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CASE REPORT

Amniotic Fluid Embolism with Trivial Deficit and Successful Subsequent Pregnancy: A Case Report

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ABSTRACT

Amniotic fluid embolism is an unpredictable, indistinct and rare condition with high morbidity and mortality for both pregnant women and her babies. We present a healthy 30 years old woman who had cyanosis, convulsion, cardiac arrest followed by disseminated intravascular coagulopathy in the beginning of the second stage of labor. Spiral CT confirmed intraluminal filling defect in segmental branch of posterior basal segment of both lower lobes of lungs. Although the diagnosis of amniotic fluid embolism was doubtful,prompt decision making and management were necessary for survival of the patient with trivial deficit. The patient survived and had another child in the following year.

Keywords: amniotic fluid embolism, cesarean section, cardiopulmonary resuscitation (CPR), coagulopathy

Introduction

Amniotic fluid embolism (AFE), a rare and life threatening obstetric condition was prescribed by Meyer in 1926[1]. Its incidence was 1:8,000-1:80,000 of pregnancies[2] varied from 1:15,200 and 1:53,800 deliveries in North America and Europe, respectively[3]. Morbidity and mortality from AFE was 61-86%. Most women who survive have permanent neurological impairment and neonatal survival is 79%[4].

Case Report

A healthy 30 years old, primigravida at 38 weeks of gestation was admitted to labor room due to labor pain. She had no previous medical or obstetric problems. Cervical dilatation was 5 cm with intact membranes. Fetal heart rate was 150 beat per minute (bpm) with reassuring pattern. Augmentation of labor with oxytocin and amniotomy was performed. Three hours after regular uterine contraction had occurred, cervix was fully dilated. The patient suddenly experienced cyanosis, acute respiratory failure, hypotension blood pressure with blood pressure (BP) of 80/50 mmHg, generalized tonic-clonic seizures, unconsciousness followed by cardiac arrest for 15 minutes after cyanosis. Endotracheal intubation was immediately performed and the patient was mechanically hyperventilated with a fraction of inspired oxygen (FiO2) of 1.0. Adrenaline 1 mg was given intravenous (IV) to her after which she developed transient ventricular tachycardia which resolved spontaneously. Central venous catheter was inserted, Magnesium sulfate 4 gram was pushed intravenously.

Blood pressure which was initially 96/30 mmHg and was stabilized at 130/70 mmHg while the pulse rate was 130 bpm with a continuous IV infusion of dopamine at 15-60 mcd/min and adrenalin 1:1 12 mcd/min. Synchronous with CPR, 10 minutes after cardiac arrest, patient was transferred to operating room for emergency cesarean section under general anesthesia. The patient was maintained on an FiO2 of 1.0 and the SpO2 reading was 100% throughout surgery. Intra-operative blood loss was 300 cc. A viable male infant weighing 3,700 gm was delivered with an Apgar scores of 2 at 1 min and 4 at 5 min, respectively. After successful cardiopulmonary resuscitation, he was transferred to the newborn intensive care unit (NICU). The patient was clinically coagulopathy, so 2.8 litres of FFP and 10 units of cryoprecipitate were transfused. Her hemoglobin (Hb) concentration was 8.0g/dl. Fluid resuscitation was continued totally 1litre of colloids, 800 cc. of packed red cells and 4800 cc of fresh whole blood. Haemostasis was secured. The patient was kept intubated and transferred to the intensive care unit (ICU) for further care. Her blood pressure was 90/70 mmHg, the pulse rate was 120 bpm and her conciousness was stuporous.

Laboratory results upon arrival at the ICU were: Hb concentration of 7.9 g/dl, platelets of 120,000 cells/cc^3, prolonged prothrombin time (PT) of more than 120 seconds (control 10-15 seconds), the partial thromboplastin time (PTT) was more than 190 seconds (control 25-40 seconds) and arterial blood gas demonstrated severe metabolic acidosis and tissue hypoxia, pH of 7.212, PaO_2 436.7 mmHg, $PaCO_2$ 34.5 mmHg and HCO_3 12.3 mmol/l. At ICU, she developed tonic-clonic seizures on her face for about 30 seconds. An active vaginal bleeding about 800 cc was observed. Diazepam 10 mg was injected intravenously. Methergin 0.2 mg was injected intramuscularly and dopamine 1:1 intravenous drip 3 ug/min was given. The central venous pressure was recorded at 16 cmH_2O from internal jugular vein catheter, an intra-arterial line through radial artery was inserted.

Hemodynamic stability was achieved after treatment, her blood pressure elevated to 133/75 mmHg, the pulse rate was 130 bpm. She remained unconscious due to diazepam. Vaginal bleeding was minimal. The patient was referred for further care to Ramathibodi Hospital in Bangkok. During transfer, she developed ventricular fibrillation which was corrected after defibrillation with 360 joules.

At Ramathibodi Hospital, her clinical condition was still unstable. Her blood pressure was 70/40 mmHg; the heart rate was 120 bpm. She did not respond to deep pain stimulation and still had generalized tonic-clonic seizures which later resolved spontaneously. On admission to the Ramathibodi ICU, the initial diagnosis was amniotic fluid embolism with respiratory failure and cardiogenic shock resulting from amniotic fluid embolism. A Spiral CT and an echocardiography showed intraluminal filling defect in segmental branch of posterior basal segment of both lower lobes of lung. Echocardiogram showed poor left ventricular ejection fraction and D-shape septum. A provisional diagnosis of postpartum cardiomyopathy, post intra-natal amniotic fluid embolism was made.

