

## Pulmonary Edema (Non-Cardiogenic) and Acute Respiratory Distress Syndrome Secondary to Amniotic Fluid Embolism Syndrome Following Preterm Delivery of Intrauterine Fetal Death: A Case Report

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### Abstract

A 27 years old healthy female of twenty eight weeks pregnancy with history of low grade fever and dry cough for one day presented with intrauterine fetal death. Following spontaneous preterm delivery of the dead fetus, within three hours, patient developed irritable cough, dyspnea, tachypnea, restlessness and cyanosis. She was put on face mask with oxygen flow of ten litres/minute and was nebulized with Salbutamol in the delivery suite but gradually she developed desaturation of 76%. As the condition of patient was worsening patient was transferred to intensive care unit. In intensive care unit patient was intubated and put on ventilator immediately. X-ray chest was showing bilateral infiltrates and arterial blood gases was showing PaO<sub>2</sub>/FiO<sub>2</sub> ratio of 55.6%. Pulmonary capillary wedge pressure was not checked as pulmonary catheterization is not practiced in our Intensive care unit. Cardiogenic component of pulmonary edema was ruled out indirectly by history, electrocardiogram, echocardiography, central venous pressure and X-ray chest (heart shadow). She was diagnosed as a case of severe acute respiratory distress syndrome (American European consensus conference guideline, 2012) and non-cardiogenic pulmonary edema due to amniotic fluid embolism syndrome. In course of management, maximum emphasis was given on lung protective ventilation and fluid, conservative strategy along with medical and other supportive management. On her eighth day on ventilator, she was extubated and on ninth day, she was shifted to maternity ward. On fourteenth day she was discharged from hospital. She came for follow up after one month of her discharge and was found to have no residual complication.

**Keywords:** Amniotic fluid embolism syndrome; Oxygen saturation; Tidal volume; Arterial oxygen tension; Fraction of inspired O<sub>2</sub> concentration; Positive end expiratory pressure; Carbon dioxide tension

### Introduction

Entry of amniotic fluid into the maternal circulation was first described in 1926 by Meyer. Once amniotic fluid embolus syndrome occurs, severe hypotension due to cardiovascular collapse, arrhythmia, cyanosis followed by pulmonary edema, acute respiratory distress syndrome and massive hemorrhage by disseminated intravascular coagulopathy followed by multiple organ dysfunction leading to maternal death. Amniotic fluid embolism syndrome factors are anaphylaxis, vascular occlusion, vasoconstriction by prostaglandin or others and disseminated intravascular coagulopathy. Non-cardiogenic pulmonary edema can be subclinical, to acute, with severe respiratory compromise. Acute dyspnoea after pregnancy is rare, but causes are pulmonary embolism, amniotic fluid embolism syndrome, pneumonia, aspiration and pulmonary edema [1-6].

### Case Report

A twenty three years old gravida 4 para 0 abortion 3 was referred to Buraimi hospital with twenty eight weeks pregnancy having preterm premature rupture of membranes for three days with gestational diabetes mellitus on diet with low grade fever and dry cough since one day. She was with cervical cerclage. Patient's weight was seventy kilograms. Her first and second pregnancies were first trimester missed miscarriages. Third pregnancy was intrauterine fetal death at seventeen weeks of gestation.

On admission findings were as follows. Temperature-38 degree centigrade, pulse rate-110/minute, blood pressure-110/70 mmHg,

SpO<sub>2</sub>-98% on room air. Abdominal examination was as follows. Uterus was irritable, non-tender, absent fetal heart sounds. Ultrasonography confirmed intrauterine fetal death corresponding to 28 weeks, placenta fundal, and liquor was minimal. Estimated fetal birth weight was 1.2 kilograms. Per speculum examination demonstrated clear liquor in



**Figure 1:** X ray chest on day one (anterio-posterior view) was showing bilateral and almost symmetrical confluent opacity in both lung fields except a few areas of both apices and peripheral lung regions.

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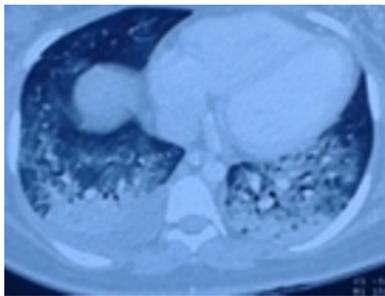
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|                                  | Day-1     | Day-2     | Day-3     | Day-4     | Day-5     | Day-6   | Day-7   | Day-8                |
|----------------------------------|-----------|-----------|-----------|-----------|-----------|---------|---------|----------------------|
| Mode                             | ACMV - VC | PSV     | PSV     | T Piece - Extubation |
| Fio2                             | 1         | 0.9       | 0.8-0.7   | 0.7       | 0.7-0.6   | 0.6-0.5 | 0.5-0.4 | -                    |
| Tidal volume                     | 380       | 380       | 400       | 420       | 450       | PS15-10 | PS10-6  | -                    |
| Positive end expiratory pressure | 18        | 18-16     | 16-13     | 13-12     | 10-6      | 6       | 6-4     | -                    |
| Spo2                             | 70-80     | 80-91     | 91-93     | 94        | 96        | 97      | 98-100  | -                    |
| Peak inspiratory pressure        | 35        | 33        | 30        | 27        | 23        | 21      | 10      | -                    |
| Pao2                             | 55.6      | 63        | 76        | 95        | 162       | 146     | 141     | 104                  |
| Pco2                             | 73        | 51        | 40        | 48        | 44        | 40      | 41      | 43                   |
| Pao2 / Fio2 ratio                | 56        | 70        | 72        | 136       | 405       | 350     | 360     | 365                  |

