AMNIOTIC FLUID EMBOLISM: A CATASTROPHIC COMPLICATION OF PREGNANCY

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INTRODUCTION

Amniotic fluid embolism (AFE) is a rare, life-threatening obstetric emergency, posing a risk to both mother and baby. It ranks 5th as the leading cause of direct maternal death. Presentation is variable, although is highly suspicious in sudden, unexplained maternal collapse with hypotension, hypoxemia with coagulation disturbances. In the UK, currently the incidence is estimated between 1 in 50,000 pregnancies with mortality rate in the range of 11% to 61%.

We present a case of AFE which was successfully managed by a multidisciplinary team.

DIAGNOSIS

There is no pathognomonic marker of AFE. The diagnosis is one of the exclusion criteria. It is aided by specific and non-specific tests (see figure 2). The presence of fetal squamous cells in broncho-alveolar lavage may support the diagnosis (see figure 3).

Table 3 Diagnosis of AFE

Test	Possible findings
Non-specific tests	
Full blood count	Low haemoglobin
Coagulation	Low platelets, increased PT and APTT, low fibrinogen
Arterial blood gas	Hypoxaemia, raised Pa_{CO2}
Chest X-ray	Normal, cardiomegaly, pulmonary oedema
ECG	Right heart strain, rhythm abnormalities
V/Q scans	*V/Q* mismatch
Echocardiogram	Right or left ventricular dysfunction, low ejection fraction
More specific tests	
Pulmonary blood sample	Presence of squamous cells coated with neutrophils and presence of fetal debris
Sialyl Tn antigen	Raised
Zinc coproporphyrin	Raised
Serum tryptase levels	Normal or raised

PT, prothrombin time; APTT, activated partial thromboplastin time; V/Q, ventilation/perfusion.

Figure 2. Diagnosis of AFE

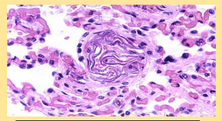

Figure 3. Squamous cell within a pulmonary arteriole

The differential diagnosis includes: obstetric causes (e.g. eclampsia, placental abruption, peripartum cardiomyopathy) and non-obstetric causes (e.g. anaphylaxis, pulmonary embolism, pulmonary aspiration, septic shock, haemorrhagic shock, myocardial infarction, drug toxicity and total spinal anaesthesia).

MANAGEMENT

The RCOG recommends early recognition, prompt resuscitation, and supportive measures.

- Oxygenation – maintenance may necessitate intubation and ventilation.
- Haemodynamic stability – rapid IV filling, direct acting vasopressors e.g. phenylephrine and inotropes are necessary to improve CO. Surgical intervention may be needed to control haemorrhage.
- Uterine tone – should be maintained using oxytocin, ergometrine and prostaglandins. Bimanual uterine massage and uterine packing may help reduce blood loss.
- Coagulation – plasma, cryoprecipitate and platelets are frequently required. Recombinant factor VII used to treat uncontrollable massive obstetric haemorrhage.
- Delivery of baby – pregnant women >20 weeks gestation who collapse with no return of spontaneous circulation after CPR, must have an immediate C-section. Delivery is recommended by 5 minutes.

PATHOPHYSIOLOGY

Risk factors include:

- Advanced maternal age; - Multiparity;
- Polyhydramnios; - Uterine rupture;
- Placenta accreta; - Chorioamnionitis.

The amniotic fluid enters maternal circulation via ruptured membranes or ruptured uterine or cervical vessels down a pressure gradient from uterus to veins. According to Clark, AFE is caused by fetal antigens in the amniotic fluid stimulating a cascade of endogenous immune mediators, producing a reaction similar to anaphylaxis. Figure 1 gives a detailed description of the pathophysiology of AFE.

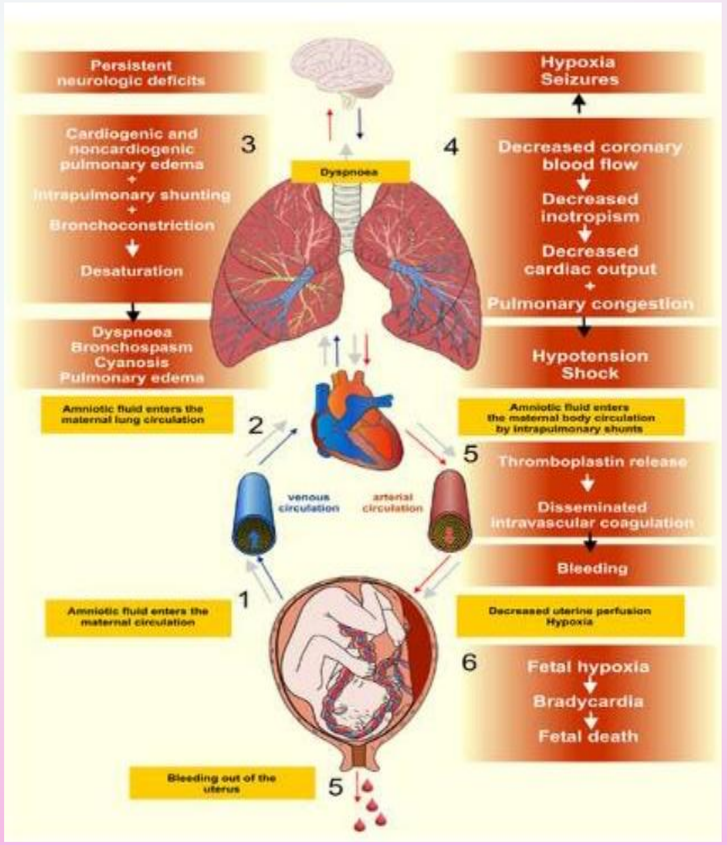

Figure 1. Pathophysiology of AFE

CASE STUDY

- A 39 year old G7P1+5 (1 term vaginal delivery) was induced at 42 weeks gestation.
- She had sudden onset of respiratory arrest, hypotension and tachycardia following artificial rupture of membranes (ARM).
- She was rapidly intubated and ventilated and delivered by Caesarean section.
- Uterine atony developed within minutes after delivery and was treated with multiple doses of uterotonics. She had an extension of the right uterine angle and total blood loss was calculated as 4500ml for which massive haemorrhage protocol was initiated.
- During an attempt to control bleeding sutures were placed at the extension to the right of the uterus. These sutures were found to be subsequently close to the right ureter causing obstruction. Once the bleeding was controlled a urologist was called and the ureter was subsequently re-implanted into the bladder.
- She had significant coagulopathy secondary to DIC for which she was given 8 units of FFP, 4 units of packed red cells and fibrinogen.
- The working diagnosis was AFE.
- The differential diagnosis also included pre-eclampsia and pulmonary embolism.
- A few days postnatal, she was diagnosed with bilateral pulmonary embolism, for which she received therapeutic Enoxaparin.
- She ultimately made a full recovery and the baby has no developmental concerns 1 year later.

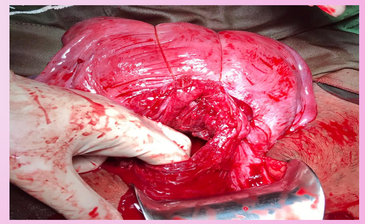

Figure 4. Uterine atony

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