
C A S E R E P O R T

Amniotic Fluid Embolism understanding pathophysiology from a successfully managed case

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Abstract:

Amniotic fluid embolism is a rare emergency in obstetrics with high mortality rate. Here we report a case of amniotic fluid embolism managed successfully.

Keyword: Amniotic Fluid Embolism

Introduction

Amniotic fluid embolism is a rare but often fatal obstetric complication caused by sudden infusion of "abnormal" amniotic fluid into the maternal circulation. Entry of amniotic fluid into the maternal circulation was reported way back by Meyer in 1926 and described by Steiner and Lush Baugh in 1941 as a distinct clinical entity.^{1,2} The incidence of amniotic fluid embolism averages 1:20,000 to 1 in 80,000 with mortality of 86%.^{1,2} Most women who survive the incident have permanent neurological impairment. The neonatal survival is 70%. There is no evidence to indicate that survivors are at risk for amniotic fluid embolism during future pregnancies.^{1,2}

Case report

A 35 years G₄ P₂₊₁ was admitted on 25/12/061 (7th April 2005) at 6.30 AM in early stage of labour at Kathmandu Medical College Teaching Hospital (KMCTH), Sinamangal. At 9.50 AM patient was in active stage of labour. Artificial rupture of membrane was done, which showed thick meconium stained liquor. She was planned for emergency LSCS for the indication of thick meconium stained liquor with big baby. At 10:10 AM, she was shifted to OT and pre-anaesthetic evaluation was done. All of a sudden the patient became rigid, cyanosed, there was sweating over forehead, and pulse and blood pressure were not recordable. Immediate airway management was done with bagging and masking followed by forceful ventilation, finally with

successful intubation. Emergency LSCS was performed at 10.20 AM, and a live male baby with Apgar score of 2/10 at 1 minute, 4/10 at 5 minute was delivered weighing 3740gms. Immediate postoperative blood pressure was 90/60, heart rate was 180 per minute, chest was full of crepitations and there was massive per vaginal bleeding and oozing from abdominal wound incision. Abdomen was closed with haemostatic suture. The patient was shifted to ICU where she was kept on ventilator with dopamine drip.

The baby developed severe birth asphyxia, and postnatal seizures but recovered and was discharged on the 10th day. Follow up at 2 years of age showed no signs of neurological deficit.

Pathophysiology

Amniotic fluid embolism usually occurs during labour but has occurred during abortion, abdominal trauma and amnioinfusion. The pathophysiology of amniotic fluid embolism is poorly understood and is believed to be from the entry of the amniotic fluid and fetal cells into the maternal circulation, triggering a 2-phase process. In phase I, pulmonary artery vasospasm with pulmonary hypertension and elevated right ventricular pressure will cause hypoxia. Hypoxia causes myocardial and pulmonary capillary damage, the left heart fails, and acute respiratory distress syndrome develops. Women who survive the above events may enter phase II. This is a haemorrhagic phase characterized by massive haemorrhage with uterine

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Table 1: Management of the patient in tabular form

Day	Condition of the patient	Significant Investigation	Treatment
0	critical, bleeding from CVP line, incision wound and vagina	PT test 46 sec control 14sec INR 4.2 APTT test 54 sec control 33 sec	Ventilator, dopamine, 6 pints of fresh frozen plasma, 4 pints of whole fresh blood, IV antibiotics, carboprost 3 total doses, methergin 1 ampule IV 6 hourly
1	critical, consciousness gained but restless, chest full of crepitation, no active bleeding from any side. uterus contracted	WBC 17,9 00BT 15min CT 10 min APTT test 40sec blood C/S no growth WBC 19,600/mm ³ , platelets 51,000/mm ³	3 pint of Fresh frozen plasma was given, Injection Dopamin stopped, Methergin 6 hourly
2	improved, BP maintained without inotropic support, chest bilateral crepitation +, No vaginal bleeding	Platelets 1,32,000/mm ³ APTT test 38 sec control 33 sec PT test 20 sec control 14 sec INR 1.47	Ventilator, IV fluids and IV antibiotics
3	improved		Ventilator, IV fluids and IV antibiotics
4	satisfactory		Ventilator off

atony and disseminated intravascular coagulation (DIC); however, fatal consumptive coagulopathy may be the initial presentation.

