Case Report

Amniotic Fluid Embolism during Emergency Hysterotomy

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Abstract

The most common obstetric surgeries performed in India are cesarean section. Unwillingness to undergo normal delivery, unfavorable anatomy, previous cesarean section, and pregnancy-induced hypertension are common indications to name. Although pregnancy is a natural process, many physiological changes occur in maternal health. Pregnant lady is at risk to develop pregnancy-related complications. Amniotic fluid embolism is one of the life-threatening complications that is seen during delivery.

Key words: Amniotic fluid, anesthesia, cardiac arrest, cesarean section embolism, pulmonary edema

Introduction

Entry of amniotic fluid containing fetal cells, hair, bile-stained meconium, and or fetal gut mucin into the maternal circulation is a rare catastrophic obstetric emergency. It may present as a sudden maternal collapse which is commonly associated with hypoxemia, hypotension, pulmonary edema, or coagulopathy. The mode of entry is via ruptured membranes or ruptured uterine or cervical vessels down a pressure gradient. Portal of entry is either placental implantation site or more commonly thought to be through small tears in lower uterine segment or endocervix. For the first time, it was reported by R Meyer in 1926.[1] In 1941, based on clinical and pathological findings, Steiner and Lushbaugh reported amniotic fluid embolism (AFE).[2]

The incidence of AFE ranges between 1 in 8000 and 1 in 80,000 pregnancies, with various estimates between these two extremes.[3] Maternal outcome following AFE is not promising. Maternal mortality ranges from 44% to 61% depending on the severity of disease.[4,5]

The exact pathophysiology of AFE is unclear, but multiple systems are affected and may be immune-mediated. Risk factors that may contribute for AFE include uterine hyperstimulation, advanced maternal age, multiparity, placenta previa, abruption, eclampsia, uterine rupture, instrumental vaginal delivery, cesarean section, and male neonate.

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Case Report

A 20-year-old primigravida with 29 weeks of gestation was referred from a private clinic with moderate pervaginal bleeding and associated with mild abdominal pain. General examination showed evidence of pre-eclampsia. Obstetrician found 250 g of clots on vaginal examination, suspected to be arising from placental abruption resulting in intrauterine death and made a decision for emergency hysterotomy.

On preoperative examination of the patient, we noted pulse rate (PR) of 150/min, blood pressure (BP) of 130/100 mmHg, respiratory rate of 26/min, and presence of pallor and pedal edema. We could not find any systemic abnormality. Laboratory reports revealed hemoglobin 13.9 g%, platelet count 1.53 lakhs/mm^3, International Normalized Ratio (INR) 1.13, and activated partial thromboplastin time (APTT) 29 s over the control of 30 s and urine albumin 3+. Both renal and liver function tests were within normal range. Placenta previa was ruled out by ultrasound examination.

We did choose general anesthesia in view of better hemodynamic stability. Ringer lactate (RL) infusion was continued through

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18 g intravenous catheter. The patient was premedicated with injection diazepam 5 mg and injection fentanyl 40 μg intravenous (IV). Preoxygenation was done for 3 min and induced with titrated dose of injection thiopentone 150 mg when eyelash reflex was abolished. Intubation was facilitated with injection vecuronium 0.8 mg/kg. Anesthesia was maintained with end-tidal concentration of 0.8% isoflurane and nitrous oxide 50% in 50% oxygen. The patient's lungs were mechanically ventilated. Intraoperatively, sufficient analgesia was provided by additional doses of fentanyl with a total dose of 100 mcg. A total of 1250 ml of RL was given during surgery, which lasted for 90 min. Hemodynamic parameters remained stable throughout surgery. Isoflurane was discontinued after the closure of abdomen, and residual neuromuscular block was antagonized with injection neostigmine 0.05 mg/kg and injection glycopyrrolate 0.008 mg/kg. The patient's spontaneous respiration was inadequate, so she was assisted with Bain's circuit with 100% O_2 and vitals were closely monitored. IV calcium gluconate 500 mg was given slowly over 10 min. Arterial blood gas (ABG) was done and it suggested severe metabolic acidosis with pH - 7.04, PO_2 - 72, PCO_2 - 47, HCO_3 - 12.7, and SO_2 - 84%.

Injection sodium bicarbonate 50 ml IV was stat administered and 50 ml in RL was slowly infused. Vitals were PR 130/min, BP 138/88 mmHg, and SpO_2 98% on 100% O_2 with ventilation on Bain's circuit. Her response was inadequate and she developed bubbling crepitation in lungs bilaterally. Pupils were mid-dilated with sluggish reaction to light. Urine output was 25 ml throughout the surgery. The patient was propped up and ventilation continued with Bain's circuit with positive end-expiratory pressure (PEEP). Titrated doses of injection lasix at a total of 80 mg, injection hydrocortisone 100 mg, and injection dexamethasone 8 mg were administered. IV fluid (RL) was continued at 60 ml/h. The patient remained unresponsive. Urine output was nil.

