Amniotic fluid embolism (AFE) is a rare catastrophic complication of pregnancy; the estimated incidence is 1 in 8000 to 1 in 80,000 pregnancies. AFE is associated with a mortality rate of 60% to 86%. Paradoxical embolism is a pathological condition in which emboli originating from the venous system reach the arterial circulation via the right-to-left shunt. Occasional reports of clot observed moving through the patent foramen ovale (PFO) have been published. Such movement has been described previously for cases of venous thromboembolism. We were not able to find any reports of paradoxical embolization complicating AFE. Herein, we report one such rare case.

Case Report

A 34-year-old woman (gravida 3, parity 1, miscarriage 1) was admitted for labor augmentation because of overdue delivery, 1 week after the expected date of delivery. Her first pregnancy had been complicated by mild pregnancy-induced hypertension, but a healthy female baby was delivered. The patient was initially given intravenous oxytocin 6 mU/min, and the labor progressed to 6-cm cervical dilatation with 90% effacement. Oxytocin infusion was later withheld because of an episode of variable deceleration of the fetal heart. Thirty minutes later, the patient had spontaneous rupture of the membranes,
and the cervix was dilated to 9 cm. Five minutes later, she became unresponsive with hypotension (undetectable blood pressure) and bradycardia. “Code blue” was called and she was resuscitated with oxygen, bolus intravenous fluids, and chest compressions and was urgently intubated. She was immediately taken to the operating room for emergency cesarean section. At that time, her heart rate was 50/min to 70/min and her blood pressure was 100/65 mm Hg.

A live male infant was delivered by emergency cesarean section, which was complicated by marked intraoperative bleeding. The neonate’s birth weight was 3.87 kg and the Apgar scores were 1 at 1 minute, 4 at 5 minutes, and 6 at 10 minutes. Analysis of umbilical cord blood revealed a pH of 6.8, $\text{PCO}_2$ of 133 mm Hg, $\text{PO}_2$ of 15 mm Hg, and a bicarbonate level of 21.2 mmol/L. The newborn baby was subsequently transferred to the neonatal intensive care unit. After cesarean section, the mother’s heart rate was 80/min and her blood pressure was 144/92 mm Hg.

During the cesarean section, immediate intraoperative consultations from cardiology, cardiothoracic surgery, intensive care, hematology, and maternal and fetal medicine services were requested. An intraoperative transesophageal echocardiogram showed a large clot in the right atrium passing through the PFO to the left atrium (see Figure). Severe right ventricular dysfunction also was noted. The mother was also thought to be at high risk for systemic embolism. Immediately after completion of the cesarean section, she was taken for cardiopulmonary bypass surgery. During the bypass surgery, a clot was noted in the right and left atria and also in the PFO. The PFO was opened and the clot was completely removed. The pulmonary artery was also explored, but no clot was noted there. Then, the PFO was closed with sutures.

After the bypass surgery, the patient remained in the surgical intensive care unit for a few days. Because she had marked bleeding due to DIC and because of her 2 major surgical operations, treatment with anticoagulants was started on postoperative day 4. Subcutaneous heparin 5000 units 3 times a day was initiated on postoperative day 4, and then she was given warfarin 10 to 12 mg/d to keep the international normalized ratio between 2.0 and 3.0 for 6 months. The patient gradually recovered and was discharged from the hospital 10 days after the operations. She had no short- or long-term sequelae.

Pathological examination of the term placenta showed calcification near the decidua basalis with unremarkable villi. The membranes contained pigment-laden macrophages consistent with meconium exposure. The 3-vessel umbilical cord showed perivascular hemorrhage. Pathological examination of the intracardiac thrombus revealed early organizing thrombus that contained a few squamous cells that were positive for cytokeratin, consistent with AFE.
Comment

Amniotic fluid embolism (AFE) can occur only when a break is present in the barrier between the amniotic fluid and the maternal circulation. The pathogenesis of AFE is still not clearly understood. The few available reports of transesophageal echocardiography, performed shortly after embolization, universally describe a marked right ventricular failure. This response appears to be caused not only by the pulmonary embolism but also by vasoconstriction produced by vasoactive substances released when amniotic fluid enters the pulmonary circulation. Interplay among left ventricular dysfunction, acute lung injury, and activation of clotting factors is suspected. Fetal components, such as squames from the skin, mucin from the gut, lanugo hairs, and occasionally meconium, are found in the amniotic fluid. The fluid has factor X–activating properties and a high procoagulant activity that increases with gestational age.

AFE remains one of the most devastating complications of pregnancy. The diagnosis is difficult to make because it is mostly a diagnosis of exclusion. Management of AFE is essentially supportive. In our case, the patient made an uneventful recovery without any neurological sequelae and with complete normalization of cardiac function. Although cardiac surgery was performed for extraction of the paradoxical embolus-in-transit, the cardiopulmonary bypass surgery may have facilitated this patient’s recovery.

This case also brings up a question that has been previously raised but inadequately addressed. DIC is one of the common potential complications of this condition, probably because of the presence of direct factor X–activating factor and thromboplastin (or tissue factor), which may initiate the clotting cascade. Published reports offer no firm recommendation about the management of thromboembolism in the context of severe bleeding in DIC. Recommendations on the management of the thrombosis and coagulopathy are limited by the absence of controlled trials. DIC signifies an ongoing tug-of-war between hemorrhage and thrombosis. Many patients do not require specific therapy for the coagulopathy associated with DIC, either because it is of short duration or because it is not severe enough to be a major risk for bleeding or thrombosis. Although the possible need for treatment with anticoagulants in cases of thromboembolism during amniotic fluid embolism has been brought up, and in rare cases such treatment has been initiated, no established guidelines are available. Once a diagnosis of DIC has been established as a component of the varied clinical spectrum of AFE, we are obliged to address this question in addition to initiating supportive measures. The administration of heparin is generally limited to patients with chronic compensated DIC who have predominantly thrombotic manifestations.

We opted to treat this patient with anticoagulants for 6 months, initially with heparin and then with warfarin. We based our decision on the presence of an organizing thrombus on the extracted clot and on the established increased thrombotic risk associated with a postpartum state. Furthermore, our literature search revealed a few cases of AFE successfully managed with cardiopulmonary bypass and pulmonary artery thromboembolectomy. Thus, this case illustrates the feasibility of cardiopulmonary bypass surgery and thromboembolectomy in the management of AFE complicated by intracardiac or pulmonary embolism.

FINANCIAL DISCLOSURES

None reported.

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