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CaseReport Open Access

Successful Resuscitation Following Amniotic Fluid Embolism During Emergency Lower Segment Caesarean Section: A Case Report

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Abstract

Amniotic fluid embolism (AE) is a rare and potentially fatal condition occurring in obstetric patients, which continues to have a high mortality rate. We report a case of AE occurring during the peri-partum period, in which due to expeditious cardiopulmonary resuscitation, both the mother and the newborn survived. High degree of suspicion and prompt action is mandatory for a favorable outcome in such scenarios.

Keywords: Amniotic fluid embolism; Cardiopulmonary resuscitation; Emergency lower segment; Caesarean section; Peripartum period

Introduction

Amniotic fluid embolism continues to have a high mortality [1] despite the technological advancements in diagnosis and management. It is also alternatively called "sudden obstetric collapse" or "anaphylactoid syndrome of pregnancy" [2]. Prompt detection and management is the key to success. We hereby report a case of near-fatal pulmonary embolism with a positive outcome.

Case report

A 27 year old primigravida, at 39 weeks of gestation presented for emergency lower segment caesarian section (LSCS). She was a known case of pregnancy induced hypertension (PIH) and Rh-negative blood group. She had no other co-morbidities with an uneventful antenatal period. All preoperative investigations including thyroid function tests and viral markers were normal. Preoperative vital parameters were as follows – Blood pressure (BP) was 140/90 mm Hg, Heart rate (HR) was 100/min and Oxygen saturation (Spo2) was 100%. After administering aspiration prophylaxis and intravenous crystalloids, she was given a subarachnoid block using 2.2 ml of 0.5% hyperbaric bupivacaine with a 25G spinal needle. Level of blockade attained was found to be satisfactory. She remained haemodynamically stable after subarachnoid block till the delivery of the baby, when a vigorous fundal pressure was given as requested by the obstetrician. Baby was extracted and the baby cried immediately. APGAR scores at 0, 5 and 10 minutes were 8, 9 and 9. Immediately after delivery, she had a sudden loss of consciousness with an episode of bradycardia. Initial HR was 50/min (regular), followed by a fall in BP to 80/40 mmHg from 130/80 mm Hg. Oxygen saturation (SpO2) also decreased to 80% from 100%. Injection atropine 0.6 mg and injection ephedrine 6 mg I.V given were given. HR, BP and Spo2 continued to fall. Immediately, patient was intubated with a 7 mm cuffed endotracheal tube and fixed after conformation of bilateral equal air entry. Patient was ventilated with 100% oxygen. Cardiopulmonary resuscitation (CPR) was started with chest compressions and injection Adrenaline 1 mg I.V was given. ECG (electrocardiogram) showed ventricular tachycardia, for which 200 joules DC shock was given. Sinus rhythm appeared with HR of 130/min, BP 120/70 mmHg and Spo2 100%. Chest was bilaterally clear. Arterial blood gas analysis (ABG) was sent for analysis and patient was shifted to intensive care unit (ICU) for elective ventilation. In the ICU, patient was put on ventilator with synchronized (SIMV) mode. Vital parameters were as follows: BP 140/100 mmHg, HR 120/min and Spo2 100%. On examination, pupils were mid dilated, sluggishly reacting to light, with uprolling of eye balls, jerky movements in hands and spasticity of both the upper limbs. Patient was febrile. All routine investigations including coagulation profile, D-dimer and fibrin degradation products (FDP) were sent to laboratory. Patient was given intravenous (I.V) furosemide 10 mg, hydrocortisone 100 mg I.V and calcium gluconate 10 ml I.V slowly. ABG (arterial blood gas) analysis revealed pH 7.34, PCO2 30 mmHg, PO2 400 mmHg, HCO3 16.3 meq/l.

Blood investigations revealed the following: Hemoglobin 11.2 gm%, TLC 19000/cumm and platelet count 1.9 lakh/mm3. Coagulation profile, liver function and renal function tests were normal. In view of jerky movements in hands and spasticity in upper limbs, loading dose of Phenytoin sodium (antiepileptic) was started followed by maintenance dose (100 mg I.V three times a day).

On day 2 in the ICU, patient was conscious with stable vital parameters and no uprolling of eye balls or jerky limb movements. Weaning trial was started and the patient was extubated after 4 hours. After extubation, HR was 88/ min, BP 120/70 mmHg, SPO2 100% and RR 24/min. On day 3 in the ICU, patient was drowsy, but arousable and afebrile. Pupils were normal sized and reactive to light, and movements of the limbs were normal. Muscle power grading was 3/5 in both upper and lower limbs. Serum FDP (80 ug/nl), D-dimer (7689 ng/ml) and Fibrinogen levels (465 mg/dL) were markedly raised. On suspicion of pulmonary embolism, patient was put on empiric heparin therapy (low molecular weight heparin: Enoxaparin 0.6 mg subcutaneous twice a day). Magnetic resonance imaging (MRI) of the brain and pulmonary CT angiography were done. MRI revealed bilateral thalamic swelling with faint hyperintense signal on T2 images, for which methylprednisolone 1 gm I.V daily was started. CT pulmonary angiography revealed the following: distension of post sub-segmental branch of right pulmonary artery supplying the region of superior segment of right lower lobe with hypodense filling defect suggestive of thrombus (Figure 1).

