Amniotic fluid embolism (AFE) is a rare peripartum complication (1 case for every 8,000 to 80,000 deliveries), with a reported mortality rate of 86%. The main clinical signs include cyanosis and shock of sudden onset, pulmonary edema, occasional neurologic signs, and coagulopathies. The diagnosis of AFE, previously made on postmortem examination of the lungs, can now be made in vivo by examination of a blood aspirate from the right heart, which shows lanugo and fetal squame. Causes of death include coagulopathy and “circulatory collapse” of unclear pathophysiology. We report a case of AFE in which severe acute left heart failure with complete resolution was observed.

**Report of a Case**

A 25-year-old woman was admitted for delivery of her first child shortly after the spontaneous rupture of her membranes. Amniotic fluid was clear; her pregnancy was full-term and had been uneventful. She had no known cardiac or pulmonary diseases and complained of no symptoms during the late stages of her pregnancy. A clinical examination performed 5 days before admission was normal (arterial blood pressure: 120/60 mmHg, heart rate: 64 beats/min).

Labor started 5 hours after admission, and oxytocin (5.10⁻⁴ IU·min⁻¹) was administered by iv continuous infusion until the second stage of labor. Between uterine contractions, heart rate was 80 beats/min and arterial blood pressure was 120/65 mmHg. Nine hours after admission, when active expulsive efforts began during the second stage of labor, cyanosis, respiratory distress, and hypotension (systolic arterial blood pressure 70 mmHg, heart rate 155 beats/min) suddenly occurred. Immediate iv administration of thiopental, 200 mg, and succinylcholine, 40 mg, permitted tracheal intubation. Large amounts of frothy pink fluid were aspirated from the trachea. Ventilation was controlled with a fractional inspired O₂ concentration (P₀₂) of 1.0 without positive

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**Left Heart Failure in Amniotic Fluid Embolism**

**PHILIPPE GIRARD, M.D.,† HERVÉ MAL, M.D.,‡ JEAN-FRANÇOIS LAINE, M.D.,‡ PATRICK PETITPRETZ, M.D.,* BERNADETTE RAIN, M.D.,§ PIERRE DURoux, M.D.¶

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Address reprint requests to Dr. Girard.

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end-expiratory pressure (PEEP); peak inspiratory pressures did not exceed 30 cm H2O. The patient received 20 mg of furosemide iv. The administration of thiopental produced no detectable deleterious effect on systolic arterial blood pressure as it increased to 80 mmHg. A normal, alive, male infant was extracted by outlet forceps operation within 10 min of onset of symptoms. The patient was subsequently transferred to the respiratory intensive care unit.

A review of the patient’s record showed no evidence of excessive administration of iv fluids. Body temperature was normal. She was in clinical shock, with systolic arterial blood pressure of 80 mmHg and heart rate of 140 beats/min. Consciousness was normal. Cardiac auscultation revealed a ventricular gallop but no evidence of valvular dys- function. There was no peripheral sign of right heart failure except for mild neck vein distention. Moist rales were heard all over both lung fields. Chest roentgenogram revealed bilateral, symmetric, predominantly perihilar, nonhomogeneous opacities consistent with pulmonary edema and a slightly enlarged heart. The ECG was normal except for sinus tachycardia. Coagulation tests, fibrinogen, platelet count, and other routine laboratory data were within normal values.

The protein content of the abundant tracheal secretions was 31 g/l, the simultaneous value in blood being 56 g/l (ratio = 0.55). Arterial blood gases during intermittent positive pressure ventilation (IPPV) with a FiO2 of 0.5 showed a PaO2 of 83 mmHg, PaCO2 of 28 mmHg, pH of 7.42, and base deficit of 4 mmol/l-1. Hemodynamic measurements obtained from a pulmonary artery-flow directed catheter inserted via a basilic vein are presented in table 1. An aspirate of blood from the pulmonary artery revealed numerous squamous cells and other debris of presumed fetal origin (fig. 1).

Because of the surprisingly low value of cardiac index without major pulmonary hypertension, echocardiography was performed 16 h after the onset of symptoms, confirming the suspected left heart dysfunction (elevated end-systolic volume and decreased fiber shortening), with evidence of cardiomyopathy as manifested by an elevated end-diastolic volume and a normal pulmonary capillary wedge pressure (PCWP) (figs. 2, 3, and table 2). Dobutamine was then given.

During the following 24 hours, the patient’s clinical status remained extremely unstable with several episodes of circulatory collapse necessitating increases in the doses of dopamine and dobutamine up to 30 µg·kg-1·min-1 each. Chest roentgenogram deteriorated, as did arterial blood gases (PaO2 of 48 mmHg during IPPV with a FiO2 of 0.6), but markedly improved with PEEP (up to 15 cm H2O). A second measurement of protein content in the abundant fluid tracheal secretions showed a concentration of 45 g/l.

On the second day after onset, hemodynamic status improved (table 1), as did clinical condition, chest roentgenogram, and arterial blood gases. The flow-directed pulmonary artery catheter was removed because of elevated temperature (39°C).

