

## CASE REPORTS

## Amniotic fluid embolism: emergency management

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**Abstract**

**A case of successful outcome is described in a patient with amniotic fluid embolism presenting to the accident and emergency department. Diagnostic features and guidelines for management are outlined.**

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Key terms: Amniotic fluid embolism; diagnosis; management

Amniotic fluid embolism is the most devastating condition known in pregnant women. The incidence is reported as 1 in 80 000 deliveries in England and Wales. The mortality rate is 86% with 50% of deaths occurring in the first hour of the onset of symptoms.<sup>1 2</sup>

**Case report**

A 36 year old pregnant woman arrived in the accident and emergency (A&E) department at 4.35 am. She was cyanosed, fitting, and had no palpable pulse or recordable blood pressure.

At about 4.15 am she had got out of bed to go to the lavatory. Her husband, a general practitioner, heard a bump and found his wife lying beside the wash basin. She was having a generalised convulsion and had a small scalp laceration. She was at 20 weeks gestation with a twin pregnancy. She had maintained good health and had had normal deliveries in each of her three previous pregnancies. In this pregnancy she was known to have a minor degree of placenta praevia with an admission to hospital two weeks previously with some vaginal bleeding. She had suffered no hypertension or proteinuria.

Initial treatment consisted of external cardiac massage, intubation, ventilation, and intravenous fluids in large volume. A wedge (foam pillow, blanket) was placed under the patient's right lumbar region to prevent compression of the inferior vena cava by the gravid uterus. She had pinpoint petechial haemorrhages over the chest wall and bleeding from the nose, mouth, vagina, and needle puncture sites.

The initial differential diagnosis included eclampsia, sepsis with a possible coagulopathy, or subarachnoid haemorrhage. A neurosurgeon and obstetrician were called.

After resuscitative measures her blood pressure improved to 70-80 mm Hg systolic. The neurosurgeon felt that the history of head injury and the presence of fitting indicated the need to exclude intracranial pathology. A computerised tomography scan of the brain revealed no abnormality.

Initial blood tests showed a picture of disseminated intravascular coagulopathy (DIC) with depletion of platelets, clotting factors, and fibrin and an increase in fibrin degradation products: Hb 12.7 g/dl on admission, dropping to 5.7 g/dl six hours later, platelets 58 000/ $\mu$ l, prothrombin time 25.4 s (normal 15-19 s), PTTK 73.1 s (normal 39-51 s), thrombin clotting time 34.4 s (normal 14-16 s), fibrinogen titre 0.7 g/litre (normal 2-5 g/litre), fibrin degradation products 320  $\mu$ g/ml (normal <10  $\mu$ g/ml). Treatment for DIC was started.

The obstetrician felt that the diagnosis was amniotic fluid embolism. Eclampsia was thought to be an unlikely diagnosis because there had been no pre-eclampsia in the pregnancy; however, management of the two conditions with this presentation would have been the same. He carried out an emergency caesarean section and evacuation of the uterus. Some old clots were present in the uterus from the previous threatened abortion and there was also fresh blood present. By the completion of surgery the patient had received 1.5 litres of crystalloid, 1 litre of colloid, 2 units of 0-negative uncrossmatched blood, 10 units of packed cells, some group specific and some crossmatched, 20 units of cryoprecipitate, 4 units of fresh frozen plasma, and 6 packs of platelets. Her pulse was 110 beats/min and blood pressure 100-110 mm Hg systolic.

She was transferred to the intensive care unit where she was electively ventilated for 48 hours. Her cardiovascular indices and coagulation screen showed continuous improvement. Urinary output remained normal. She was extubated two days after her presentation and made an uneventful recovery with no permanent sequelae.

