CASE REPORT

An example of prompt and appropriate multidisciplinary management leading to an exceptionally good outcome: a case complicated by amniotic fluid embolism

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SUMMARY

Amniotic fluid embolism (AFE) is a rare and serious phenomenon; we describe a rare case of AFE occurring after a second trimester surgical termination in a private clinic, which, with prompt transfer and appropriate multidisciplinary management, had an exceptionally good outcome. The patient developed hypotension, respiratory arrest and disseminated intravascular coagulopathy following the procedure but with aggressive management she made a full recovery without any neurological or long-term sequelae.

BACKGROUND

As second trimester amniotic fluid embolism (AFE) is exceptionally uncommon, it may be easily missed as a potential diagnosis. This case is very important because it demonstrates that with quick recognition, immediate transfer to an appropriate unit and aggressive multidisciplinary management, a rare condition such as AFE, with a high incidence of morbidity and mortality, can be efficiently managed to achieve an exceptionally good outcome.

CASE PRESENTATION

A 20-year-old para 0+1 Caucasian woman presented to a private clinic for termination of pregnancy at 17 weeks and 6 days gestation. She opted for dilation and evacuation. The pregnancy was unplanned and uncomplicated, and the decision for termination was under Grounds C. The patient had no significant medical history. Cervical preparation was with 400 µg of misoprostol. propofol and alfentanil were used for anaesthetic induction. The procedure itself was uncomplicated, with 100 mL blood loss. Immediately postoperatively, the patient became hypotensive and hypoxic, with ST elevation and fast atrial fibrillation changes on her ECG. She remained intubated because she was also in respiratory arrest, and was transferred promptly to our hospital, the nearest district hospital.

Arterial blood gas results on arrival are outlined in [table 1](#). There was significant vaginal bleeding noted on the inco-sheets, which, when measured, was estimated to be about 800 mL. Ten units of Syntocinon followed by an infusion of 40 units in 500 mL of normal saline and 1 g of tranexamic acid were given. On examination, the patient's abdomen was soft and her uterus was well

contracted to about 10-week size. On vaginal examination, large blood clots were noted in the vagina, which were removed, revealing that the cervical os was closed and there was no active bleeding at that time. A mini abdominal scan was performed, which revealed that the uterus was empty, and did not show any free fluid in the abdomen. Bleeding was, however, noted from the endotracheal tube, which was still in situ.

Initial blood results included haemoglobin of 99 g/dL and an APTT of 77.2 ([table 2](#)). The rest of the coagulation screen taken on arrival could not be analysed as the blood sample failed to clot; this confirmed suspected DIC (disseminated intravascular coagulopathy).

Following aggressive management by the A&E department, Anaesthetic and Gynaecology teams, the patient was transferred promptly to intensive therapy unit (ITU), where vaginal bleeding was noted again, as well as bleeding from the endotracheal tube and from venipuncture sites. Aggressive replacement of blood products with the active involvement of the haematology team, due to deranged clotting results, included 4 units of cryoprecipitate, 4 units of fresh frozen plasma, 2 g of fibrinogen, 1 pool platelets and 2 units of packed red cells.

Subsequent blood results are outlined in [tables 3](#) and [4](#).

The total measured blood loss following replacement of blood products and control of the bleeding was 2 L. The patient improved quickly and her clotting normalised within a few hours. The following day, she was extubated and, following an exceptional recovery with no obvious sequelae, she was discharged home the next day. She was given a depot injection for contraception prior to discharge. The final multidisciplinary diagnosis was AFE, as the sequence of events and clinical features were strongly in keeping with this rare condition.

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Table 1 Blood gas result on arrival to hospital

pH	7.27
PO ₂	16
PO ₂	6
Lactate	3.45

PO₂, partial pressure of oxygen.

Table 2 Initial blood results; the remaining coagulation screen could not be analysed as the blood sample failed to clot

Hb	99
HCT	0.304
WCC	25
Sodium	135
Potassium	3.8
Urea	4.4
Creatinine	72
CRP	<5
APTT	77.2

APTT, activated partial thromboplastin time; CRP, C reactive protein; Hb, haemoglobin; HCT, haematocrit; WCC, white cell count.

TREATMENT

- ▶ Blood products
- ▶ Supportive treatment
- ▶ Appropriate ventilatory support

OUTCOME AND FOLLOW-UP

The patient was stepped down from ITU the next day and had a full recovery.

DISCUSSION

AFE is a rare and serious complication occurring only in pregnancy. The most recent UK Obstetric Surveillance System (UKOSS) report between 2005 and 2014 found that, in the UK, the incidence is 1.7/100 000 maternities and the estimated fatal incidence is 0.3/100 000 maternities. During the study period, 23 women died due to AFE.¹ In the surgical treatment of second trimester terminations or miscarriages, the incidence is even rarer, with around 10 cases reported in the literature.

