

# AMNIOTIC FLUID PULMONARY EMBOLISM

## (A Case Report)

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### Introduction

Amniotic fluid embolism is a rare complication of pregnancy and labour, but has been described as the most dangerous, unpredictable and untreatable condition in Obstetrics (Courtney, 1973 and Morgan, 1979). The clinical and pathological features of the condition was first described by Steiner and Lushbaugh (1941). The cardinal signs of amniotic fluid embolism are respiratory distress, cyanosis, cardiovascular collapse and coma (Schnider and Moya, 1961). The present case deals with the problem of acute cardiopulmonary insufficiency due to amniotic fluid embolism without any bleeding diathesis.

### CASE REPORT

Mrs. A., aged 19 years, primigravida was admitted on May 5, 1980 at 11 p.m. as a case of mild pre-eclamptic toxæmia in spontaneous labour at term. The patient was in labour since last 24 hours. Her vitals at the time of admission were; pulse 92/min., temperature 37°C, respiratory rate 20/min., B.P. 130/90 mm of Hg

with mild oedema feet. Systemic examination of respiratory and cardiovascular systems revealed no abnormality. On abdominal examination uterus was of term size, head engaged, foetal heart sounds present, regular 136/min. uterine contractions were occurring every one to four minutes and lasting for 20 to 45 seconds. On vaginal examination cervix was 4 fingers breadth dilated with intact bag of forewaters.

About 2 hours later following artificial rupture of membranes and syntocinon drip, patient had an attack of shivering and tightness in the chest associated with mild haemoptysis and tachycardia. Immediately, resuscitative measures carried out. Foetal heart sounds could not be localized and within 5 minutes, a fresh still born infant weighing 2.8 Kg was delivered by forceps. The placenta and membranes expelled out completely and the blood loss was estimated as 400 ml. On examination, the patient was dyspnoeic, cyanosed with a pulse rate of 150/min., B.P. 90/60 mm of Hg and respiration 50/min. Chest examination revealed diminished air entry on right base with extensive crepitations and rhonchi all over the chest. The provisional diagnosis of acute pulmonary oedema following amniotic fluid embolism was made.

On investigations, her haemoglobin was 9.4 gm% with normal platelet counts: bleeding, clotting and prothrombin time. Blood urea was 68.5 mg% with normal fundus examination. E.C.G. showed sinus tachycardia. X-ray chest revealed evidence of pulmonary embolism (Wedge shaped shadow on right base), followed by appearance of pulmonary oedema in second film, taken after 2 hours of the initial film.

Treatment consisted of back rest, moist oxygen, intravenous frusemide, aminophylline and

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corticosteroids along with broad spectrum antibiotic coverage. With the above treatment, initially, patient showed some improvement in her condition, although, she remained dyspnoeic and cyanosed. Later on, she deteriorated and went into cardio-respiratory arrest on May 6, 1980 at 2 P.M. from which she could not be revived. As the relatives of patient refused for autopsy, sample of blood was aspirated from right ventricle which after centrifugation showed a buffy layer rich in amniotic fluid debris.

#### Discussion

Whenever unexplained breathlessness, cyanosis and/or disseminated intravascular coagulation occur in the post partum period, amniotic fluid embolism should be suspected. In the present case, patient had no bleeding, hence one is presented with a differential diagnostic problem consisting of respiratory distress and shock in the early post partum period. All forms of pulmonary embolization should be considered. Vascular emboli occur later in the puerperium with the maximum incidence being between the 9th and 14th days and are usually preceded by the evidence of venous thrombosis. Pulmonary air embolus at delivery is rare and may be excluded by the absence of churning or water wheel murmur. Aspiration pneumonitis (Mendelson's syndrome) should also be considered, but the history is usually conclusive. All forms of myocarditis including rheumatic must also be excluded since they may be the basis for acute cardiac decompensation (Shelley *et al* 1959). Cardiac failure secondary to toxæmia of pregnancy should be included in the differential diagnosis, but it is usually seen

in the late phases of eclampsia as a terminal event.

Risk factors for the development of amniotic fluid embolism includes difficult labour, use of uterine stimulants, intra-uterine death of foetus, multiparity, advanced maternal age, premature rupture of membranes and meconium staining in amniotic fluid (Liban and Raz, 1969). Amniotic fluid embolism has been thought to be associated with a bleeding diathesis, however, at the present time such an association may not be always clinically evident (Hunter *et al* 1956). The diagnosis of amniotic fluid embolism is usually made on post mortem examination, but if autopsy is refused, diagnosis may be possible by aspirating the blood from right ventricle as done in present case. This sample is centrifuged, separating it into the red cell component and a layer of white cells above which is a buffy coat rich in amniotic fluid debris (Gross and Benz, 1947).

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