**Discussion**

Clark<sup>3</sup> postulates that amniotic fluid embolism occurs when abnormal amniotic fluid enters the maternal circulation: normal amniotic fluid may enter without ill effect. The two main pathological effects are haemodynamic collapse and coagulopathy. A biphasic model<sup>1 3</sup> has been postulated to describe the haemodynamic consequences. The initial response to the amniotic fluid is vasospasm with resultant transient pulmonary hypertension and profound hypoxia. This phase lasts 15-30 minutes, and may account for the 50% of patients who die in the first hour. The second phase involves left heart failure with a variable secondary increase in pulmonary artery pressure and return to normal right heart function. The left ventricular failure may be due to

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hypoxic injury to the left ventricle secondary to reduced coronary artery blood flow or myocardial depression.<sup>3-5</sup>

In addition to the haemodynamic collapse, 40% of patients develop coagulopathy, ranging from DIC to a minor disturbance of platelet count. The mechanism is not clearly understood but the potent thromboplastic-like effects of trophoblast are well recognised and may trigger the coagulopathy.

#### CLINICAL PRESENTATION

The condition most frequently presents during labour but cases have been reported in first and second trimester abortion and as late as 48 hours postpartum.

The first symptom is the sudden onset of dyspnoea and hypotension, which is often followed in minutes by cardiovascular collapse and respiratory arrest. In 10–20% of cases these initial events may be heralded by seizure-like activity, as in this case. The fall against the wash basin was obviously due to the cardiovascular collapse. In 40% of patients this is followed by coagulopathy. In 10–15% of patients coagulopathy may be the presenting manifestation<sup>1 2 6</sup>

#### DIAGNOSIS

This is based on the clinical presentation of cardiovascular collapse and the laboratory findings of coagulopathy in a pregnant woman. The differential diagnosis includes aspiration pneumonia, acute myocardial infarction, pulmonary embolus, and in cases where coagulopathy is a dominant feature, placental abruption, septic abortion, intrauterine infection, pre-eclampsia, and eclampsia. Blood abnormalities show depletion of fibrinogen, platelets, and other clotting factors, increase in fibrin degradation products, and prolonged partial thromboplastin and prothrombin times.

Squamous cells and other debris of fetal origin may be demonstrated in blood aspirated from the central veins of pulmonary artery circulation. Recent studies of pregnant women undergoing pulmonary artery catheterisation, however, have shown that the detection of squamous cells in the maternal pulmonary artery circulation is a common finding and not diagnostic of amniotic fluid embolism.<sup>7-9</sup>

#### TREATMENT

The three goals of treatment are aggressive oxygenation, treatment of circulatory collapse, and combating the coagulopathy.

##### *Circulatory collapse*

In patients with no cardiac output, cardiopulmonary resuscitation is begun. Oxygen should be given at high concentrations and unconscious patients should be immediately intubated and ventilated. Intravenous access with

wide bore cannulae should be obtained. The preload should be optimised by giving rapid infusion of intravenous fluids, and dopamine should be used to improve the left ventricular failure.

##### *Coagulopathy*

As soon as there is any concern, 15 ml of blood should be taken and used as follows: 2.5 ml into EDTA for full blood count with emphasis on the packed cell volume and platelet count; 4.0 ml into citrate for coagulation screen and fibrin degradation products, and the rest into a plain tube for cross matching. All these tests are straightforward and should be available from any routine haematology laboratory; a high powered coagulation laboratory is not necessary.

Once the blood has been taken, treatment should be begun before the results of the tests are known. Fresh frozen plasma (FFP) does not have to be cross matched but should be the same ABO and rhesus group as the patient.

While waiting for FFP and blood, circulating volume must be restored with plasma substitutes to avoid renal shutdown. If effective circulation is restored without too much delay, fibrin degradation products will be cleared from the blood mainly by the liver, which will further aid restoration of normal haemostasis. This is an aspect of management which is often not appropriately emphasised.<sup>10</sup>

#### CONCLUSIONS

Amniotic fluid embolism is a rare but dangerous complication of otherwise normal pregnancy. The rapid onset means that these patients may present to the A&E department. The condition carries a high mortality which may be reduced by aggressive early resuscitation.

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