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Amniotic Fluid Embolism After Intrauterine Fetal Demise

Kristensen K¹, Langdana F¹, Clentworth H¹, Hansby C¹ and Dalley P²

Abstract

Amniotic fluid embolism is a rare but devastating condition. The underlying pathophysiology involves the entry of foetal antigens into the maternal circulation followed by a complex sequence of reactions and activation of the pro-inflammatory mediator systems similar to the systemic inflammatory response syndrome. We present a case of the successful treatment of severe amniotic fluid embolism in a 41 year old woman undergoing emergency caesarean section at 36 weeks gestation for placental abruption with intrauterine foetal demise. The treatment included prolonged cardiopulmonary resuscitation, emergency obstetrical hysterectomy and re-operation with pelvic-abdominal packing and intra-aortic balloon pump (IABP). The coagulopathy was treated with packed red cells, Platelets, FFP, cryoprecipitate and recombinant factor VIIa transfused and titrated with point-of-care testing using Thromboelastography. The woman's recovery was remarkable with normal neurological, cognitive and near normal cardio-pulmonary functions. The patient suffered from severe renal impairment and remained on outpatient dialysis treatment. We would like to highlight the importance of a multidisciplinary approach and ongoing briefing and continuous evaluation of patient status in complicated cases of amniotic fluid embolism.

Keywords: Amniotic Fluid Embolism; intra-aortic balloon pump; emergency obstetrical hysterectomy; pelvic-abdominal packing; intrauterine foetal demise; disseminated intravascular coagulation; renal failure

- 1 Department of Womens Health, Wellington Regional Hospital CCDHB and Otago University, Wellington, New Zealand
- 2 Department of Anaesthesia and Pain Management, Wellington Regional Hospital CCDHB and Otago University, Wellington, New Zealand

Corresponding author: Karl Kristensen

✉ karl.kristensen@med.lu.se

Department of Womens Health, Wellington Regional Hospital CCDHB and Otago University, Wellington, New Zealand.

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Introduction

The amniotic fluid embolism syndrome (AFES) is a rare and often fatal obstetric condition and it remains one of the main causes of maternal mortality in the developed countries. The incidence varies from 2 to 6 per 100,000 depending on the inclusion criteria and the type of study [1,2]. Series restricted to patients in whom all classic signs and symptoms of AFES are present suggest mortality rates exceeding 60% [1].

The classic triad of sudden hypoxia, hypotension and coagulopathy with an acute onset during labour or immediately after delivery forms the hallmark of the AFES diagnosis. Variant presentations in which one or more components may be absent or minimal have been described and in such cases, the diagnosis is more difficult and must include the exclusion of plausible alternative diagnoses [3].

In the most classic form a woman in labour or shortly after

vaginal or caesarean delivery sustains an acute onset dyspnoea and desaturation followed by sudden cardiovascular collapse. This is commonly followed by cardiac arrest and coagulopathy. Patients whose initial clinical manifestations do not include fatal cardiac arrest often develop a severe coagulopathy, which may ultimately be the principle cause of death [1].

The underlying pathophysiology is not completely understood but involves the entry of foetal antigens into the maternal circulation followed by a complex sequence of reactions and activation of the pro-inflammatory mediator systems similar to the systemic inflammatory response syndrome (SIRS) [1,4,5]. Case reports have indicated an initial transient period of pulmonary and systemic hypertension followed by a profound depression of left ventricular function with normal pulmonary arterial pressures by trans-esophageal echocardiogram [1,6].

AFES is primarily a clinical diagnosis of exclusion. Several specific laboratory or autopsy findings have been proposed to confirm the

diagnosis. The laboratory findings generally support the theory of an inflammatory based mechanism of disease, but none of these findings are of significant or clinical use [1]. Moreover, several conditions and factors have been associated with AFES however none of these have been established as a causative link. Therefore the AFES remains both unpredictable and unpreventable [1].

In this case we present a successful maternal outcome of severe amniotic fluid embolism after abruption of the placenta and intrauterine foetal demise.

