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Anaphylactoid Syndrome of Pregnancy: A Diagnostic Dilemma Shiddappa Gundikeri^{1*}, Kashinkunti M. D.²

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Abstract: Anaphylactoid syndrome of pregnancyor Amniotic fluid embolism (AFE) is a rare obstetric catastrophe. Amniotic fluid embolism is a rare syndrome with potentially lethal outcomes. Complications include cardiorespiratory failure, disseminated intravascular coagulation, seizures, neurological deficits, and death. We describe a case of 24 year old primigravida with 8 weeks of amenorrhoea with anembroynic pregnancy. Following D & C patient developed breathlessness circulatory collapse and had disseminated intravascular coagulation. Patient was successfully reviewed by inotropes, heparin and mechanical ventilatory support. Amniotic fluid embolism is life threatening and difficult to predict or prevent, which should be always be kept in mind in a parturient with sudden cardiovascular collapse, so that resuscitation measures taken immediately, as early intervention is essential for a positive outcome.

Keywords: Anaphylactoid syndrome of pregnancy, Amniotic fluid embolism, disseminated intravascular coagulation, neurological deficits

INTRODUCTION

Amniotic fluid embolism (AFE) is a rare catastrophe with an estimated frequency of approximately 7.7 per 100 000 deliveries [1]. It was described first in1941 by Steiner and Luschbaugh with evidence of fetal debris in pulmonary circulation in a pregnant woman [2]. Recently, a more descriptive term was suggested as "anaphylactoid syndrome of pregnancy" [3, 4]. This is a rare but important cause of peripartum death in the UnitedStates making up for 10% of all maternal deaths with anoverall mortality rate as high as 60% to 80% [1]. Data from Indian literature is lacking.

More than 50% of patients die within first hour and about two-thirdwithin 5 hours of the event with high incidence of severeand permanent neurological damage among survivors. We report a primigravida who had cardiac arrest following dilatation and curettage resulting in disseminated intravascular coagulation (DIC).

CASE REPORT

A 24 years old third gravida female presented with 8 weeks of amenorrhea and history of leaking per vaginum since 1 day and was admitted for further observation and management. She had regular antenatal checkups in the institution with no history any comorbid illnesses or any allergies. Her transvaginal ultrasonography showed gestational sac of 6 weeks and anembroynic pregnancy condition. She underwent dilation and curettage for the above condition and her

Intra and immediate post op was uneventful. Her pulse rate was 68bpm, BP was 100/60 mmHg after the procedure and was shifted to wards.

Her initial laboratory parameters showed Hb of 11.8 g/dL, platelet count of 129 000 cells/mm3, total leukocyte count of 9200 cells/mm3, normal coagulation parameters, liver function tests (including enzymes) were also within normal limits and her arterial blood gas on face mask oxygen at 4 l/min showed pH 7.37; pO2 92 mmHg; pCO2 36 mmHg; HCO3- 24.5 mmol/L; SaO2 96%.

Within 3hrs after the procedure patient had a bout of vomiting followed by chest pain and breathlessness. She had gradually progressive deterioration of her sensorium. Her pulse rate was 120bpm and BP was 80/50 systolic. Immediate anesthetist and physician opinion was taken. Patient was resuscitated with fluids. In view of her low GCS, tachycardia and tachypnea she was put on mechanical ventilator. Her 2D echo revealed dilated right atrium and ventricle. She had abnormal bleeding per vaginally.Immediately a bedside clotting test was performed using a capillary tube which showed an abnormally increased clotting time. Resuscitation was continued with fluids and inotropic support was started with infusions of dopamine at 12 μg/kg/min and nor epinephrine at 0.1 μg/kg/min as blood pressure was not responding to intravenous fluids. 2 units of packed red blood cells and 4 units of fresh frozen plasma were transfused.

Mechanical ventilation was continued with the controlled mode of ventilation with 100% oxygen and an abnormal soakage of the dressing with collection of significant amount of blood in the abdominal drain was noticed. She remained hypotensive in the ICU inspite of inotropic supports with dopamine, nor epinephrine and epinephrine infusions. Her laboratory values showed Hb of 6 gm/dL, platelet count of 21000 cells/mm³, prothrombin time 14s, of activated thromboplastin time of 45.4 s with elevated D-dimer and fibrin degradation products thus raising suspicion of disseminated intravascular coagulation. A total of four units of packed red blood cells, 15 units of fresh frozen plasma and 10 units of platelet rich plasma were transfused. A repeat arterial blood gas analysis showed pH 7.23; pO₂ 132 mmHg; pCO₂ 31.5 mmHg; HCO₃-16.2 mmol/L; BE/BD-12; SaO₂ 99%.

