# Amniotic Fluid Embolism: A Review of the Literature and a Case Report with Recovery

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DOI: http://dx.doi.org/10.5915/17-2_3-12758

**Abstract:**

*Amniotic fluid embolism (AFE) is a rare but catastrophic complication of labor. Its mortality is 86%. Only 39 patients are known to have recovered. A case of AFE with fibrinolysis and renal failure is presented. After treatment with fibrinogen, blood transfusion and dialysis, the patient recovered.*

*The clinical syndrome was first described in 1941. The mean age of patients is 32 years; 88% are multiparas. Benirschke was the first to establish a clinical diagnosis by finding amniotic fluid debris in centrifuged central venous blood, above the white cell layer.*

**Key Words:** Amniotic fluid embolism, placental toxin, prostaglandins, consumption coagulopathy, fibrinolysis, hypofibrinogenemia, placental toxin, disseminated intravascular coagulopathy.

## Introduction

AFE remains the most catastrophic of all the complications of pregnancy and labor. In the words of Courtney,[1] it is "the most dangerous and untreatable condition in Obstetrics." It carries a mortality rate of 86%[2] and remains a major cause of death, accounting for 5 to 20% of all maternal deaths.[1, 3-9] Fortunately, however, it is rare: 1 in 25,000 deliveries.[1] Most obstetricians will probably never encounter such an unusual but urgent situation more than once.

Few survivors have been reported.[10-21] Wasser,[20] in 1979, could find only 25 cases that survived; and Morgan[2] collected 39 cases in 1979. This high mortality combined with the paucity of cases reported in the literature, makes it imperative to report the recovery in the following case.

## Case Report

FAB, aged 26, gravida 3, para 4, was admitted on July 13, 1977, in labor (OH 99372). Her blood group was O Rh +. At full dilatation, she passed out at 4:00 PM and was immediately delivered by forceps, of a live male infant. Post-partum hemorrhage was profuse; the woman, who had been given both oyxtocin and methylergonovine, became livid and her blood pressure unobtainable. She was given 1000 ml of blood; fluids were administered intravenously through two catheters, one in each ankle vein. The PT was 11 seconds (control 10.5), the PTT was 32 seconds (control 35), the platelet count was 53,000, the P02 (room air) was 38mm Hg, the PC02 26mm Hg, the pH 7.32, the bicarbonate 18meq/L. Positive end expiratory pressure resulted in a slight response. She was surgically explored. She barely required any anesthesia beside oxygen. Her uterus was found flabby but not ruptured. She was again given oxytocin and methylergonovine. After the incision was closed, the uterine cavity was packed. She however continued to bleed from the uterus and from needle puncture sites in her cubital fossae. The pulse was 130/minute but the blood pressure was unobtainable; the respiration was 40/min.; she was pale and unresponsive. Her pupils did not react to light. As she was still bleeding per vaginum and from multiple needle sites in her limbs which had large echymotic areas, she was given 11 units of blood, Ringer's lactate, plasma and 14 units of fibrinogen. As she continued to bleed, the uterine packs were removed, oxytocin and methylergonovine were given again. Although the hemoglobin was only 5 Gm % and the fibrinogen 0, she started to improve and the outflowing blood began to clot; the systolic pressure went up to 90mm Hg. However as she was still bleeding, a hysterectomy was performed. Following the operation, she successively developed the following:

1. congestive heart failure which responded to digitalis and diuretics;
2. renal shut-down which responded to multiple peritoneal dialyses;
3. pelvic hematomas which were evacuated;
4. urinary tract infection which was treated with antibiotics;
5. unexplained pyrexia which soon subsided.

The patient was finally discharged on August 22, 40 days after her delivery, in good condition.

## Historical Review

Fetal debris was first described in the maternal pulmonary vessels in 1926.[22]

The clinical syndrome was first described in 1941.[23]

The post mortem diagnosis by right heart blood samples was first reported in 1947.[24]

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The first report of hypofibrinogenemia and increased fibrinolysis was made in 1952.[25]

A review of 60 cases was published in 1962[3] and another of 272 cases in 1979.[2]

**Experimental**

In 1953, the clinical syndrome was reproduced in laboratory animals by the intravenous infusion of unfiltered amniotic fluid.[26] In 1962, isoproterenol was shown to reverse the changes of experimental AFE in sheep.[27] In 1965, the IV injection of filtered amniotic fluid produced a drop in systemic arterial pressure and a rise in pulmonary vascular resistance, but these responses were not as great as those that followed the injection of unfiltered amniotic fluid.[28] In 1967, human amniotic fluid was found to contain prostaglandin PGF2alpha only during labor.[29] In 1972, it was shown that amniotic fluid from pregnant women who were not in labor produced the acute fall of blood pressure in 14% of the animals compared to 70% when amniotic fluid was obtained from women in labor. The same results were obtained by the injection of PGF2alpha.[30] These effects were preventable by aspirin[31] which is a potent inhibitor of prostaglandin synthesis.