Case Report

A 41 year old pregnant woman, gravida 5 para 4 was admitted with a small antepartum hemorrhage at 36 weeks of gestation. The medical and obstetrical history was unremarkable and there were no antecedent risk factors during the prenatal care.

The clinical presentation involved severe hypertension of 180/110 mmHg (settling to 160/95 mmHg after single 200 mg dose of Labetalol) and mild tachycardia 100 bpm. The other vital signs were satisfactory. On examination the patient presented with a firm, tender uterus. The bedside ultrasound scan revealed intrauterine foetal demise, the placenta was located anterior high and a retro-placental hematoma was identified. The presenting part was high above the pelvic and cervix was posterior and closed with only a small amount of old blood on vaginal examination. Initial blood count revealed an Hb 130 g/L, platelets $150 \times 10^9/L$, creatinine was notably increased at 150 $\mu\text{mol/L}$ and there were subtle signs of coagulopathy (INR 1.1 (ref. value 0.8-1.2), APTT 38 sec (ref. value 28-38 sec) and Fibrinogen 1.5 g/L (ref. value 2-4.3 g/L).

Two senior consultants examined the patient and discussed the situation. The situation was considered an emergency situation (placental abruption with renal failure and coagulopathy) with the need of urgent delivery. The mode of delivery was discussed and delivery by emergency Caesarean section was favored rather than attempting induction of labour.

The patient was consented to an emergency lower segment Caesarean section under regional anaesthesia. The Caesarean section revealed a small amount of blood-stained ascites and a Couvelaire uterus. A female infant of 2.5 kg (Apgar scores 0/0/0) was delivered with small amounts of coagulated blood and complete abruption of the placenta. After two-layer suturing with satisfactory haemostasis and tone (Estimated blood loss < 500 mL) the patient went into sudden cardiac arrest with the reading of pulseless electrical activity on the electrocardiogram (ECG). Cardiopulmonary Resuscitation (CPR) with endotracheal intubation was commenced. The patient remained unresponsive to the initial resuscitation and treatment with repeated doses of Epinephrine as per Advanced Cardiovascular Life Support (ACLS) protocol [7].

Due to the continuing haemodynamic instability a trans-esophageal echocardiogram (TEE) was performed. This demonstrated profound ventricular dysfunction with predominantly left sided cardiac failure. The TEE demonstrated reasonable right ventricular function and was able to exclude other causes of sudden cardiac arrest, including massive

pulmonary thromboembolism, aortic dissection, coronary ischemia, pericardial tamponade and valvular dysfunction. After >20 minutes of CPR a weak femoral pulse was present but a second round of CPR was restarted after 5 minutes. Following the echocardiographic findings high dose infusions of Adrenaline (epinephrine), Milrinone and Noradrenaline (norepinephrine) were commenced. A Vasopressin infusion was subsequently added.

After about 8 minutes of additional CPR (in total about 28 minutes of CPR) the patient regained circulation (heart rate 140 bpm and blood pressure 70/30 mmHg), the uterus remained bulky and oozing despite treatment with intra-myometrial Carboprost injections and B-Lynch sutures. Subsequent to a multi-disciplinary discussion including the on-call Obstetricians, Anesthesiologists and Intensive care specialists the decision was made to perform a hysterectomy. A subtotal hysterectomy was performed. Wide drains were inserted into the peritoneal cavity and abdomen closed in layers.

Due to ongoing haemodynamic instability over the following 20-30 minutes with ventricular dysfunction despite high dose inotropic support the decision was made to use mechanical circulatory support. There were no obvious signs of myocardial infarction on the ECG. Due to the more rapid availability of intra-aortic balloon pump (IABP) this was organized and inserted by cardiothoracic surgery with significant improvement in cardiac function immediately obvious. The option to proceed to extracorporeal membrane oxygenation (ECMO) was also discussed but proved unnecessary.

