Amniotic Fluid Embolism with Haemostasis Complications: Primary Fibrinogenolysis or Disseminated Intravascular Coagulation?

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Key Words
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Abstract
Amniotic fluid embolism (AFE) is characterized by the passage of amniotic fluid (AF) into the maternal circulation during or just after childbirth. AFE is a rare disorder occurring in 1/8,000 to 1/80,000 deliveries but with a maternal morbidity ranging from 26% in a recent report to 86% in earlier ones [1–3]. In patients who survive, AFE may affect coagulation resulting in severe bleeding [3, 4]. While disseminated intravascular coagulation (DIC) is usually seen in such cases, we reported a case of AFE in which the hemostatic abnormalities were compatible with primary fibrinogenolysis rather than with DIC.

Discussion
The distinction between primary fibrinogenolysis and DIC with secondary fibrinolysis is often difficult to draw. It is complicated by the changes in the coagulation and fibrinolytic systems during normal pregnancy. However, the absence of clinical evidence of systemic thrombosis (hepatic and renal tests remain normal) and some biological data may suggest predominant fibrinogenolysis: a normal platelet count, a very low fibrinogen level and factor V level in contrast to normal factor II, VII, X ones, the absence of soluble fibrin, a high level of fibrin degradation products 150 μg/ml (<10), D-dimer 20 μg/ml (<0.5), tissue plasminogen activator (t-PA) 35 ng/ml (1–12), and plasmin inhibitor (previously α2-antiplasmin) <5% (70–120). These values were quite similar to another blood sample drawn after 20 min except for the hemoglobin which fell to 5.2 g/dl. The following diagnoses were excluded: anaphylaxis, septic shock, pulmonary embolism, preeclampsia, HELLP syndrome, placenta previa or retroplacental hematoa. Therefore the most likely diagnosis was AFE. A symptomatic management was instituted including fresh frozen plasma (4 units), packed red cells (4 units) and fibrinogen concentrate infusion. A cesarean section was performed. However, an hemostatic hysterectomy was required to stop the bleeding. The post-operative course was uneventful. The woman survived without any sequelae while the neurological prognosis for the child remained uncertain. All the hemostasis parameters were within the normal range when the patient was discharged 7 days later.

Case Report
A 30-year-old G1P1 woman was admitted at 37 weeks and 5 days of gestation because of a premature rupture of membranes. Suddenly during labor, she presented with dyspnea and hypotension followed within minutes by cardiorespiratory arrest. The laboratory results were: hemoglobin 9.6 g/dl (normal values not taking pregnancy into account: 12–16), platelets 115 g/l (150–400), prothrombin time 43% (80–100), fibrinogen 0.3 g/l (1.9–4), factor II 62% (70–140), factor V 6% (60–140), factor VII 146% (70–140), factor VIII 31% (50–150), antithrombin 57% (80–130), absence of soluble fibrin, fibrin degradation products 150 μg/ml (<10), D-dimer 20 μg/ml (<0.5), tissue plasminogen activator (t-PA) 35 ng/ml (1–12), and plasmin inhibitor (previously α2-antiplasmin) <5% (70–120). These values were quite similar to another blood sample drawn after 20 min except for the hemoglobin which fell to 5.2 g/dl. The following diagnoses were excluded: anaphylaxis, septic shock, pulmonary embolism, preeclampsia, HELLP syndrome, placenta previa or retroplacental hematoa. Therefore the most likely diagnosis was AFE. A symptomatic management was instituted including fresh frozen plasma (4 units), packed red cells (4 units) and fibrinogen concentrate infusion. A cesarean section was performed. However, an hemostatic hysterectomy was required to stop the bleeding. The post-operative course was uneventful. The woman survived without any sequelae while the neurological prognosis for the child remained uncertain. All the hemostasis parameters were within the normal range when the patient was discharged 7 days later.

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products, and a very low plasmin inhibitor level with high values of t-PA. Antithrombin levels decreased slightly during pregnancy reaching values published in the literature ranging from 40 to 132% during the third trimester [5].

The mechanisms by which the AFE could have been associated with primary fibrinogenolysis remain to be elucidated. The potential of the AF to induce coagulation is linked to the presence of a functionally active tissue factor and a factor X activating substance [6, 7]. But AF also contains a t-PA, a urokinase-like plasmin activator which can enhance fibrinolysis [8]. Using thromboelastograph analysis, Liu et al. [9] found a tendency toward increased lysis with the addition of full-term AF, though this was not significant. Moreover a recent study has found that all patients suffering from premature membrane rupture with confirmed intrauterine infection and two thirds of those without infection showed an elevated AF tumor necrosis factor-α (TNF-α) and interleukins (IL-1, IL-6) [10]. This is of interest since TNF-α may be considered as a major factor for triggering fibrinolysis [11].

In conclusion, AFE is a rare and dramatic obstetric complication associated with coagulopathy. Nevertheless, generalized fibrinolysis was the predominant feature; quick recognition of the event and aggressive treatment may enable survival.

References