With intensive resuscitation her bleeding stopped and gradually her sensorium improved. She was out of mechanical ventilator within 4 days and was discharged home after 7 days with hemoglobin of 10.2 g%, platelet count of 2.1 laks and normal coagulation profile.

DISCUSSION

Amniotic fluid is produced by the fetal membranes and the fetus. The fluid has a neutral pH and increases from 50 mL at 12 weeks to 1000 mL at 38 weeks. Amniotic fluid contains fetal components such as squamous cells from the skin, mucin, vernix, lanugo hairs, platelet activating factor, prostaglandins, complement activating factors, procoagulants, and sometimes meconium. Anaphylactoid syndrome of pregnancy is a dreaded complication of pregnancy. It gives no signals and warning signs.

The unfortunate event is sudden and drastic and may kill patient in no time despite aggressive management in good set up. For patient to survive requires intensive care and follow up most likely in Intensive Care Unit (ICU) [5]The condition is also referred to as anaphylactoid syndrome of pregnancy is a leading cause of maternal mortality [6]. Exact incidence in United States of America the condition occurs in about In 1 of 20000 to 1 of 80000 pregnancies [7].

Maternal mortality related to this syndrome ranges from 26% to 61%; however, as few as 15% may survive without neurological impairment [3, 8]. Of all affected patients, 50% die within the first hour [9].

Amniotic fluid embolism was first described in 1926 by Meyer, [10] Steiner and Lushbaugh in 1941, described autopsy findings from 8 pregnant women in whom pulmonary edema and shock developed during labor [2]. They suggested that powerful or tetanic contractions caused an embolism of squamous cells,

mucin, and/or otheramorphous debris, presumably from the fetus, to lodge in the patients' pulmonary vasculature. They termed this as "maternal pulmonary embolism by amniotic fluid" characterized by shock that developed during labor or shortly after delivery.

Benson presented a new clinical definition after summarizing 3 case reports of women who survived amniotic fluid embolism. The definition included sudden onset of cardiovascular collapse, sustained tachycardia, and the absence of other illnesses that could explain the signs and symptoms [11]. Anaphylactoid Syndrome of Pregnancy seems to occur after maternal intravascular exposure to fetal tissue. This exposure routinely occurs during normal labor and delivery and can also take place after placement of an intrauterine catheter, after uterine rupture, during cesarean section, or during spontaneous or surgical abortion. Under certain conditions, when amniotic fluid and its fetal components enter the maternal circulation along with endogenous mediators (e.g., prostaglandins, leukotrienes, histamine, bradykinin, cytokines, thromboxane, complement- activating factors, and platelet activating factor).

Symptoms depend on the amount and distribution patterns of the circulation obstruction. The powerful chemical mediators may incite a striking anaphylactic reaction. Both types of reaction may be seen together or separately. The pathological maternal response results in hypoxia, homodynamic instability, and/or consumptive coagulopathy [3, 4].

Differential diagnoses for this condition includes; hemorrhagic shock, sepsis, pulmonary embolism, air embolism, eclampsia, placental abruption, uterine rupture, uterine atony, uterine inversion, myocardial infarction, cardiomyopathy, anaphylactic reaction to local anesthetic agents and Hemolytic uremic syndrome of pregnancy [3, 11, 12].

The amniotic fluid emboli are described in brain, kidney, adrenal glands and many other organs. It is quite possible that many cases of pregnancy related Hemolytic Uremic syndrome may be in reality manifestation of amnioticfluid embolism. Renal and skin biopsies with meticulous examination of vasculatures of these organs may furnish more insight into the entity.

CONCLUSION

Anaphylactoid syndrome of pregnancy is a life threatening and difficult to predict or prevent condition that should be kept in mind in a pregnant lady especially having risk factors such as known history of anaphylaxis, advanced gestational age at termination of pregnancy, rupture of membranes, or meconium staining of amniotic fluid.

The main purpose to report this case is to stress on this uncommonly reported condition in India and difficulty in predicting and preventing this rare but fatal complication of pregnancy especially in centers withlimited resources where an algorithm for suspicion can be really helpful, as immediate resuscitation is essential for a positive outcome. It also stresses the importance of intensive care backup facilities for such cases.

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