

AMNIOTIC FLUID EMBOLISM

(Report of a case with survival of mother and baby)

by

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Pulmonary embolism, due to particulate matter contained in the amniotic fluid, was introduced in the literature by Steiner and Lushbaugh in 1941. It is a rare obstetric emergency ending fatally in most cases. Seltzer and Schuman (1947) described the first non-fatal case.

Aguillion and associates (1962) analysed 60 proved cases in the literature by 6 different authors, from 1941 to 1957, and added 5 cases of their own including 1 survival. According to Ian Cope (1964) less than 75 cases have been reported in the literature with 12 survivals. He added 1 case of his own with survival of both mother and foetus.

The case reported here, presented with the typical clinical picture of the syndrome and the author had the opportunity of studying the radiological chest findings, correlating with the clinical picture.

Case Report

Mrs. H. N. K., aged 32 years, of height 5 feet (152 cm), weight 127 pounds (58 Kg), 2nd gravida, had been attending antenatal clinic since 20 weeks of gestation. Her expected date of delivery was 5th June 1964. Antenatal check-up for this preg-

nancy was normal. Her obstetric history was, that 8 years ago her first labour was terminated by caesarean section for face presentation and uterine inertia, after she had been in labour for 46 hours.

She was admitted in the hospital on 2nd June 1964 awaiting delivery. On clinical examination her blood pressure was 110/70 mm of Hg, pulse 78 per minute; there was slight oedema on both the ankles. Heart and lungs were normal. On abdominal examination, there was a lower right paramedian scar, vertex was floating and presenting in left occiput-posterior position. Foetal heart rate was 140 per minute and regular. On vaginal examination there was no cephalo-pelvic disproportion. It was decided to allow her to go into natural labour. Her urine showed no albumin or sugar. Haemoglobin was 12.0 g. per 100 ml. Bleeding time was 1 minute 50 seconds and clotting time 3 minutes 48 seconds.

On 10th June 1964 at 1.30 a.m. she started mild labour pains. At 6 a.m. abdominal examination revealed that vertex had engaged, foetal heart rate was 140 per minute in the left occiput-transverse position. On vaginal examination the cervix was thinned out and the cervical os was 2 fingers loose, membranes were intact; the vertex was in the left occipito-transverse position in the mid-cavity. She was sedated with an injection of pethidine 50 mg. At 8 a.m. membranes ruptured spontaneously and the vertex was seen at the outlet. Till 9.50 a.m., in spite of fairly strong uterine contractions, there was no progress of labour. Foetal heart rate had gone up to 160 per minute and mother's pulse was 90 per minute with blood pressure of 110/70 mm of Hg. An injection of atropine gr 1/100 (0.65 mg) was given at 9.50 a.m. A Ryles

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tube was passed and a small amount of fluid was aspirated from the stomach. General anaesthesia was started with gas and oxygen and an endotracheal tube was passed. The foetal head was manually rotated from occiput-transverse position to occiput-anterior and low forceps applied. Before the foetus was extracted, it was reported by the anaesthetist that the patient was deeply cyanosed. A male baby was extracted at 10.25 a.m. with the help of forceps and by performing a mediolateral episiotomy, which was stitched in layers. The placenta was expelled completely 15 minutes later. The infant, weighing 7 pounds (3.17 Kg) was deeply asphyxiated and was revived by clearing of the air passages and oxygen inhalation. It was noticed that the mother was not only cyanosed, but her pulse rate had gone up to 140 per minute, her blood pressure had fallen to 90/60 mm of Hg and the respiratory rate was raised to 36 per minute. There was no unusual vaginal bleeding and the uterus was well contracted. The possibility of amniotic fluid pulmonary embolism was thought of. In the meantime the patient regained consciousness and complained of heaviness and pain in the chest. On auscultation of the chest, heart sounds

were normal, though rapid. Few moist rales were heard on the right side of the chest. Patient looked very apprehensive. She was given oxygen inhalation by intranasal catheter at 6 litres per minute. A dextraven drip was started and also hydrocortisone 100 mg and phenergan 25 mg were given intravenously. Injection of achromycin 100 mg was also given intramuscularly. Urgent electro-cardiogram was taken and it revealed a normal pattern. With a portable x-ray machine, skiagram of the chest was taken at 11 a.m. The skiagram (Fig. 1) showed scattered woolly patches in both lung fields. Patient's pulse, blood pressure and respiratory rates remained the same. Looking at the skiagram of the chest, intravenous fluid was restricted.