Her course in ICU remained uneventful. She was discharged from ICU on the fourth post-operative day and left the hospital on the eleventh post-operative day without any neurologic sequelae. Her baby was deemed normal on subsequent follow-up at the outpatient department. All problems were resolved without any remaining deficit during follow up for 2 years. She became pregnant again later and delivered a healthy male baby weight 3,700 grams by elective cesarean section without any complication.

Discussion

Amniotic fluid embolism, a rare condition, postulated by amniotic fluid, fetal cell, hair entering in to the maternal circulation. The neonatal survival is around 79%[2], depending on the prompt recognition and treatment. Some babies may have permanent neurological impairment. At present, data from National Amniotic fluid Embolus registry suggested that AFE- like anaphylactic reaction is more likely than embolism[5]. The term "Anaphylactic reaction of pregnancy" is usually used instead because no fetal components are universally found in women who present with sign and symptom of AFE[5]. Some time during pregnancy, fetal

part such as fetal red blood cell, may be found in the maternal circulation. The diagnosis of AFE usually is based on clinical signs and symptoms. Most patients have no evidence of fetal components in blood circulation[6]. Risk factors are poorly identified due to low incidence of this condition. Maternal age over than 35 years, operative procedure for delivery, cervical laceration, uterine rupture, placenta previa, pre-eclampsia or male fetus may increase risk of amniotic fluid embolism[7].

The patho-physiology of this condition is poorly understood, it may be related to complement activation. AFE can occur only when there is a breach in the barrier between the amniotic fluid and maternal circulation. The three most common routes of entry are the endocervical veins, the placental site and a traumatized uterine site[8,9]. The complements are activated. Pulmonary artery spasm occurs immediately at that time. It produces pulmonary hypertension, increase right ventricular pressure, hypoxia, pulmonary and myocardial capillary damage, left ventricular failure and acute respiratory distress syndrome (ARDS) respectively. Hemorrhagic phase usually follow, producing a massive bleeding and disseminated intravascular coagulopathy (DIC).

Currently, there is no definite diagnostic test available. The United States[10] and United Kingdom[11] AFE registries have recommended the following 4 criteria, all of which must be present to make the diagnosis:

1. Acute hypotension or cardiac arrest
2. Acute hypoxia
3. Coagulopathy or severe hemorrhage in the absence of other explanations
4. All of these occurring during labor, cesarean delivery, dilatation and evacuation, or within 30 minutes of postpartum with no other explanation of findings.

Because the situation is unpredictable and unpreventable so prompt and differential diagnosis are very important. There is unfortunately no routine or standardized diagnostic scheme to confirm AFE and hence it remains a diagnosis of exclusion. A review of the largest case-series to date concluded that the physiologic and hematologic sequelae of AFE resemble septic or anaphylactic shock rather than an embolic phenomenon[6]. We should also be aware of anaphylaxis, aortic dissection, pulmonary embolism, myocardial infarction, septic shock, eclampsia or postpartum hemorrhage with shock[8]. Recent studies[12-14] used the new method of diagnosis by detecting zinc-coproporphyrin I, TKH-2 monoclonal antibody reaction with mucin and detection of tryptase.

Histological findings from autopsy showed evidence of fetal debris in blood vessels in the lung, 80% of which is fetal squamous cell[15]. Interstitial cell sometimes were found in multiple organs such as kidney, left ventricle, interventricular septum. Alcian blue periodic acid-Schiff (PAS) stain was positive for mucin in the vasculature and oil red O stain for lipid was positive in the lungs[14].

Treatment of amniotic fluid embolism is mainly supportive treatment[7]. Medical care includes oxygen support, CPR, intravenous fluid, pulmonary artery catheter insertion, fetal monitoring and correction of coagulopathy. Emergency cesarean section is needed to save the fetus, hysterectomy or uterine artery embolization or ligation may be needed for the treatment of the subsequent postpartum hemorrhage.

In our case, clinical condition was typical for AFE because there were symptoms of sudden dyspnea and respiratory failure during second stage of labor, followed by seizure, cardiac arrest and DIC respectively. With prompt CPR and active management she survived with no trivial deficit and was able to have subsequent pregnancy as previously report[16].

Conclusion

Because critical point of AFE is unpredictable, unpreventable condition with high morbidity and mortality for pregnant women and her child, awareness of diagnosis and immediate treatment is the most important. The others are multidisciplinary approach and awareness of other differential diagnosis of this condition. This report case of AFE treated successfully. The patient and her baby survived without deficit and she was able to have another baby subsequently.

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รายงาน ผู้ป่วยภาวะน้ำคร่ำอุดตันในกระแสเลือดรอดชีวิตและมีบุตรคนต่อมา

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บทคัดย่อ

ภาวะน้ำคร่ำอุดตันในกระแสเลือดเป็นภาวะที่ไม่สามารถจะคาดเดาว่าจะเกิดเวลาใดได้พบไม่บ่อยแต่เป็นอันตรายถึงชีวิตทั้งมารดาและทารก กรณีศึกษาหญิงไทยอายุ 30 ปีในขณะเริ่มต้นระยะที่สองของการคลอด มีภาวะขาดออกซิเจนอย่างรุนแรง ชัก และหัวใจหยุดเต้น การวินิจฉัยยืนยันด้วยการตรวจเอกซเรย์คอมพิวเตอร์ชนิดหมุนรอบพบจุดว่างในหลอดเลือดของเส้นเลือดในปอดส่วนล่าง ถึงแม้การวินิจฉัยที่ยังไม่ชัดเจนแต่การตัดสินใจและการรักษาที่ทันท่วงทีก็สามารถช่วยชีวิตมารดาและทารกได้โดยไม่พบภาวะผิดปกติ จนสามารถมีบุตรได้อีกหนึ่งคนในอีกสองปีต่อมา
