Amniotic Fluid Embolism

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The purpose of this paper is to report two cases of fatal amniotic fluid embolism occurring in young mothers at this hospital. They happened within a three-month period and represent the only two maternal deaths in the obstetrical department in ten years. This department averages approximately 1,000 deliveries annually. The diagnosis in both cases was confirmed by autopsy findings.

Embolization of the contents of the amniotic sac to the lungs is a rare complication of pregnancy, but may occur at any time after the membranes have ruptured. The incidence is approximately one case in 30,000 deliveries; the number of nonfatal cases is unknown, but these probably outnumber the fatalities.

Amniotic fluid embolism was first recognized by Meyer in 1926, but it was Steiner and Lushbaugh of Chicago who published the first cases in 1941. Previously these had been diagnosed as obstetric shock, idiopathic postpartum uterine atony with hemorrhage and acute pulmonary edema of pregnancy.

Predisposing factors are: uterine tetany, frequent and strong contractions, meconium in the amniotic fluid, intrauterine death of the fetus, an oversized baby, multiparity, and advancing age of the mother.

Clinical signs are: rapid onset of dyspnea, sudden shock, restlessness, tremors or convulsions, marked cyanosis, thready pulse, and hypotension.

Amniotic fluid may enter the general circulation through venous sinuses at the placental site and then circulate to the pulmonary capillaries. Complications such as a ruptured uterus, marginal separation of the placenta, normal separation with partial placental accreta, or Caesarean section keep the venous sinuses abnormally open. A more frequent entrance, however, may be through the endocervical veins. These veins are often lacerated during normal labor and account for the "bloody show." If the membranes rupture before labor begins, amniotic fluid may come into contact with the myometrium.

At autopsy following fatal cases, the capillaries and arterioles of the lungs are filled with a mucin-like substance containing squamous cells, particles of meconium and lanugo hair obstructing the pulmonary circulation. In some cases where mechanical obstruction is not enough to cause death, it has been demonstrated that the presence of foreign bodies in the pulmonary vascular tree produces reflex vasospasm in the unoccluded vessels. In any case, death is due to obstruction of the pulmonary circulation causing acute right heart failure.

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In nonfatal cases amniotic fluid embolism can be demonstrated by squamous cells, meconium, and lanugo hairs being present in a centrifuged peripheral blood specimen. If the patient survives the initial episode, afibrinogenemia accompanied by uncontrolled bleeding may occur. In spite of all supportive measures, the patient usually expires in a few hours. Death is due to irreversible shock, pulmonary edema, or uncontrolled hemorrhage. The hemorrhage is due to incoagulability of the blood, which may be caused by depletion of plasma fibrinogen, destruction of fibrinogen, or release of a heparin-like substance present in the amniotic fluid.

**MANAGEMENT AND TREATMENT**

When embolism occurs, immediate treatment is necessary if the patient’s life is to be saved. She should be placed in slight Trendelenburg position; the respiratory passages should be checked for emesis and oxygen administered to treat the anoxemia and hypoxia. Impending pulmonary edema may be treated by positive pressure ventilation with oxygen. The anesthetist should anticipate excessive postpartum hemorrhage and start intravenous fluids until blood is available. Blood volume must be restored and maintained, but care must be taken not to overload the system causing further pulmonary edema.

Fibrinogen has to be administered as soon as possible to counteract the afibrinogenemia and to restore normal blood clotting. Shnider and Moya state that “Four grams of fibrinogen is usually necessary but possibly three times this amount may be required.”

Some authors point out that oxytocics must be used with caution, as they may precipitate the amniotic fluid embolism syndrome. It is also stated that epinephrine is contraindicated because it may predispose to production of pulmonary edema. Drugs recommended for the treatment of hypotension are: methoxamine, mephentermine, methamphetamine, and ephedrine. Hydrocortisone, 100-200 mg. intravenously, may be useful to combat histaminic reaction. Digitalis and atropine may help to restore the cardiovascular system.