By 2 p.m. the patient's condition deteriorated, pulse was 160 per minute, blood pressure was 98/68 mm of Hg, respiratory rate 40 per minute. Clinically moist rales were heard on both sides of the chest. Another electro-cardiogram was taken, which showed sinus tachycardia. X-ray of chest was repeated at 2 p.m. (Fig. 2). It showed marked increase of the woolly patches in both the lung fields. She was given Intravenous digoxin 0.5 mg slowly. Injection

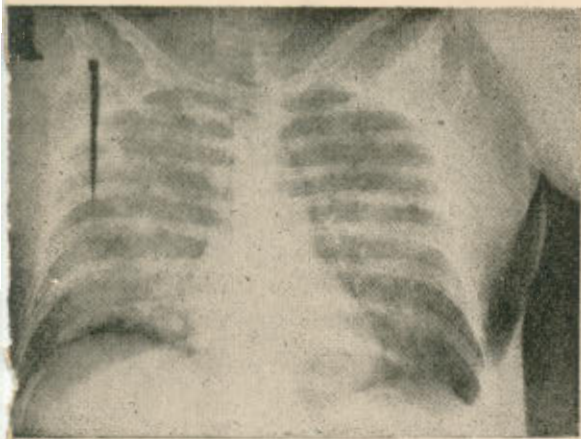


Fig. 1

Chest skiagram 35 minutes after delivery, showing scattered woolly patches in lungs due to amniotic fluid pulmonary embolism

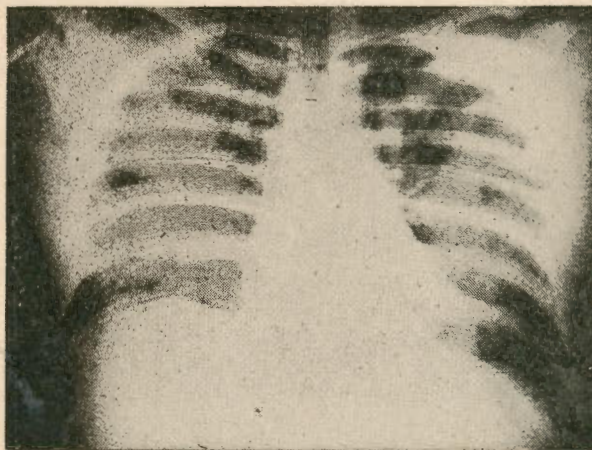


Fig. 2

Same 3 hours 35 minutes after delivery showing marked increase of the woolly patches in lungs.

phenergan 50 mg was repeated intramuscularly. Dextraven transfusion was replaced by whole blood transfusion with 100 mg of hydrocortisone in the drip.

The other investigations revealed that the prothrombin time of the patient was 29 seconds, whereas the control test was 19 seconds. Her blood urea was 39 mg per cent, haemoglobin was 10.0 g per cent. Total white cell count was 15,500 per cmm with polymorphs 80 per cent, lymphocytes 16 per cent, eosinophils 3 per cent and monocytes 1 per cent. Her bleeding time was 2 minutes 5 seconds and clotting time was 4 minutes 2 seconds. Blood fibrinogen was 0.3 gm/100 ml.

By 2.45 p.m. patient's condition had slightly improved clinically, cyanosis was less, respiratory rate was 34 per minute, pulse was 140 per minute and blood pressure was 110 mm of Hg. There was no change in the physical signs in the lungs.

By 5.50 p.m. her condition had deteriorated again with pulse rate of 155 per minute and blood pressure 100/68 mm of Hg. with respiratory rate 45 per minute. There was slight cyanosis in spite of oxygen inhalation. Under E.C.G. control, digoxin 0.5 mg was repeated intravenously and then 0.25 mg was given every 6 hours.