In 1974, it was found that the injection of amniotic fluid was not lethal but placental extract was.[32] Infusion of amniotic fluid in dogs produced deficiency in thromboplastin antecedents.[33] Amniotic fluid was shown experimentally to contain an activator of Factor X.[34] The amount of this activator increases in relation to the duration of gestation.[35] There was also marked activation of the fibrinolytic system with consumptive coagulopathy.[36] Amniotic fluid was also shown, in 1974, to have an inhibitory effect on uterine muscle.[1]

**Clinical**

The mean age is 32 years. Most patients (90%) were in labor and 88% were multiparas.[2]

The shock that follows the entry of amniotic fluid into the maternal circulation, usually strikes the normally parturient woman, like thunder out of a blue sky, either during labor or in the immediate postpartum period, or may rarely be delayed as long as 32 hours post-partum.[15]

The shock is sudden and severe; the drop in blood pressure is rapid; tachycardia and cyanosis are soon followed by clinically unexplainable uterine hemorrhage; hyperpnea develops and soon cyanosis turns into pallor and lividity; the patient gasps for air; death is impending. More than a third of the patients die during the first hour after the onset of symptoms.[4] If the patient survives the first hour, severe hemorrhage due to hypofibrinogenemia dominates the picture. The shock is due to pulmonary embolism with all its consequent effects: pulmonary hypertension, acute cor pulmonale, decreased left atrial pressure, decreased cardiac output and peripheral vascular collapse.[37] The hemorrhage, at first uterine, soon becomes generalized. It is due to hypofibrinogenemia or afibrinogenemia which is thought to result from a disseminated vascular coagulation which consumes, in a very short time, all the available fibrinogen.[16, 26]

The renal shutdown that sets in, is thought to be due to the lowered blood pressure and the embolisation of the renal vessels.

**Pathology**

Autopsies have revealed that the branches of the pulmonary artery contain all the constituents of amniotic fluid: epithelial squames, vernix caseosa, mucus and even lanugo hair. These structures have also been found in the vessels of other organs like the head, kidneys, brain, liver, spleen and pancreas.[7]

**Diagnosis**

Since Benirschke[16] first established a clinical diagnosis in 1976 by finding amniotic fluid debris in centrifuged central venous blood above the white cell layer, the search for fetal epithelial squames in blood samples from a central venous pressure line—by simple microscopic examination under polarized light—has become a standard diagnostic procedure[5, 38] that is rapid and pathognomonic.[15] Diagnosis can be confirmed by finding fetal squames in maternal sputum stained with Nile blue.[39]

**Treatment**

Treatment should be rapid and aggressive; it aims at:

1. Maintaining respiration and circulatory efficiency by:
   a. giving oxygen under pressure
   b. using positive end expiratory pressure[15]
   c. judiciously administering adequate fluid, blood and diuretics[15] under hemodynamic monitoring.
2. Combating the bleeding diathesis and coagulopathy with the judicious use of fibrinogen, heparin, macrodex and/or epsilon aminocaproic acid, depending on laboratory deteminations:[37]
   a. Fibrinogen is used in increments of 2 Gm. It has been recently argued that this is an incorrect approach.[8, 40] If fibrinogen is to be given at all, it should be preceded by heparinisation.[41]
   b. Heparin has been suggested[8] and used,[12, 38] in doses of 5,000 to 10,000 units IV, especially after the bleeding is under control.[41]
   c. Epsilon aminocaproic acid is necessary. Seven grams have been recommended[41] to inhibit fibrinolysis. Theoretically this may have a dangerous potential for widespread thromboses.[42]
   d. Macrodex (dextran) has also been suggested[41] to prevent microthrombi.
   e. Fresh whole blood remains the best way of

compensating for the deficiency in essential coagulation factors.[41]

3. Overcoming acute renal failure when it occurs.

Pluronic F-68, an industrial surfactant was shown[43] to restore systemic blood pressure after AFE in rabbits. Doubt has been cast[2] on whether the same result could be obtained in humans and whether this chemical will ever be tried clinically.


**Summary**

AFE is caused by the entrance into the maternal circulation of amniotic fluid with a placental toxin, one or more prostaglandins and an inhibitor of the uterine muscle. This causes a rise in pulmonary vascular pressure, a drop in systemic arterial pressure, a deficiency in thromboplastin antecedants, an increase in an activator of Factor X with marked activation of fibrinolysis and consumption coagulopathy.

The clinical syndrome consists of pulmonary hypertension with acute cor pulmonale, shock with hemorrhage, disseminated intravascular coagulation with hypofibrinogenemia and finally, renal shut down.

Diagnosis is arrived at by finding fetal squames in venous blood or in the patient's sputum.

Treatment consists of assisting respiration and circulation with oxygen, positive end expiratory pressure and the judicious use of fluids, blood, diuretics, fibrinogen, heparin, epsilon aminocaproic acid and macrodex.


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