hr}$. Extubated was effected 15 hr after ICU admission and remained well till discharge from the hospital on the ninth post-operative day.

The baby was intubated 5 min after delivery and was transferred to the neonatal ICU. Umbilical cord blood showed that there was laboratory evidence of perinatal asphyxia (pH 7.193, PCO_2 21.5 mmHg, base excess -18mmol/l , and standard bicarbonate 8.1 mmol/l). The baby was discharged well after 13 days stay in hospital and subsequently had normal developmental milestones.

DISCUSSION

Amniotic fluid embolism (AFE), also known as anaphylactoid syndrome of pregnancy, was first described by Meyer in 1926 but it was only brought to prominence in 1942 when Lushbaugh and Steiner reviewed the unexpected deaths of 8 patients and found material consistent with

amniotic fluid debris in the pulmonary vasculature (1). The pathogenesis of AFE is still nebulous; historically, cardiovascular collapse was believed to result from mechanical obstruction of the pulmonary circulation by amniotic fluid debris. Currently, however, a combination of left ventricular dysfunction, acute lung injury and clotting factor activation has been invoked (2).

The incidence of this syndrome ranges from 1 in 8000 to 1 in 80 000 pregnancies (3) and this variability can be attributed to the inaccuracies in reporting the cause of maternal death, differences in clinical presentation (the disease spectrum ranges from a subclinical entity to one which is rapidly fatal) and the difficulty in confirming the diagnosis in survivors, sans autopsies (4). A literature review by Morgan >20 yr ago (3) elicited that 86% of cases are fatal, with 50% of maternal deaths occurring within 1 hr of symptom onset. A more recent report by Clark et al. (5) cited a 61% maternal mortality rate (MMR), with 15% of the survivors remaining neurologically intact. In addition, the demise rate of fetuses in utero at the time of a symptomatic AFE event was 61% (5). However, Gilbert and Danielsen reported in their 1999 population-based series that the MMR is only 26.4% (6), which is significantly lower than previously reported. Analogous to our report, documentation of survival from AFE has appeared in the peer-reviewed literature very recently (2,7,8,9).

The classic presentation of AFE was described as sudden, profound and unexpected shock followed by cardiovascular collapse disproportionate to the amount of hemorrhage, and in most cases, death (10). The incidence was also thought to be higher in elderly multiparous patients who had rapid and intense labour (4). Although cardiovascular collapse was almost invariably present, the presenting signs and symptoms in Morgan's series were respiratory distress in 51%, hypotension in 27%, coagulopathy in 12% and seizures in 10% (3). Our subjects typically presented with hypotension and cardiovascular collapse respectively. Clark et al. noted that for prepartum occurrence of AFE, seizures and respiratory distress were present in ~30% of cases, whereas postpartum hemorrhage secondary to an isolated coagulopathy was the presenting feature in 54% of postpartum cases (5).

AFE can occur only when there is a breach in the barrier between the amniotic fluid and maternal circulation. The three most common routes of entry are the endocervical veins, the placental site and a traumatised uterine site (11).

There is unfortunately no routine or standardized diagnostic scheme to confirm AFE and hence it remains a diagnosis of exclusion. A review of the largest case-series to date concluded that the physiologic and haematologic sequelae of AFE resemble septic or anaphylactic shock rather than an embolic phenomenon (12) and hence this obfuscates matters. The plethora of differential diagnoses include air or thrombotic pulmonary emboli, septic shock, acute myocardial infarction, cardiomyopathy, anaphylaxis, aspiration pneumonitis, placental abruption, eclampsia, uterine rupture, transfusion reaction and local anaesthetic toxicity (13). There are no laboratory investigations which are pathognomonic of AFE but the following may support the diagnosis: complete blood count, coagulation profiles, arterial blood gases, serum tryptase, chest x-ray, ventilation-perfusion lung scans, ECG and echocardiography (4). Masson et al. (13) also described a method based on blood obtained from the pulmonary microvasculature where components of amniotic fluid i.e., squamous cells and mucous strands can be identified. Other novel studies which involve testing maternal serum for TKH-2 antibodies (14) and zinc coproporphyrin (15) seem promising, though further studies are needed to elucidate their practical utility and reliability (4).

The management of AFE is essentially empirical and supportive, directed towards the maintenance of oxygenation, ventilation, circulatory support and correction of coagulopathy (4). If the fetus is sufficiently mature and undelivered at the time of maternal cardiac arrest, caesarean section should be instituted as soon as possible to facilitate maternal CPR and limit fetal morbidity and wastage (16, 17).

Oxygenation should be maintained at normal levels to avoid irreversible neurological injury. Haemodynamic stability should be sustained with volume loading, vasopressors and/or inotropes. Coagulopathy should be treated with FFP and platelets and haemorrhage with packed red blood cells. Cryoprecipitate has been deemed useful in cases with hypofibrinogenaemia or adult respiratory distress syndrome (18). Recently, the successful application of extracorporeal membrane oxygenation and intraaortic balloon counterpulsation (9), as well as cell-salvage combined with blood filtration (19) has been documented. Other proposed modalities which allegedly have salutary effects on AFE outcome include serine protease inhibitors, cardiopulmonary bypass, nitric oxide, inhaled prostacyclin and high-dose corticosteroids (4).

CONCLUSION

In summary, we described 2 parturients with no premorbid history who presented with typical signs and symptoms suggestive of AFE, i.e., sudden onset of hypoxia, cardiovascular collapse and coagulopathy during the immediate peripartum time frame. The salutary maternal and fetal outcomes that ensued are uncommon and underscore the importance of expeditious maternal resuscitation and prompt caesarean section following maternal collapse.

AFE continues to be one of the most feared and devastating complications of pregnancy. Despite recent documented evidence that attendant mortality rates may be on the wane (6), it yet remains an enigmatic, unpredictable, unpreventable and for the most part, untreatable disease with generally dismal clinical outcomes. It thus behoves obstetric care personnel to maintain a high index of suspicion for the disorder so as to facilitate early recognition and management.

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