Hydrocortisone 50 mgm and phenergan 25 mgm were given every 8 hours.

The patient's condition remained critical throughout the night. By 6 a.m. next day, on 11th June 1964, her general condition had slightly improved. Pulse was 130 per minute, blood pressure was 100/60 mm of Hg. and respiration rate was 36 per minute. Heart sounds were normal. Auscultation of the lungs revealed fine moist rales at both the bases, more on the right side. Electro-cardiogram taken at 6 a.m. showed effect of digitalis, but there was no evidence of digitalis toxicity. X-ray of chest was repeated (Fig. 3) which showed diminution of the woolly patches in both lung fields. Total fluid given by intravenous route was 1 litre. She was started on digoxin tablet 0.25 gm every 6 hours and the injections were omitted. Other treatment given:— intravenous administration of aminophyllin 10 cc (0.25 g.) every 8 hours, and tablet fofane (Benzthiazide) 50 mg daily with potassium chloride gr 15 (1 g.) every 8 hours.

Gradually the patient's condition started improving. X-ray of chest was repeated on 12th June 1964 (Fig. 4), which did not show much improvement as compared to that of the previous day.

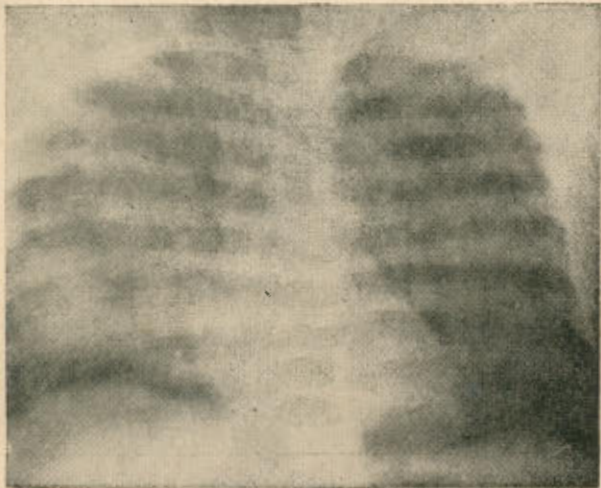


Fig. 3

Same one day after delivery showing diminution of the woolly opacities of both lungs.

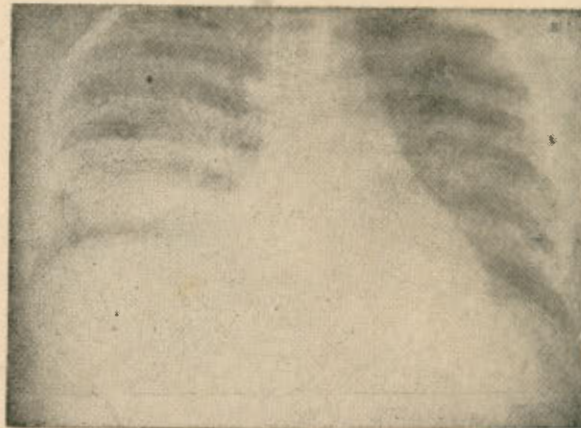


Fig. 4

Same two days after delivery showing lung opacities similar to the previous day.

On 13th June 1964 her general condition had much improved. There was no cyanosis, pulse was 118 per minute, respiratory rate was 32 per minute and blood pressure 105/60 mm of Hg. She had mild pyrexia of 99.6°F (37.5°C). On auscultation of lungs—few rales with rhonchi were heard on the right side only. Skiagram of the chest taken on 13th June 1964 (Fig. 5) showed some clearing up of the woolly patches of the lung fields specially on the left side. The infant was put to breast for the first time.

As the patient's general condition started improving, digitalis, aminophyllin and fovane were gradually omitted in the course of the next two days.

By 18th June 1964, she was very much better and cheerful. Since 13th June 1964 she was regularly feeding her baby. Clinically her chest was clear. X-ray of chest taken on that day (Fig. 6) showed lung fields had become clear on both sides.

