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CASE REPORTS

AMNIOTIC FLUID EMBOLISM

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A 37 year old multi-para was admitted to Castle Street Hospital for Women with reduced foetal movements and lower abdominal pain in her 37th week of gestation. She was not a registered patient and was admitted as a casualty. All her previous pregnancies were normal and she had normal vaginal deliveries. Initial general and cardio vascular examination findings were normal and Cardio Tochogram (CTG) was reactive.

90 minutes later she complained of reduced foetal movements and the CTG showed a deceleration pattern. Artificial Rupture of Membrane was done, liquor was seen to be clear and she was taken to the labour room and labour was augmented with 4 units of syntocinon. One hour later she delivered a live non-asphyxiated baby boy vaginally.

Soon after the delivery she complained of sudden onset difficulty in breathing and became drowsy. At that time her blood pressure was 60/40 mmHg and with a heart rate of 120/minute. Anaesthetic registrar was called for help. On examination she was tachypnoeic, with cold and clammy peripheries, the pulse rate was of 120/min and blood pressure was 60/40mmHg. Immediately a large-bore IV cannula was inserted and rapid saline infusion commenced. Her SpO2 showed 85%, and endo-tracheal intubation was done, and she was taken to the Intensive Care Unit.

She was ventilated with a small dose of midazolam and suxamethonium. She had bleeding per vagina and from the puncture sites. Therefore, she was given blood, fresh frozen plasma and platelets. In-spite of fluid replacement her blood pressure was less than 80mmHg, therefore a dopamine infusion was started at 10µg/Kg/min. She had an episode of bradycardia with a heart rate of less than 40/minute. Then she was given an adrenaline bolus followed by an adrenaline infusion.

Five hours after delivery she had a cardiac arrest, and cardio pulmonary resusitation was performed for more than one hour. She died in-spite of maximum effort. A post-mortem was performed which revealed the presence of foetal hair follicles in the maternal pulmonary bed and histology revealed the presence of foetal cells.

Discussion

Amniotic fluid embolism (AFE) is a rare obstetric emergency in which amniotic fluid, foetal cells, hair or other debris enter maternal circulation causing cardio-vascular collapse. In 1941[8] . Steiner and Luschbaugh described AFE for the first time after they found foetal cells in the pulmonary circulation of a woman who died during labour.

Current data from the National Amniotic fluid registry suggest that the process is similar to anaphylaxis than to embolism and suggest a new term "ANAPHYLACTOID SYNDROME OF PREGNANCY" [1].

Patho-physiology:

The patho-physiology is poorly understood. The amniotic fluid and foetal cells enter maternal circulation, possibly triggering an anaphylactic reaction to foetal antigen. Benson et al tested two hypothesis concerning patho-physiology of AFE[6]:

(1) Clinical symptoms resulting from mast cell degranulation with release of histamine and tryptase.
(2) Clinical symptoms resulting from activation of complement pathway.

Two phases have been described. In phase I, there is pulmonary artery vasospasm, pulmonary hypertension and elevated right ventricular pressure causing hypoxia. This can result in myocardial capillary and pulmonary capillary damage resulting in left heart failure and Adult Respiratory Distress Syndrome (ARDS). Phase II, is a haemorrhagic phase characterised by massive haemorrrhage with uterine atony and Disseminated Intravascular Coaggulation (DIC). Fatal consumption coagulopathy also could be a feature [5].

Incidence, morbidity and mortality:
Incidence is 1:8000 to 80,000 of all pregnancies. But there could be sub-clinical manifestations which would have not been detected [1].
There is no racial or ethnic predilection, Risk factors involved are advanced maternal age, multi-parity, cervical laceration, intra-uterine foetal death, short or precipitated labour, placenta accreta, polyhydramnios, chorioamnionitis and use of oxytocin [3].

AFE could be seen after abortion, abdominal trauma and amniocentesis.
Mortality rate can be as high as 80%. 50% of mothers die within one hour of onset of symptoms. Survivors can have neurological impairment [4].

Clinical presentations:
Common presentation is acute onset of cough, shortness of breath and hypotension. Patient might experience convulsions and massive haemorrhage. This can result from cardiac arrest and death.

Hypotension manifests with loss of diastolic measurement. Tonic-clonic seizures are mostly scen. As hypoxia progress cyanosis could be noticed [8].

Differential diagnosis [6, 8]
Anaphylactic reactions.
Eclampsia.
Local anaesthetic toxicity.
Placental abruption.
Pulmonary thrombo embolism.
Uterine rupture.
Aortic dissection.

Investigations [1, 8].
Arterial blood gas: acidosis, hypoxia, hypercarbia and increased base excess.
Full blood count: low Hb, low platelet count and increased white cells.
Coagulation studies: low fibrinogen, increased APTT and elevated FDP.
Imaging: CXR may be normal, an effusion or cardiomegaly can be present.
ECG: sinus tachycardia, right ventricular enlargement.
2D ECHO: right ventricular enlargement with right atrial / right ventricular dilatation.

Procedures:
Arterial cannulation is ideal for blood pressure measurement and frequent blood gas analysis.
Pulmonary artery floatation catheter is indicated to monitor cardiac output, wedge pressure and tissue oxygenation.

Management:

Medical management:
Oxygen administration may require endo-tracheal intubation.
Cardio-vascular resuscitation is needed in case of a collapse.
Hypotension is treated with crystalloids, colloids or blood.
Coagulopathy is corrected with FFP, platelets and cryoprecipitate. Lim and Colleagues have treated coaggulopathy with activated factor VII [2].
Vasopressors or sympatho-mimetic agents that can be used are dopamine, dobutamine and adrenaline since anaphylaxis is considered as the pathology involved [1].
To improve the uterine contractions oxytocin, and prostaglandin analogues are used.
Other manoeuvres like uterine massage, uterine packing are also used.
Corticosteroid therapy may be beneficial since immune mediated reactions are involved. Large doses of hydrocortisone 500mg 4-6 hourly [7, 8] may be required.

Surgical management:
Emergency caesarean section is required.
If there is severe bleeding not responding to medical management it might require hysterectomy if the other parameters are satisfactory [3].
Use of proton pump inhibitors or histamine II receptor blockers, sedation and analgesia, proper nursing care, nutritional support, regular use of bronchodilators, chest and limb physiotherapy are other measures taken during the management.

Medico-legal pit falls [1, 8] are:
Failure to respond to an emergency.
Failure to stabilise the patient.
Delayed recognition after abortion and amniocentesis.
Failure to perform and abdominal delivery five minutes after unsuccessful Cardio Pulmonary Resuscitation (CPR).

Summary:
Amniotic fluid embolism is a sudden onset life threatening complication during pregnancy which involves a series of pathologies. Diagnosis is essentially clinical with a high degree of suspicion. It involves the cardio-vascular, respiratory system, haematological and central nervous systems.
The treatment is mainly supportive and requires aggressive intensive care management. In established patients overall prognosis is poor.

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