AFE was first reported in 1926, though not widely acknowledged until 1941, when a series of postmortems of eight women found fetal squamous cells in maternal pulmonary vessels. These women had died from sudden shock in labour.²⁻³

The presentation of AFE varies from mild organ dysfunction to cardiopulmonary collapse, DIC and death. In our case, the initial presentation was hypotension and respiratory arrest followed quickly by DIC. Amniotic fluid, fetal cells or other debris enter the maternal circulation through ruptured membranes, endocervical veins, uterine trauma or the placental site.⁴ It is thought that the pressure gradient and small tears in the lower segment and endocervix are the most common sites. In the maternal circulation, it can cause an inflammatory or anaphylactoid response with cardiopulmonary collapse.⁴⁻⁵

Most cases of AFE, around 70%, occur during labour, followed by 19% in caesarean section and 11% after vaginal

Table 4 Subsequent coagulation screen results

Prothrombin time	12.7	10.4	10.4	10.7
INR	1.2	1	1	1
Fibrinogen	1.21	2.14	1.81	2.11
APTT	35.6	25	25.9	26.8

APTT, activated partial thromboplastin time; INR, international normalised ratio.

delivery. Rarely, as in the case described, it can occur after second trimester terminations, amniocentesis and abdominal trauma.⁵

AFE is associated with older maternal age, multiple pregnancy, placenta praevia and induction of labour. Postnatal AFE is associated with instrumental and caesarean deliveries.¹

The pathophysiology is not well understood. Historically, it was thought to be due to fetal cells and amniotic fluid entering the pulmonary circulation, causing vasospasm, heart failure and hypoxia. In 1995, Clark suggested that AFE is caused by fetal antigens in the amniotic fluid stimulating a cascade of endogenous immune mediators causing an anaphylactoid reaction. Biochemical mediators initially cause pulmonary vasospasm, followed by pulmonary hypertension and right heart failure, which subsequently leads to hypoxia and hypotension. The next phase is left ventricular failure, pulmonary oedema and DIC triggered by biochemical mediators. The mechanical obstruction by debris has minor effects.⁵

AFE is a diagnosis of exclusion, the presence of fetal squamous cells in pulmonary vessels at postmortem was previously thought to be diagnostic, however, fetal cells can be found in the circulation of labouring women who do not develop AFE.⁶ It is now suggested that it should be called anaphylactic syndrome of pregnancy, as the physiology suggests an inflammatory process.³⁻⁵⁻⁷

Non-specific tests include low haemoglobin, coagulopathy, hypoxaemia and hypercapnia. These results were demonstrated in our initial arterial blood gas and blood results, except for hypoxia (which was because the patient remained intubated and ventilated). In AFE, the chest X-ray may show cardiomegaly and pulmonary oedema, and although this was not present in our case, there was left lower lobe collapse and some consolidation seen on the patient's chest X-ray. ECG may show right heart strain and rhythm abnormalities, and there were ECG abnormalities in our case, including fast atrial fibrillation. More specific tests include pulmonary blood sampling, finding squamous cells covered in neutrophils and the presence of fetal debris. Raised levels of Sialyl Tn antigen and Zinc coproporphyrin are also features seen in AFE, as well as normal or raised serum tryptase. The serum tryptase level was normal in this case but the other more specific tests were not carried out in our case as the patient made a rapid recovery and the likely diagnosis was clear.⁵

The treatment is supportive. The airway should be rapidly controlled and hypoxaemia should be prevented or corrected. Haemodynamic stability should be achieved by aggressive resuscitative measures to ensure adequate tissue perfusion. Haemorrhage should be treated early and coagulopathies corrected with blood products. Uterotonics should be used in cases of atony and, if not controlled, a hysterectomy should be considered. In our case, DIC quickly ensued and was aggressively treated with blood products achieving quick and full recovery.⁸

CONCLUSION

AFE is a very rare complication of second trimester termination or miscarriage. The mortality rate of AFE historically was

Table 3 Subsequent blood results

Hb	87	85	91
HCT	0.259	0.253	0.274
WCC	13.1	11.3	10.6
Sodium	138	138	139
Potassium	3.3	3.6	3.8
Urea	4.6	4.2	3.6
Creatinine	86	80	71
CRP		12.9	
Serum tryptase			3.4

CRP, C reactive protein; Hb, haemoglobin; HCT, haematocrit; WCC, white cell count.

86%,⁹ however, in recent years, this has improved to 19%, although 7% of survivors had permanent neurological damage.¹ Our case had an exceptionally fast recovery uncomplicated by end organ damage, neurological sequelae or long-term complications. The importance of rapid recognition, appropriate initial resuscitation and speedy transfer to the nearest hospital able to provide high level multidisciplinary management cannot be overemphasised. Although there were 23 deaths due to AFE reported in the most recent UKOSS study between 2005 and 2014, overall there has been a reduction in mortality rates, no doubt due to prompt and highly skilled multidisciplinary input,

as demonstrated in our case. AFE is particularly rare in the second trimester, however, with high clinical suspicion and appropriate management the outcome can sometimes be positive, as in this case.

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Learning points

- ▶ Presentation of amniotic fluid embolism can vary, and although it is a rare complication, it should be considered as a differential diagnosis for a patient presenting with cardiorespiratory arrest and coagulopathy.
- ▶ Prompt recognition and appropriate management is vital in obtaining the best possible outcome.
- ▶ An aggressive multidisciplinary approach can reduce morbidity and mortality.
- ▶ Successfully managed cases should be reported to increase awareness among clinicians that such cases if managed appropriately can have a good outcome.

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