Mother and infant were discharged well on 24th June 1964, fourteen days after delivery. At that time her haemoglobin was 13.5 g. per cent.

She was seen 2 weeks after discharge from the hospital; except for slight general weakness she had no other complaints. She was seen again after 2 months and then

after 8 months on 30th April 1965. Both mother and baby were doing well.

Discussion

This case presented the typical clinical picture of amniotic fluid embolism. She was of short stocky build, multigravida, 32 years old, and had been having strong uterine contractions. She was delivered of a large foetus of 7 lb (3.27 Kg.). These features are typical of cases noted by others (Aguillon *et al.* 1962, Shotton and Taylor 1949). There was sudden onset of profound shock, accompanied by dyspnoea, cyanosis and pulmonary oedema, occurring in the second stage of labour. In this case there was no hypo-fibrinogenaemia, though there was slightly prolonged prothrombin time, but there was no unusual haemorrhagic tendency. The shock is regarded as anaphylactoid by some (Eastman 1948), hence the anti-histamine, phenergan, was given liberally. The portal of entry of

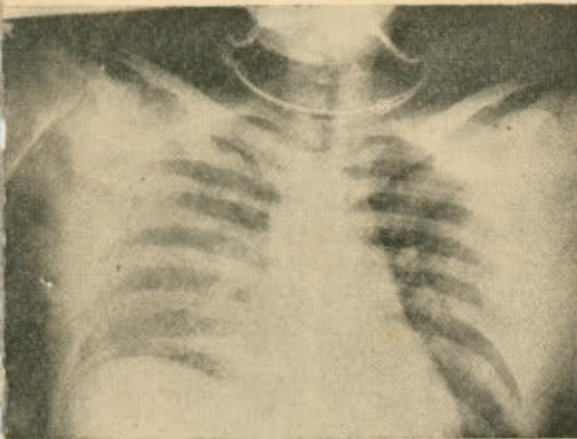


Fig. 5

Same 3 days after delivery, showing some clearing up of the body woolly opacities of lungs specially of the left side.

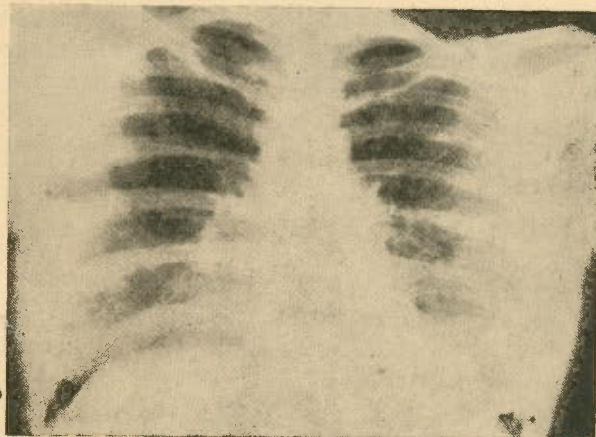


Fig. 6

Same 8 days after delivery. The lung fields have become clear on both sides.

amniotic fluid embolism in this case may be assumed to be through the site of the weak previous caesarean scar, the contributory factors being the strong expulsive effort and the plugging beyond this weak spot by the deeply engaged presenting part.

She was treated with oxygen inhalation, phenergan, digitalis, antibiotics and hydrocortisone in the acute stage. Diuretics and aminophyllin also helped in relieving the congestion of the lungs. Within 8 days her lungs were clear clinically and radiologically.

Summary

A case of amniotic fluid embolism with survival of the mother and child is reported. She presented the typical picture of the syndrome, sudden onset of shock with cyanosis, tachycardia and pulmonary oedema during the 2nd stage of labour with strong expulsive efforts. She made a complete recovery with treatment by oxygen inhalation, digitalis, antihistamine, antibiotics and diuretics. Within 8 days her lungs were clear. The serial chest skiagrams are pre-

sented. Subsequent check up after 8 months revealed no abnormality.

Acknowledgement

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