Maternal cardiac arrest during emergency Caesarean section: A likely case of amniotic fluid embolism

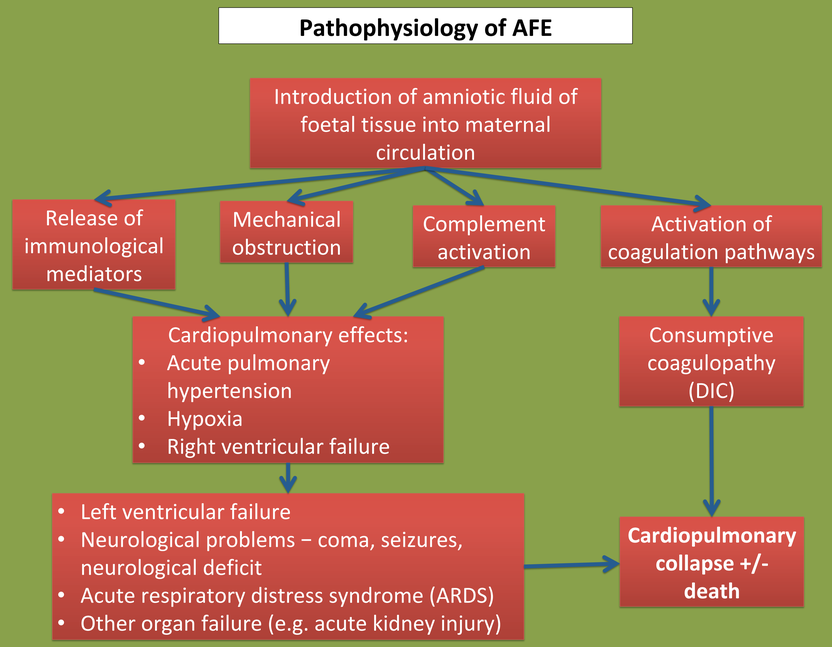
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Maternal cardiac arrest occurs very rarely in the developed world, with an incidence of 1:12,000-36,000 women per year[1]. We describe the case of an intra-operative cardiac arrest during an emergency Caesarean section (CS).

Case Report

- A 34-year old nulliparous woman with dichorionic diamniotic (DCDA) twins and pregnancy-induced hypertension (PIH) presented in the early hours of the morning due to an antepartum haemorrhage at home.
- She was taken to theatre for a category 1 CS for foetal distress.
- General anaesthesia was uneventful, and the infants were both delivered within 5 minutes. The patient then became pale, tachycardic (~150/min) and had a rapid decline in oxygen saturation and end-tidal CO2 over a few seconds. No central pulses were palpable, so cardiac arrest was declared and cardiopulmonary resuscitation (CPR) was commenced.
- After 1 minute the patient regained cardiac output without requiring adrenaline or defibrillation.
- The surgical findings were of placental abruption, so the major haemorrhage protocol was activated. The bleeding was eventually arrested and the intra-operative estimated blood loss was 4.2L. She was transfused four units of packed red cells, four units of fresh frozen plasma, platelets, cryoprecipitate and tranexamic acid.
- She was successfully extubated at the end of the procedure and went to the intensive care unit (ICU) post-op. Shortly after extubation she became hypertensive so was treated with Magnesium Sulphate for pre-eclampsia.
- CT imaging demonstrated acute liver infarcts, renal cortical necrosis and right middle and lower lobe segmental pulmonary emboli. Laboratory investigations were suggestive of disseminated intravascular coagulation (DIC) (thrombocytopaenia, prolonged prothrombin time and low fibrinogen) and acute hepatic failure. She developed anuric acute kidney injury and required haemofiltration during the seven days spent on ICU.
- She was subsequently transferred to a local renal unit for ongoing dialysis, which she required for a further 2 weeks before being discharged home.

Pathophysiology of AFE



Discussion

Rapid recognition of cardiac arrest and prompt multidisciplinary management contributed to an ultimately positive clinical outcome for this patient.

While it's possible the aetiology of this patient's cardiac arrest was simply hypovolaemia secondary to major haemorrhage, the rapid loss of cardiac output and development of pulseless electrical activity raises a high suspicion of amniotic fluid embolus (AFE). This is also supported by the subsequent development of DIC and rapid progression to multi-organ failure, which is unlikely to be explained by the brief cardiac arrest and hypoperfusion alone.

AFE is a rare but life-threatening condition involving a profound systemic inflammatory response following the introduction of foetal cells into the maternal circulation and can present as consumptive coagulopathy and cardiovascular collapse. Risk factors in this patient include delivery by CS, multiple pregnancy, PET and abruption.[1,2] Diagnosis is largely clinical and one of exclusion, although cytological analysis of bronchoalveolar washings or central venous blood may demonstrate foetal cells or cell debris, such as mucin or hair.

Acknowledgements:
Many thanks to the patient who gave her written consent to present her case

References:
1. Metodiev Y, Ramasamy P, Tuffnell D. Amniotic fluid embolism. BJA Education 2018, 18(8): 234-238.
2. Fitzpatrick KE, Tuffnell D, Kurinczuk JJ, et al. Incidence, risk factors, management and outcomes of amniotic-fluid embolism: a population-based cohort and nested case–control study. BJOG 2016; 123: 100–109.