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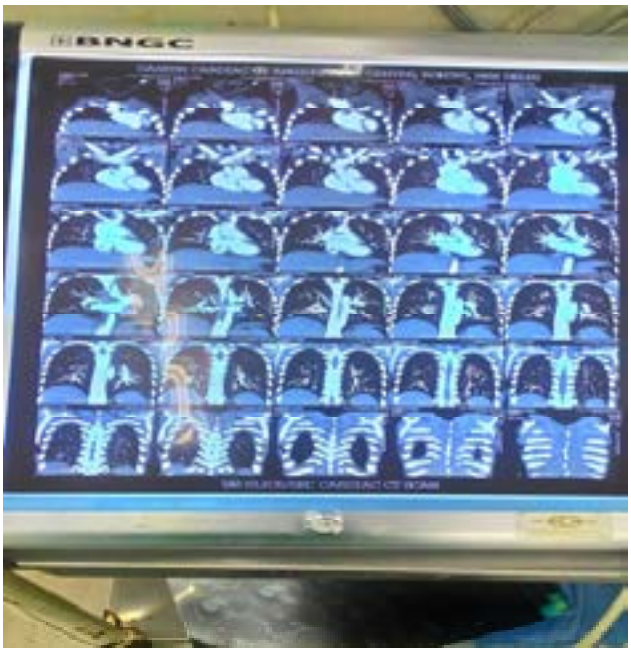

Figure 1: CT pulmonary angiography.

On day 4 in the ICU, the patient was conscious, oriented, was able to walk with support and allowed orally. Two-dimensional echocardiography was done which showed a normal study. LMWH therapy was continued and the patient was observed in the ICU for two more days and then shifted to ward. She was later discharged home on oral Warfarin therapy in a stable condition.

Discussion

The commonly reported incidence of amniotic fluid embolism (AE) ranges between 1:8000 to 1:80000 institutional deliveries [3]. It still remains a diagnosis of exclusion with varying incidence rates in different communities. In developing countries, where home deliveries are still rampant, it may go undetected. It typically occurs during labor and delivery or immediate post-partum. It classically presents as sudden cardiovascular collapse, hypoxia and coagulopathy. It can also cause adverse fetal effects including fetal distress and death, if occurring before delivery. Its etiology [4] involves mechanical obstruction of the maternal pulmonary vasculature by the amniotic fluid, followed by a cascade of immune responses due to the presence of amniotic fluid in the maternal circulation. These humoral mediators [5] which contribute to coagulation abnormalities, increased vascular permeability and bronchoconstriction include prostaglandins, leukotrienes, platelet activating factor and tissue factor. Mortality rates are still high, ranging between 20-40%.

The key risk factors [6] for the development of AE include placental abnormalities, Caesarian section, forceps or vacuum assisted delivery, eclampsia, advanced maternal age (>35 years) and induction or augmentation of labor. The criteria for its diagnosis include acute hypotension and/or cardiac arrest, acute hypoxia heralded by dyspnea, cyanosis and/or respiratory arrest, and coagulopathy or severe clinical haemorrhage in the absence of other explanations. Its differential diagnosis [7] commonly includes air embolism, anaphylaxis, myocardial ischemia, arrhythmias, local anesthetic toxicity or high spinal block, uterine rupture, peripartum cardiomyopathy, severe eclampsia or acute hemorrhage. Treatment is largely supportive, where aggressive resuscitation can critically affect maternal and fetal survival [8].

Our patient had a history of PIH (controlled) in the antenatal period and had presented for an emergency LSCS. She was also given fundal pressure for extraction of the fetus. Immediate action was taken by the anesthesia team on development of sudden hypotension and hypoxia. Aggressive CPR was administered, including shock apart from endotracheal intubation. A probable diagnosis of pulmonary embolism, possibly due to amniotic fluid was made and the patient was started on Heparin (LMWH). Management in the ICU was largely supportive. After extubation, pulmonary computed tomographic angiography was done, whose findings confirmed the diagnosis of pulmonary embolism. Once the patient was stabilized, she was shifted to ward and finally discharged home on oral warfarin therapy.

Our case highlights that a high index of suspicion for AE must be maintained during caesarian deliveries as well as during normal labor in patients with co-existing risk factors. Prompt diagnosis and expeditious management can improve both maternal and fetal survival.

Conclusions

Amniotic fluid embolism is a near fatal condition unique to the obstetric population, where mortality rate continues to be high. It is usually a diagnosis of exclusion, as pulmonary angiography may not be available or feasible for confirming the diagnosis. Aggressive resuscitation, maintaining adequate oxygenation, empiric heparin therapy and supportive care are mandatory for a favorable outcome. We could save the life of both the parturient and the fetus due to prompt diagnosis and management, both in the emergency operating room and the ICU.

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