

Amniotic fluid embolism

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To the editor: Amniotic fluid embolism (AFE), first described in 1926, is a rare, catastrophic complication of pregnancy and labor. Mortality remains high, despite aggressive therapeutic interventions [1,2]. We report a case of a possible AFE, in a patient in whom ulinastatin and gabexate mesilate appeared to be effective in the treatment of disseminated intravascular coagulation (DIC).

A 32-year-old secundigravida was admitted to our labor and delivery unit at 38 weeks of gestation. Forty-five min after rupture of the amniotic membranes, the patient suddenly lost consciousness, and her blood pressure could not be measured. Intermittent positive pressure ventilation of the lungs and external cardiac massage were instituted. Her trachea was intubated. Circulation was managed by the infusion of fluid, and the administration of drugs, including ephedrine, epinephrine, dopamine, and NaHCO₃, and cardiac massage. The resuscitation had to be continued for 30 min until her blood pressure was measurable. After the resuscitation, an emergency Cesarean section was performed while she was on inotropes and mechanically ventilated without anesthesia. However, the fetus was not saved. Neither utero-cervical ruptures nor lacerations were found. Her central venous pressure was 6 mmHg at the time of the Cesarean section. She was transferred to the intensive care unit (ICU), where good urine output was documented at all times. She developed DIC (fibrin degradation products, >40 mg/ml [normal, <10 mg/ml]; partial thromboplastin time, undetectably long [normal, <35 s]; prothrombin time, 40.3 s [normal, <14 s]; fibrinogen, 0.9 g/l [normal, >1.7 g/l]; and platelet count decreased to 42,000 cells/ml] and she bled vaginally, orally, and from the surgical suture. Immediately, 10 units of concentrated red cells, 14 units of fresh frozen plasma, and 20 units of platelet concentrates were transfused. Ulinastatin (200,000 units) and antithrombin (AT)-III (2000 units) were administered, and continuous infusion of gabexate mesilate was started (60 mg/h). About 8 h after the cardiopulmonary arrest, the

bleeding had completely stopped. Pulmonary edema was not seen. For brain protection, Glyceol and methylprednisolone sodium succinate were used. However, the patient was left with severe diffuse neurological impairment, despite having received 30 sessions of hyperbaric oxygen therapy. Electroencephalogram showed burst and suppression pattern.

The primary defect created by an AFE is presumed to be mechanical blockage of part of the pulmonary arteries and vasoconstriction of the remaining vessels, perhaps through the release of undefined chemicals such as prostaglandins, leukotrienes, serotonin, endothelin, or histamine. Pulmonary artery pressure increases, resulting in cardiac arrest. In the present patient, central venous pressure was not elevated 2 h after the cardiac arrest. The increase in the pulmonary pressure may have been very transient, although the DIC persisted for much longer.

Complex effects on coagulation have been reported in the 40% of patients who do not die of cardiovascular collapse. A hemorrhagic tendency is one of the main clinical features seen. Liu et al. [3] showed, using a Thrombelastograph (Haemoscope, Skokie, IL, USA), that amniotic fluid accelerates clot initiation and propagation. In our patient, ulinastatin, AT-III, and gabexate mesilate were used instead of heparin, although we cannot rule out the possibility that the very large amounts of blood components used, rather than these agents, were effective. No sign of thrombosis was seen and DIC did not lead to multiple organ failure.

References

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