Clinical features

- Advanced maternal age, thick meconium stained liquor and male fetus are associated factors.
- Hypotension: blood pressure may drop significantly with loss of diastolic measurement.
- Dyspnea: Labored breathing and tachypnea may occur.
- Seizure: The patient may experience tonic-clonic seizures.
- Cough: this is usually a manifestation of dyspnea.
- Cyanosis: As hypoxia /hypoxemia progresses, circumoral and peripheral cyanosis and changes in mucous membranes may manifest.
- Fetal bradycardia: In response to the hypoxic insult, fetal heart rate may drop to less than 110 beats per minute (bpm). If this drop lasts for 10 minutes or more, it is a bradycardia. A rate of 60 bpm or less over 3-5 minutes may indicate a terminal bradycardia.
- Pulmonary edema: This is usually identified on chest film.
- Cardiac arrest
- Uterine atony: Uterine atony usually results in excessive bleeding after delivery. Failure of the uterus to become firm with bimanual massage is diagnostic.

Diagnosis

- No single laboratory finding by which amniotic fluid embolism can be diagnosed.
- Chest X-ray is not specific
- Ventilation perfusion scan aid in the diagnosis
- ECG may show right ventricular strain
- Echocardiography confirms severe left ventricular failure
- Sampling from the right side of heart if sedimented will show fetal squamous cells, vernix, lanugo and bile containing mucin
- Various blood coagulation test may be deranged

Monitoring and treatment of amniotic fluid embolism: a suggested approach

1. Intubate and ventilate with 100% O₂ and maintain positive end expiratory pressure (PEEP).
2. If there is no pulse, start external cardiac massage.
3. Insert two large-bore intravenous cannulas, a pulmonary artery catheter, a bladder catheter, and if possible an arterial line.
4. Monitor oxygen saturation, ECG and heart rate, pulmonary and systemic blood pressures, cardiac indices, and neurologic function.
5. Aspirate blood from the right side of the heart for pathologic examination.
6. Draw blood for baseline coagulation studies, cross matching, and arterial blood analysis. Notify

the blood bank of the diagnosis and the probable need for red blood cells, fresh frozen plasma and platelets.

7. Administer NaHCO_3 to correct acidosis.
8. Fetus and placenta should be delivered as soon as feasible within 4 min.
9. Administer sympathomimetic drugs to treat left ventricular failure and augment cardiac output and peripheral perfusion.
Dopamine 2 – 5 $\mu\text{g}/\text{kg}/\text{min}$
Dobutamine 15 – 30 $\mu\text{g}/\text{kg}/\text{min}$
Isoproterenol 0.05–0.10 $\mu\text{g}/\text{kg}/\text{min}$
Norepinephrine/epinephrine
0.1 – 0.4 $\mu\text{g}/\text{kg}/\text{min}$ or
0.15 – 0.30 $\mu\text{g}/\text{kg}/\text{min}$
10. If CVP is rising, digoxin (0.5 mg) or deslanoside (0.8 mg) and furosemide (10–40 mg) may be administered intravenously.
11. Administer hydrocortisone in 1gm intravenous boluses every 6 hr for 48 hr.
12. It is desirable to manage the patient in a multidisciplinary intensive care unit.
13. Maintain vascular volume by infusing crystalloid. Treat disseminated intravascular coagulation (DIC) and bleeding with red blood cells, fresh frozen plasma and platelets, depending on the patient's clinical condition and serial coagulation results.