**CASE STUDY I**

A twenty-year-old primipara was admitted to delivery room at 8:55 p.m. on 8-24-64. The cervix was completely dilated following a period of apparently normal labor. Blood pressure was 120/80. Atropine 0.40 mgm. was given at 8:45 p.m. intramuscularly. The membranes had been ruptured artificially during labor at 4:40 p.m. Uterine contractions appeared almost constant.

Nitrous oxide and oxygen 50/50 intermittently was given to the patient for three or four contractions. Due to almost constant hard contractions, she was unable to relax during this period of labor. The patient made the comment that she “felt sick,” but she did not vomit. She experienced a mild generalized convulsion, became unconscious, her pupils started to dilate, and she developed increasingly severe cyanosis. The mouth could not be opened because of trismus, so a nasopharyngeal airway had to be inserted. Oxygen was given by bag and mask. The lungs were difficult to ventilate due to increased resistance. A weak radial pulse was present for a few seconds, then disappeared. No apical pulse could be heard. The patient was intubated with no difficulty; the pharyngeal cavity was dry, no emesis was present, and the cords were open. External cardiac massage was performed for ten minutes with no response. During this period marked distention of the peripheral and neck veins was clearly evident.

A viable baby was delivered with forceps immediately; no resuscitation was required. The patient expired at 9:30 p.m.
Summary of autopsy report: No specific findings found grossly. No evidence of pulmonary aspiration.

Microscopic findings: 1. The pulmonary vessels were distended and in many areas contained accumulations of typical squamous cells. Many of the alveoli were filled with red cells and hyaline edema fluid. 2. Squamous cells were found in veins of adrenals, kidneys and liver. 3. Uterus: Several large sinusoids contained accumulations of squamous cells.

CASE STUDY II

A twenty-year-old white female, weighing one hundred fifty pounds, gravida two, para one, at term was admitted to the hospital at 1 p.m. in good health. Following a diagnosis of false labor, she was discharged. Eight hours later she was re-admitted, having strong frequent contractions, but extremely drowsy. She was given demerol 100 mg. and 0.3 mg. scopolamine at 9:00 p.m.

At 10:00 p.m. a mixture of Penthrane, nitrous oxide, and oxygen was administered with one contraction only; with the next contraction the patient had a mild convolution and appeared cyanotic so the anesthetic mixture was changed to pure oxygen.

A large viable male infant, weighing 10 lbs. 2 ozs., was delivered by low forceps. The infant, though a little slow in crying, responded well to resuscitation. His Apgar score was 6. The placenta, which was delivered a few minutes later, showed evidence of partial separation. Cyanosis was observed even in the exposed tissues around the vagina. At this time the blood pressure reading was 70/50 and the pulse was 100. The blood pressure then rose to 110/80 and the patient's color improved. She was given Methergine 0.2 mg. intramuscularly. Several large clots were expressed from the vagina and the uterus was firm, so the patient was prepared for transfer to the postpartum floor. She continued to be extremely drowsy and several times stated, "I'm so sleepy; it's so hard to keep awake."

Twenty minutes later she became markedly cyanotic again. Her blood pressure fell to 60/40, then to 30/10, and her pulse was rapid and thready. Heavy bleeding was noted and her uterus was soft and flabby. Oxygen 100% was given intravenously. Fibrinogen level was 0.08 gm. An intravenous infusion of 1000 cc. 5% dextrose in water with 10 units of pitocin was started, and then one unit of dextran was substituted. By this time the blood pressure had become inaudible, but an apical beat could be heard. The patient remained conscious and rational, but oxygen did not improve the cyanosis and it was noted that the blood did not clot.

Her condition quickly deteriorated. She became restless, delirious at times, her pupils dilated, and no radial pulse could be felt. Two venotomies were performed, blood was pumped under pressure and fibrinogen was given. After six units of whole blood and ten grams (5 units) of fibrinogen, the fibrinogen level was 0.115 grams percent, so both were continued.

