Amniotic Fluid Embolism

Pete Smith
Westmead Hospital
Which risk factors were present for AFE?

- Placenta praevia
- Antepartum haemorrhage/abruption
- Caesarean Section

- Also included (but not present here) – advanced maternal age, medical induction, but of note, no risk factors are particularly good at predicting risk of suffering an AFE; some studies report strong associations; these are frequently contradicted.

Clark Obstet. Gynecol. 123 (2) 1 p337
Clinical Presentation of AFE a la Westmead

- Blood pressure – 40/15 per arterial line
- Absent capnograph
- Chaotic ECG trace
- Absent pulses
- Rapidly declining SpO2

Other typical findings – cardiovascular collapse, dyspnea/hypoxia, bleeding, uterine atony. If awake – dyspnea, frothing from mouth, seizures, premonitory symptoms,
Resuscitation phase

- 18 minutes total resuscitation time.
- 7 mg adrenaline total
- Discussions made re – appropriateness of ECMO or bypass

Early anticipation of coagulopathy, based on assumption this was an AFE. Liaison with haematology on call and preparation for massive transfusion (of note – no MTP at that stage!)
Post resuscitation TOE
TOE loop - post resuscitation
## Coagulation profile

<table>
<thead>
<tr>
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<th>Preoperative</th>
<th>Post arrest</th>
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<tbody>
<tr>
<td>INR</td>
<td>1.0</td>
<td>&gt;10</td>
</tr>
<tr>
<td>PT</td>
<td>12 sec</td>
<td>&gt;150 sec</td>
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<tr>
<td>APTT</td>
<td>32 sec</td>
<td>&gt;150 sec</td>
</tr>
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Coagulopathy and ongoing haemorrhage

Expedited surgery

Multiple lines of attack in factor replacement – PCs, FFP, cryoprecipitate Prothrombinex, FVIII, FIX and eventually FVII

CVC sited, commencement of noradrenaline and dobutamine.

Thermal hygiene

Ongoing TOE interrogation of ventricular performance and pulmonary pressures
24 hours post arrest

- Further transfusion of packed cells and products in ICU
- Ventilator wean – decreasing FIO2 and PEEP requirements
- Extubated 21 hours after arrival in ICU, with apparently normal neurology
- Discharged home with baby day 6.
- Follow-up with O&G and respiratory physicians – all grossly OK
The Unholy Trinity

Hypoxia

Hypotension <-> Coagulopathy
Historical Aspects

Meyer - 1920s, and Steiner & Luschbaugh - 1940s emphasized mechanical occlusive phenomena by amniotic components.

This approach complicated by the presence of similar debris in non-AFE patients.
Subsequent developments

- Putative immunological mechanisms – but which ones?

- Clinically, AFE shares some features of anaphylaxis; results relating to tryptase and complement levels are inconsistent

Clark 2014 Obstet. Gynecol
Post mortem findings in AFE

- Foetal squamous cells, meconium and amniotic fluid elements have been isolated in the pulmonary vasculature of women who did not die from AFE.

- Histologic and immunohistochemical stains – including some specific to mucinous glycoproteins and meconium elements – are able to demonstrate the presence, but not the significance, of amniotic and foetal elements in maternal pulmonary circulation.
Incidence and mortality

- AMOSS quotes range 1:8,000 to 1:80,000.
- Published incidence varies worldwide, but attempts to standardize diagnostic criteria for research purposes have been made (Clark et al)
- Mortality rates likewise vary – due probably to differences in reporting rates, and diagnostic inclusion criteria (less severe cases will likely have lower death rates)
- Mortality from AFE is likely to be dropping.
AFE – subsequent pregnancies?

- Given a (survived) episode of AFE, what are the chances of recurrence in subsequent pregnancies?
- Given the rarity of the condition and the scarcity of survivors, sample numbers are very low, but multiple case reports of unaffected subsequent gestations exist.
- Clark (2014): “What is a woman’s risk of recurrent amniotic fluid embolism? We just do not know”. 
Other thoughts….

- Perhaps whatever causes widespread clotting/DIC is the primary event?
- This idea doesn’t change the need to search for mediators and triggers, and doesn’t change the basics of supportive management.
- It might give context to phenomena related to clinical presentation of AFE
Summary

- AFE is uncommon, but a leading cause of maternal mortality
- Prediction is difficult
- Diagnosis is clinical, and rapid identification is important
- Treatment is supportive
- Severe coagulopathy must be anticipated and treated early
- Teamwork is essential