Differential diagnosis

1. Other types of pulmonary embolism
2. Aspiration pneumonitis
3. Toxicity caused by local anaesthetics
4. Eclampsia
5. Intracranial haemorrhage
6. Haemorrhagic shock
7. Acute heart failure

Discussion

This case of amniotic fluid embolism is a rare emergency obstetric event who had a marvellous recovery, leaving us enriched with a lifetime experience; this being possible with immediate management in OT itself with the help of the anaesthetist and surgical team..

Following amniotic fluid embolism, treatment of DIC was directed at blood volume replacement and circulatory support with the expeditious delivery of fetus and placenta. DIC is self-limiting within 24 hr after the uterus has been emptied with the removal of initiating stimulus (amniotic fluid, placental thromboplastin, or both). Levels of coagulation factors are usually adequate for hemostasis, even if agents specifically directed at correction of abnormal coagulation are not administered.^{1,2}

Although both crystalloids and colloids can restore blood volume, in ongoing hemorrhage, transfusion of

packed red blood cells is necessary to restore oxygen carrying capacity. Stored blood is deficient in factor V, VIII, and platelets. These deficiencies can be overcome by giving fresh frozen plasma and platelets. When possible, single-donor platelets and fresh frozen plasma collected by apheresis techniques should be given. Both are better blood components, and because the patient is exposed to single rather than multiple donors, the risks of transmitting infectious agents are reduced.^{1,2}

The outcome of DIC is determined by dynamic interactions between pathologic depletion and compensatory repletion of coagulation factors and platelets. Serial laboratory tests used to guide ongoing therapy must be interpreted while keeping the clinical findings in mind.

Although mentioned in previous reports, transfusion of fibrinogen is usually unnecessary, and administration of heparin or inhibitors of fibrinolysis may be dangerous. Although heparin has anti-thrombin effects, no controlled studies have shown heparin to be valuable in treating DIC from amniotic fluid embolism. If bleeding in DIC is due to more anticoagulant breakdown products of fibrinogen than to depletion of clotting factors, the surge of fibrinogen may precipitate generalized clotting. Likewise, antifibrinolytic agents, which block the thrombin-limiting action of plasmin, may also produce generalized thrombosis.^{1,2}

Massage of the uterus, administration of oxytocics and prostaglandin $\text{F}_{2\alpha}$, and uterine packing can be attempted to increase uterine tone. However, uterine atony is resistant to most therapy and is terminated only with restoration of circulatory status.^{1,2}

Possible late sequelae after amniotic fluid embolism and DIC include (a) renal failure caused by cortical necrosis; (b) neurologic deficits and convulsions usually of central origin; (c) myocardial ischemic injury and infarction; and (d) liver damage.^{1,2}

To sum up, the pathogenesis of AFE in humans remains unclear, as the similar kind of pathophysiological and clinical alterations has not been accurately reproduced in animal models studies. However, the proposed explanation is the recognition of the fetal cells (antigens) entering the maternal pool in susceptible mothers bypassing the maternal immunological barrier subsequently triggering the maternal immune system to release endogenous mediators that are responsible for dramatic pathophysiological disturbances including the initiation of DIC. AFE is seen to involve more of anaphylactic shock and less of an embolic phenomenon; hence, "Anaphylactoid syndrome of pregnancy" seems better and appropriate terminology, as has been suggested.³ Patients surviving the initial insult are equally at high risk for multiple organ failure. Although successful pregnancy and birth have been

recorded following the survival from AFE indicating AFE to be a sporadic event.⁴

AFE has occurred during second trimester induction of abortion for an intrauterine dead fetus.⁵ It has also complicated a twin in preterm labor.⁶ The mortality from AFE remains high and fascial devices has been implicated to control intraoperative bleeding.⁷ At present, there are no therapy that has been found to consistently improve the outcomes in women with AFE. Yet there are few non-fatal case of amniotic fluid embolism with survival, those that have been discovered during a routine caesarean section.^{7,8} This one being as presentable example.

Conclusion

Although the mortality is still very high, maternal death from amniotic fluid embolism need not be inevitable in every case, survival improved with the prompt recognition and appropriate treatment of the syndrome.

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