**Table 1:** Day wise ventilatory parameters and arterial blood gases with Pao2 / Fio2 ratio.

|                                | Day-1            | Day-2            | Day-3            | Day-4           | Day-5           | Day-6         | Day-7    | Day-8    |
|--------------------------------|------------------|------------------|------------------|-----------------|-----------------|---------------|----------|----------|
| <b>Central venous pressure</b> | <b>13</b>        | <b>13</b>        | <b>12</b>        | <b>13</b>       | <b>11</b>       | <b>10</b>     | <b>8</b> | <b>8</b> |
| Inotropes                      | Dopa-mine 20 mcg | Dopa-mine 15 mcg | Dopa-mine 10 mcg | Dopa-mine 8 mcg | Dopa-mine 5 mcg | Dopa-mine off | -        | -        |
| Fluid balance                  | +800             | +650             | +200             | +150            | +50             | -200          | -50      | -150     |

**Table 2:** Day wise central venous pressure, inotropes and fluid balance.



**Figure 2:** Pulmonary CT showed no pulmonary embolism, bilateral pulmonary edema, bilateral consolidation (right > left) and mild right pleural effusion. (Patient was hemodynamically unstable hence pulmonary CT with contrast was not done).

vagina. Cervical suture was removed. Vaginal examination showed cervix was soft, uneffaced, with 2 cm dilatation (Figure 1).

Investigations at admission were as follows. Hemoglobin-8.9 gm/dl, white blood cell count-27.3 K/ul. Normal platelet count, coagulation profile and liver function test and raised serum creatinine. With this findings patient was started with intravenous fluids, Cephalosporins and Metronidazole. Spontaneously patient delivered dead female fetus weighing 1.030 kilograms with foul smelling hind water. Placenta delivered spontaneously. She developed signs of respiratory failure which was managed with oxygen but SpO<sub>2</sub> dropped to 76%, so patient was intubated (profuse blood stained endotracheal secretion) and shifted to intensive care unit with ventilatory mode (assist control mechanical ventilation-volume control). In intensive care unit her heart rate was 145/minute, blood pressure was 85/60 millimeter of mercury and SpO<sub>2</sub>-70% (Tables 1 and 2). On auscultation of chest there were bilateral coarse crepitations. Electrocardiogram was showing sinus tachycardia. Echocardiography excluded left ventricular failure. Arterial blood gases were showing hypoxia with metabolic acidosis. There was no evidence of left atrial hypertension (history, electrocardiogram, echocardiography, X-ray chest).

Blood culture was negative. Activated prothrombin time was 120 with international ratio of 2.05 and D-dimer was positive (Figure 2). X-ray chest on day one (anterio-posterior view) was showing bilateral and almost symmetrical confluent opacity in both lung fields except a few areas of both apices and peripheral lung regions.

## Diagnosis

In view of the acute respiratory distress syndrome with absence of pulmonary embolism in X-ray chest and pulmonary CT, in a patient of post-partum period is highly suggestive of amniotic fluid embolism syndrome for which no definite diagnostic tests are available. Amniotic fluid embolism syndrome diagnosis is generally reached based on the clinical criteria and radiological findings. In this patient, amniotic fluid embolism is a high possibility.

Hence patient was diagnosed as severe peripartum acute respiratory distress syndrome due to amniotic fluid embolism syndrome [7] with differential diagnosis of septicemia and pulmonary embolism.

## Management

There is no specific treatment for amniotic fluid embolism syndrome. The goal of therapy is to correct hypoxemia and hypotension to avoid ischemic consequences (for example, hypoxic brain injury and acute kidney injury) Delivery of the fetus is ensured in women who have not delivered. For our patient the treatment was given as follows. Ventilatory management (lung protective ventilation) 18- Lung protective ventilation is low tidal volume & high positive end expiratory pressure. Drugs used were Cisatracurium, Fentanyl and Midazolam Day wise ventilatory parameters and arterial blood gases with PaO<sub>2</sub>/FiO<sub>2</sub> ratio.

## Medical management and other supportive care

Patient received antibiotics, heparin, stress ulcer prophylaxis, nutritional support from day three with good glycemic control and correction of electrolyte imbalance [8-11].

The complications encountered during course of management were severe hypotension, renal impairment which was managed with fluid and inotropes. Coagulation derangement treated with fresh frozen plasma. Pleural effusion resolved spontaneously. Hypernatremia was managed with 5% Dextrose and plain water through nasogastric tube (Ryle's tube). Patient was shifted to Maternity ward on day nine and discharged on day fourteen.

X-ray chest on day fourteen showed improved lung fields. No pleural thickening or small cysts [3]. Follow up after one month revealed no complications (cognitive disorder, muscular weakness [12-14].

## Conclusion

Pulmonary edema and acute respiratory distress syndrome secondary to amniotic fluid embolism is rare, but fatal. When a woman has sudden cardiopulmonary collapse and hemorrhage, during parturition or post natal, immediate cardiopulmonary resuscitation and delivery of fetus has good prognosis. But neurologic damage caused by hypoxia cannot be prevented.

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