Three and one-half hours after delivery, the patient lapsed into unconsciousness with shallow gasping respirations. No apical beat could be heard and the hemorrhage continued. External cardiac resuscitation was performed; trismus prevented oral intubation so a nasopharyngeal airway was inserted and positive pressure oxygen administered. Resuscitation was successful—an apical beat returned, a radial pulse could be felt, and a lid reflex was present. The severe cyanosis was never corrected.

A few of the first units of blood were cross-matched, later units were not. With the hope that fresh blood would produce clotting, one unit was donated by the attending obstetrician.

A laparotomy was performed in the delivery room under 100% oxygen and the hypogastric arteries were ligated in an attempt to reduce the uterine blood flow. The hemorrhage remained uncontrollable, so a supra-cervical hysterectomy was carried out.

A second cardiac arrest occurred and cardiac resuscitation was restarted. The patient could now be intubated orally. An apical beat returned but soon became weaker. Hemorrhage continued and no evidence of clotting could be observed. The patient died at 3:25 a.m., a little less than five and one-half hours after delivery. A total of eighteen units of whole blood,

*Normal level 0.200 - 0.400 GM %.
fourteen grams of fibrinogen and one unit of dextran had been given. Other drugs used were:

- Neosynephrine drip. 20 mg. in 500 cc. normal saline.
- Neosynephrine 1% (0.5 to 1 mg. intravenously).
- Hypertensin 2.5 mg. intravenously.
- Adrenalin 1-1000 solution 0.1 to 0.2 mg. intravenously.
- Adrenalin 1-10,000 solution 10 cc. in divided doses.


Gross findings: 1. Lungs: Right—only anterior middle lobe was aerated. Left—no portions aerated. Each thoracic cavity contained 300 cc's of dark, liquid blood. 2. Uterus: Supracervical portion—internal surface was ragged and congested. Remaining cervix was large, edematous, and hemorrhagic with the broad ligaments filled with blood.

Microscopic findings: 1. Lungs: Generalized diffuse partial atelectasis; many small arteries were occluded by mucoid material with some squames and intermixed polymorphs; there were numerous syncytial masses in capillaries. 2. Uterus: Hypertrophy of pregnancy. Many venous sinuses were dilated and contained poorly-formed thrombus material with many white blood cells, a few squames, and some mucoid material (the obvious source of amniotic fluid emboli). Cervix showed severe congestion and generally diffuse hemorrhage.


DISCUSSION

1. All literature reviewed states that this complication usually occurs in elderly multipara. These two patients were a twenty-one-year-old primipara and a twenty-year-old gravida two.

J. Am. A. Nurse Anesthetists

2. In both cases the first attempt to intubate was unsuccessful because of trismus.

3. The lungs were difficult to inflate in both patients and increased pressure on the breathing bag was necessary for adequate ventilation.

4. Extreme drowsiness was noted more than an hour before delivery in the second case; in retrospect, this symptom was discussed as being present in the first case, though not noted at the time.

5. Both anesthetists remember checking the gas machine several times to make sure they were administering oxygen and not nitrous oxide, because cyanosis was so sudden and did not improve with oxygen.

6. The literature speculates as to the incidence of unsuspected nonfatal cases. Would it be of value to examine a centrifuged blood specimen for amniotic debris on all maternity patients with unexplained respiratory or circulatory symptoms?

SUMMARY

Amniotic fluid embolism should be suspected in every case of severe shock during labor or the immediate puerperium. It represents the most common cause of death during this period. There are four cardinal signs: 1. respiratory distress, 2. cyanosis, 3. peripheral vascular collapse, 4. coma. In addition, afibrinogenemia with excessive bleeding develops. Urgent treatment includes artificial ventilation with oxygen, vasopressors, blood replacement, and fibrinogen.

Two cases of amniotic fluid embolism have been reported. They occurred in young mothers during a
short period of time in a small obstetrical service. In the first case, death was due to massive catastrophic embolism. Death in the second case was caused by massive uncontrollable hemorrhage due to afibrinogenemia.

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BIBLIOGRAPHY