On the next day, an endomyocardial left ventricle biopsy was performed: after local anesthesia with lidocaine, a King catheter was inserted in left femoral artery and retrogradely positioned in the left ventricle; a fibreoptic bronchoscopic biopsy forceps then was introduced through the catheter; and the biopsies were performed under fluoroscopic control. Left ventricular end-diastolic pressure (LVEDP) measured during this maneuver by direct connection of the King catheter to a pressure transducer was 18 mmHg (end-expiratory pressure during IPPV). Anatomical examination of myocardial fragments showed no evidence of myocarditis or degenerative cardiomyopathy, and the specimens cultured for viruses remained negative.

Subsequent evolution was excellent and permitted progressive weaning from supportive drugs. A second echocardiogram performed 5 days after admission to the intensive care unit showed clear improvement in left heart function (table 2), and a discrete pericardial effusion. Exudation of the trachea was possible 6 days after admission because chest roentgenogram (clear lungs, normal size of the heart), and blood gases while breathing room air were normal. ECG showed nonspecific, diffuse repolarization changes. A repeat echocardiogram performed

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<th>Conditions</th>
<th>H + 5 hours</th>
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<td>Dobutamine</td>
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H = hour of onset of symptoms; RAP = right atrial mean pressure; PAP = pulmonary artery mean pressure; PCWP = pulmonary capillary wedge mean pressure; CI = cardiac index; SBP = mean systemic blood pressure; HR = heart rate; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance; PEEP = positive end-expiratory pressure.

All inotropic pressures (RAP, PAP, and PCWP) were measured during the end-expiratory phase of the respiratory cycle.

10 days after admission was normal (fig. 3; table 2). The patient was discharged 18 days after admission in excellent clinical condition except for mild dyspnea on exercise. Treatment consisted of digoxin, 0.125 mg·day-1.

One month later, clinical examination, chest roentgenogram, arterial blood gases, and echocardiography were normal. The ECG had returned to normal. Digoxin was discontinued.

**DISCUSSION**

The commonly accepted mechanism for pulmonary edema in AFE is increased permeability of the damaged alveolar–capillary membrane. The reduced cardiac output and decreased systemic blood pressure have been attributed to an increased pulmonary vascular resistance.1-5 Associated transient left heart failure, a potential explanation of the observed hemodynamic disorders, has been described in four recent reports,6-9 but proven in only one with mildly depressed left ventricular function.6 We report here the first case of AFE in which unequivocal data demonstrating a contemporary left heart failure could be obtained.

In our case, the diagnosis of AFE was made on the association of a typical clinical presentation (sudden shock, cyanosis, hypotension, and pulmonary edema) with demonstration of amniotic fluid debris in the pulmonary vessels (fig. 1). However, the protein content of edema fluid,7 radiologic changes, and hemodynamic data, all consistent with hemodynamic pulmonary edema, led us to suspect associated left heart failure. Subsequent echocardiography showed elevated end-systolic volume and decreased fiber shortening (fig. 3; table 2), which confirmed severe left
heart dysfunction. The elevated LVEDP found 2 days later was further proof.

The association of a high end-diastolic volume with a normal PCWP suggested a compliant left ventricle. However, PCWP might underestimate LVEDP under conditions of decreased ventricular compliance, such as acute myocardial infarction. Such a decreased ventricular compliance might be suspected here because there was a significant discrepancy between PCWP and LVEDP, the latter being significantly elevated.

The severity of myocardial impairment, not previously described in AFE, initially suggested peripartum cardiomyopathy, in spite of an atypical clinical presentation. The absence of any prepartum symptoms, the normal myocardial biopsy, and the rapid return to normal myocardial function evaluated on clinical, radiologic, electrocardiographic, and echocardiographic features virtually eliminated this diagnosis. The absence of well-documented left heart failure in all but one other published report of AFE can be explained in several ways: 1) like other manifestations, such
as disseminated intravascular coagulation or neurologic signs, left heart failure could be inconstant in AFE; 2) the hypothesis of increased permeability pulmonary edema, supported by the protein content of edema fluid in a few reported cases, was accepted without exploration of left ventricle function, and 3) when present, such severe left heart failure was probably fatal within a few hours before the common use of dopamine and dobutamine and the extensive use of mechanical ventilation with PEEP. All these reasons might explain the almost complete lack of sequential evaluation of myocardial function in AFE.

The correct explanation for the left heart failure described in our patient remains to be found. It does not appear to be related to mechanical ventilation-induced myocardial dysfunction, since initial hemodynamic and echocardiographic data were obtained without end-expiratory pressure. Myocardial hypoxia as a cause of myocardial dysfunction also seems improbable in our patient because gas exchanges were only slightly altered. The normal myocardial biopsy virtually eliminated "degenerative" or "inflammatory" cardiomyopathy. Therefore, the direct or indirect effect on the cardiac muscle itself of potent vasoactive substances, such as prostanoids present in the normal amniotic fluid, previously suspected by others, appears a potential explanation for at least some of the hemodynamic consequences of AFE.

Severe, acute left heart failure with complete gradual recovery was observed in a patient with AFE. This myocardial dysfunction constitutes a possible partial explanation for both the hypotension and the pulmonary edema commonly observed in AFE. It requires early intensive therapy with vasoactive and positive inotropic drugs associated with symptomatic treatment. A systemic assessment of left heart function in patients with AFE (e.g., by echocardiography) might improve both the management of such patients and the understanding of hemodynamic changes.

### References


![Fig. 3. Cross-sectional echocardiography. Short axis views of the left ventricle during diastole (A) and systole (B). Ten days after admission, there is a dramatic improvement in left ventricle dimension and function.](image-url)