nant woman may, beside the risk of bleeding, interfere with performing a cesarean delivery should it be indicated upon arrival at the hospital. For these reasons, we think that pre-hospital thrombolysis should be considered in pregnant women with suspected massive PE only during cardiopulmonary resuscitation (CPR) for cardiac arrest.

The second issue is the use of extracorporeal life support (ECLS) for PE. Extracorporeal life support should be considered in cases of massive PE associated with persistent severe hypoxemia and cardiovascular failure, either as a bridging measure to maintain vital signs until definitive therapy (thrombolysis or embolectomy) is implemented or when definitive therapy fails or is contraindicated. In addition, ECLS represents a rescue definitive therapy (thrombolysis or embolectomy) is either as a bridging measure to maintain vital signs until persistent severe hypoxemia and cardiovascular failure, be considered in cases of massive PE associated with port (ECLS) for PE. Extracorporeal life support should only during cardiopulmonary resuscitation (CPR) for cardiac arrest.

We report the case of a woman with amniotic fluid embolism (AFE)-induced cardiac arrest, with coexisting hemorrhagic shock, who was successfully treated by veno-arterial extracorporeal membrane oxygenation (VA ECMO).

A 35-year-old, gravida 1 para 0 woman, at 39 weeks of gestation, was admitted to the hospital because of a fetal death. Labor was induced with misoprostol. Her investigations before delivery were normal: s. hemoglobin 12.9 g/dL, platelet count 251 × 10⁹/L, prothrombin ratio 100%, activated thromboplastin time 34.4 s (control to 32 s), and s. fibrinogen 3.67 g/L. The anesthesiologist provided epidural analgesia, with no aspiration of cerebrospinal fluid or blood from the epidural catheter.

Four hours later, the woman suffered loss of consciousness, with stertorous breathing and hypoxia (oxygen saturation 80%). The epidural analgesia was suspended, high-flow oxygen administered and the patient transferred to the operating room. She developed hypotension (blood pressure 92/51 mmHg) and tachycardia (121 beats/min). An overdose of local anesthetic was suspected and lipid emulsion was infused. She recovered consciousness, was hemodynamically stable and commenced to push. The maternal effort was ineffective because of a high cephalic fetal presentation.

At that point, the laboratory alerted the medical team that she had disseminated intravascular coagulation, with a platelet count of 68 × 10⁹/L (150–450 × 10⁹/L), prothrombin ratio <10% (70–100%) and s. fibrinogen <0.4 g/L (3.4–6.4 g/L). Amniotic fluid embolism was suspected. A cesarean section under general anesthesia commenced and was complicated by a postpartum hemorrhage leading to hemorrhagic shock. The patient received a massive transfusion, oxytocin and sulprostone infusions, and surgical hemostatic procedures. Despite these interventions, a cardiac arrest occurred, but this was treated successfully by external cardiac massage and intravenous epinephrine. The bleeding persisted and a hysterectomy was required. The total blood loss was estimated to be 5 L. Restoration of normal hemostasis required 16 units of concentrated red blood cells, 14 units of fresh frozen plasma, 20 units of platelets, 6 g of fibrinogen, 3 g of tranexamic acid, 3 g of calcium gluconate and 6 mg of activated factor VII. To assist with hemodynamic stabilization, a continuous intravenous infusion of norepinephrine was needed.

The patient was transferred to the intensive care unit (ICU) of the nearest university-affiliated hospital center. On arrival, a computerized tomography scan demon-
strated a distal right-sided pulmonary embolus. Transthoracic echocardiography showed acute cor pulmonale. Her hemodynamic status remained in a precarious state, with recurrence of hemorrhagic shock and further blood product transfusion. The patient showed signs of acute respiratory distress syndrome. After discussion between the anesthesiologists, intensivists and cardiologists, and because of respiratory failure and acute right ventricular dysfunction, VA ECMO was undertaken (femoro-femoral cannulation). We chose a "heparin-free" strategy for the first 48 h after implementation of ECMO because of the coagulopathy. Once the bleeding had been controlled, low-dose heparin (800 IU/h) was started, to achieve an activated clotting time >150 s. The ECMO cannulas were removed on day four, after the resolution of acute respiratory distress syndrome and with adequate right-heart function, documented by echocardiography. Extubation was possible on day 10. Thirty-nine days after the onset of AFE, the patient was transferred from the ICU to the obstetric ward. Anucleate squames and a single hair found on a maternal blood smear taken on day zero, associated with the clinical situation, supported the diagnosis of AFE. In AFE, CT imaging may show multifocal bilateral areas of ground-glass opacity that are indistinguishable from other causes of acute pulmonary edema, interlobular septal thickening and pleural effusion. However, microthrombi and thrombotic sediments have been demonstrated in some pathologic examinations of the lungs of AFE victims. There are also reports of patients with co-presentation of thrombotic pulmonary embolism. Nevertheless, we cannot exclude a thromboembolic event, particularly due to the infusion of rFVIIa.

Few reports describe the successful use of extracorporeal life support to manage cardiac arrest or refractory circulatory failure secondary to AFE. To the best of our knowledge, none describes the effectiveness of ECMO in a case of AFE with coexisting hemorrhagic shock. We chose to implement VA ECMO urgently, to provide circulatory and respiratory support, despite disseminated intravascular coagulation. Normally, anticoagulation is mandatory with ECMO to avoid thrombosis of the extracorporeal circuit and to maintain patency. Exposure of blood to the artificial surfaces of the circuit leads to an inflammatory response and initiation of the coagulation pathway. We avoided heparin by using a heparin-bond circuit and maintaining a high ECMO blood flow (>3.5 L/min) to reduce the risk of thrombosis. This strategy has been described in a case of post-traumatic hemorrhagic shock.

The application of ECMO after AFE is challenging. Our experience suggests that this therapy should be considered promptly in AFE, when available, for life-saving cardiac and respiratory support, even if a bleeding diathesis is present.

References


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It’s not easy being green, or is it? Alkaline battery versus A/C power for programmed intermittent epidural bolus pumps

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