After approximately 90 minutes with patient still in OT, the patient presented with severe coagulopathy/ disseminated intravascular coagulation (DIC) with epistaxis and massive blood loss from the abdominal drains. Tranexamic acid was administered and re-operation was performed with pelvic-abdominal packing, extra sutures, diathermia and administration of Flow-Seal for haemostasis.

The DIC was aggressively treated after invoking our institutional massive transfusion protocol (stepwise transfusion of blood products). Packed red cells (12 units), Platelets (3 units), FFP (12 units), and cryoprecipitate (6 units) were transfused and titrated with point-of-care testing using Thromboelastography. After discussion with a transfusion medicine specialist recombinant factor VIIa (rFVIIa) was given at 90 mcg/kg before the transfer to the ICU.

The DIC resolved within the following 8-10 hours, the pelvic-abdominal packs were removed on day 3 and the patient remained intubated for 5 days.

Recurring episodes of low grade fever and CRP-levels 100-200 was treated with iv Tazocin, Vancomycin and Fluconazole followed by oral Augmentin. An abdominal-wall collection (8x6x2 cm) was drained under USS guidance, however all cultures came back negative.

Immediate after the systemic collapse the patient went into an acute renal failure with anuria for 7-8 days and slow recovery and the patient was discharged to out-patient dialysis after 5 weeks in-

hospital. The pituitary hormone levels were normal at discharge. Repeat echocardiography at three weeks revealed a mild left ventricular failure (ejection fraction of 28%) and with mild to moderate mitral valve regurgitation, most likely a complication to the prolonged period of hypoxia. The neurological recovery was remarkable and after intensive physiotherapy and top-up blood transfusions the patient was discharged with no obvious signs of neurological or cognitive morbidity. The patient remains on out-patient hemodialysis twice weekly at 6 months after the AFES. The creatinine levels are slowly improving (current level 200-250 $\mu\text{mol/L}$), but the patient could become a candidate for renal transplantation.

Discussion

The treatment of AFES is primarily supportive and based on the observed pathophysiology. After cardiac arrest the standard and advanced cardiovascular life support algorithms are applied. Coagulopathy and ensuing hemorrhage are managed by aggressive blood and component replacement. However mortality rate remains high despite successful management of cardiorespiratory collapse and expert management of bleeding and component replacement.

The reported estimates of maternal mortality have varied greatly and appear to be largely dependent on the criteria of inclusion of cases of AFES. Series restricted to patients in whom all classic signs and symptoms are present suggest mortality rates exceeding 60% [3].

This case demonstrates that with efficient CPR combined with aggressive and persistent treatment of AFE and DIC with IABP and blood-products a favorable outcome with a minimal degree of

morbidity is possible even in a situation with prolonged (about 28 minutes) CPR and DIC. During the intense hours in the OT and the following weeks there was a superb multidisciplinary teamwork with frequent communication and support.

We would like to highlight the importance of a multidisciplinary approach and ongoing briefing and re-evaluation of patient status. In the literature we have found one previous report of the successful use of IABP for AFE [8]. The IABP is a well-known and accepted treatment for acute cardiogenic shock.

The use of recombinant factor VIIa to successfully manage disseminated intravascular coagulation from AFES is controversial and recommended only for AFE when the hemorrhage cannot be stopped by massive blood component replacement [9].

The packing of the pelvic-abdominal cavity has proven safe and effective in controlling major obstetrical hemorrhage and should be considered when there is refractory massive bleeding from raw surfaces or suture-lines in complicated obstetrical or gynecological operations [10].

A multidisciplinary approach is the key to managing a complicated situation, ongoing discussion and frequent feedback/briefing is essential. The patient and her family were updated by the Obstetrical and ICU teams about the critical condition on a day by day basis. Psychological support and counseling was offered and accepted by the patient and her family. A team debriefing was held on day three and in hindsight we should probably have arranged an additional debriefing with the same team after two-three weeks to catch up. The case will be discussed at the multidisciplinary mortality and morbidity meeting in Wellington Hospital.

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