SUDDEN CARDIAC ARREST DURING CESAREAN SECTION

- A Possible Case of Amniotic Fluid Embolism -

- Case Report -

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Abstract

Amniotic Fluid Embolism (AFE) is a rare obstetric catastrophe that occurs in approximately 1/50,000 pregnancies and has a mortality rate in excess of 80%. AFE is a condition that is poorly understood and often difficult to diagnose. We report a case of a healthy 27 yr-old gravid two, 35 wk gestation parturient with a previous Cesarean section two years previously, and presently admitted for emergent Cesarean section due to premature uterine contractions.

Induction of general anesthesias was performed with no problem and a male preterm infant with Apgar 8 at 1min was delivered. Amniotic fluid was bloody and 40% placental abruption existed. Following delivery of the placenta, patient suddenly became plethoric and O₂ saturation began to decrease and no pulse could be palpated! Immediate CPR was successful but she was hemodynamically unstable and signs of right heart strain was obvious. Right jugular venous catheterization was performed, vasopressors were administered. After a two hours period of relatively stable vital signs, patient’s reflexes returned to normal, however, profound coagulopathy on lab data was reported and she was treated with 10 unit Packed Red Blood Cells (PRBCs), 10 unit FFP and 8 unit platelets, Sodium bicarbonate, oxytocin and Methergine. The patient remained hemodynamically unstable while laparotomy-hysterectomy was performed to stop the bleeding. Unfortunately attempts were unsuccessful and patient died four hours later in ICU. Post-mortem findings showed signs of Disseminated Intravascular Coagulation (DIC), no fetal squamous cells in pulmonary vasculature were found and special staining of Cytokeratin marker shows no positive cells in lumen of vessels.

The post-mortem diagnosis of AFE is challenging to forensic investigators and pathologists and can be confirmed by histological confirmation of amniotic fluid contents in the pulmonary vasculature, although they may be difficult to identify. In recent years it has been suggested that AFE is an anaphylactoid reaction to fetal antigens and an elevated serum tryptase level is increasingly being used to support the diagnosis.

Sudden onset of cardiovascular collapse and early signs of right heart strain and fulminant DIC supports the diagnosis of AFE in this case, although no fetal debri could be find in pathologic staining.

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Right jugular venous catheterization was performed and infusion of dopamine 5μg/kg/min and epinephrine 5 μg/min was started. She had relatively stable vital signs for two hours and her reflexes (cough, spontaneous ventilation, pain withdrawal…) returned to normal. However, profuse vaginal bleeding was observed and patient’s systolic blood pressure fell to 40 mmHg. Lab data reported metabolic acidosis and profound coagulopathy on lab data were reported (PT >2 min & PTT >3 min). She was treated with 10 units of Packed Red Blood Cells (PRBCs), 10 unit FFPs and 8 unit platelets, sodium bicarbonate, oxytocin and methergine. The patient remained hemodynamically unstable, in the meantime laparotomy-hysterectomy was performed to stop the bleeding. Unfortunately attempts were unsuccessful and patient died four hours later in ICU.

Post-mortem findings showed signs of Disseminated Intravascular Coagulation (DIC), no fetal squamous cells in pulmonary vasculature were found and special staining of Cytokeratin marker showed no positive cells in lumen of vessels.

Discussion

Amniotic Fluid embolism (AFE) has been reported during pregnancy, labour, Cesarean section and the postpartum period. While the syndrome remains poorly understood, it can be described as a two-stage process. In the first stage, amniotic fluid and fetal cells enter the maternal circulation, triggering the release of several endogenous mediators. Pulmonary artery vasospasm and pulmonary hypertension lead to elevated right ventricular pressure, and the resultant hypoxia causing myocardial and pulmonary capillary damage5. Approximately half of all patients who survive enter a second stage characterized by hemorrhage and DIC, possibly because amniotic fluid contains Thromboplastin6.

The post-mortem diagnosis of AFE is challenging to forensic investigators and pathologists and can be confirmed by histological confirmation of amniotic fluid contents in the pulmonary vasculature of the mother, although difficult to identify as some contests are lodged in small pulmonary capillaries. Multiple lung sections are submitted when diagnosing AFE which increases the probability of finding elements of
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In recent years it has been suggested that AFE is an anaphylactoid reaction to fetal antigens and an elevated serum Tryptase level is increasingly being used to support the diagnosis. In the absence of a definitive diagnostic test, Benson proposed a clinically based definition of AFE. According to his criteria, a diagnosis of AFE would be appropriate for any patient experiencing sudden onset of cardiovascular collapse during pregnancy or 48 hours postpartum. Other illnesses ought to be ruled out that might explain the signs and symptoms.

The sudden onset of cardiovascular collapse in this case preceded by plethoric discoloration of the patients face suggests anaphylactoid reactions, and early signs of right heart strain followed by progressive fulminant DIC, supports the diagnosis of AFE, though no fetal debri could be found in the pathologic staining of pulmonary vasculature.

References