After about 45 min, she suddenly developed bradycardia with heart rate of 30/min, unrecordable BP, and SpO_2 decreased to 60% despite ventilation with 100% O_2. Immediately, cardiopulmonary resuscitation (CPR) was started with >100 chest compression per minute. Injection adrenaline 1 mg IV was repeated 4 times for every 5 min. CPR was continued. Injection dobutamine infusion at 10 μg/kg/min was started. There was pink frothy fluid on suctioning of endotracheal tube.

After 30 min, electrocardiography showed sinus rhythm with HR 110/min, BP 84/56 mmHg, and SpO_2 96%, but B/L crepitation persisted. ABG showed pH 6.84, PO_2 - 57, PCO_2 - 72, HCO_3 - 12.3, and SO_2 - 59%, despite mechanical ventilation with FiO_2 of 1. Injection sodium bicarbonate 50 ml IV and 50 ml in RL was repeated. Injection dobutamine was titrated and increased to 20 μg/kg/min. Four units of fresh frozen plasma (FFP) were administered.

Postoperative blood investigation revealed Hb 9 g%, platelet count 96,000 cells/mm^3, INR 1.81, and APTT 70 s over the control of 30 s, serum glutamic-oxaloacetic transaminase 945 and serum glutamic pyruvic transaminase 460.

On examination, PR was 130/min, BP was 104/52 mmHg, and SpO_2 was 94% RS B/L crepts were still present; pupils were mid-dilated, sluggishly reacting to light. The patient was shifted to Intensive Care Unit and continued mechanical ventilation continued with BIPAP mode with PEEP 14 mmHg, but her response was poor. Injection lasix 20 mg IV was repeated but urine output was nil.

After 1 h, again the patient's SpO_2 decreased to <60%, ABG showed pH 7.01, PO_2 - 25, PCO_2 - 47, HCO_3 - 11.9, and SO_2 - 20%. The patient developed sudden cardiac arrest after about 5 h of surgery from which she could not be revived in spite of all resuscitative measures.

Discussion

The classic triad of AFE is sudden cardiovascular collapse, dysponea, and coagulopathy.[6] Profund hypotension and fetal distress occur in almost all cases with pulmonary edema, acute respiratory distress, cyanosis, coagulopathy, and cardiopulmonary arrest occurring in 80–90% cases. Nonspecific symptoms such as nausea, vomiting, chest pain, paresthesia, impending sense of doom, and agitation may be seen waxing and waning up to 4 h before the cardiovascular collapse.

There will be only mild to moderate elevations in pulmonary artery pressures with associated evidence of severe left ventricular (LV) dysfunction. Based on the previous reports, a biphasic model was put forward. It explains the hemodynamic abnormalities that occur with amniotic fluid emboli.

According to this model, the first phase that lasts for about 30 min was due to a anaphylactoid reaction to fetal antigens resulting in the release of biochemical mediators leading to pulmonary vasospasm, pulmonary hypertension, and right ventricular (RV) dysfunction resulting in hypoxia, hypotension, and capillary damage. These biochemical mediators also trigger disseminated intravascular coagulation. In the second phase, it leads to uterine atony and massive hemorrhage. Pulmonary edema and LV failure are pathognomonic of AFE possibly due to myocardial ischemia or direct myocardial depressant effect of AFE is seen during the second phase.[7] Products of fetal tissue are responsible only for minor effect resulting in actual mechanical obstruction.[8]

Diagnosis of AFE is by exclusion criteria depending on clinical presentation rather than on histopathological findings. It is aided by nonspecific and specific diagnostic tests. Nonspecific tests are INR, prothrombin time, APTT, hypoxemia on ABG, RV strain pattern on electrocardiogram, decrease in C_3–C_4 complement levels, and demonstration of RV or LV dysfunction on ultrasound/transesophageal echocardiography. Diagnostic tests include cytological analysis of central venous blood and broncho-alveolar fluid, Sialyl-Tn antigen test, zinc coproporphyrin concentration, and serum

tryptase concentrations,.[9] and more recently, measurement of insulin-like growth factor binding protein.[10]

The key factors in the management are early recognition, oxygenation by intubation and ventilation, intravenous fluids, vasopressor, and inotropes. FFP, cryoprecipitate, platelets, and recombinant factor VII should be considered for consumptive coagulopathy. Other treatment methods tried are inhaled nitric oxide,[11] aerosolized prostacyclin[12] steroids, hemofiltration,[13] cardio-pulmonary bypass, thrombolysis and pulmonary artery thrombectomy,[14] exchange transfusion,[15] and intra-aortic balloon pump.[16]

Conclusion

Although the mortality due to AFE has shown a decline in number, especially in developed countries, it may be due to early re-organization and aggressive management. The pathophysiology of AFE remains an enigma. Although certain risk factors have been identified, AFE cannot be completely prevented.

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Conflicts of interest

There are no conflicts of interest.

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