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Temporary Extracorporeal Circulatory Support and Pulmonary Embolectomy for Catastrophic Amniotic Fluid Embolism

Michael S. Firstenberg, MD,[1] Erik Abel, PharmD,[1] Danielle Blais, PharmD,[1] Katja Turner, MD,[2] Mona Halim-Armanios, MD,[2] Galina Dimitrova, MD,[2] David Cohn, MD,[3] Philip Samuels, MD[3]

[1]Division of Cardiac Surgery, [2]Department of Anesthesiology, and [3]Department of Obstetrics and Gynecology, The Ohio State University Medical Center, Columbus, Ohio, USA

ABSTRACT

Amniotic fluid embolism is usually a life-threatening complication of an otherwise healthy pregnancy. Medical management of the coagulopathy and cardiovascular collapse is challenging and is often unsuccessful. We present a case and advocate the use of temporary circulatory support and pulmonary embolectomy in what would otherwise have been a fatal scenario.

INTRODUCTION

Amniotic fluid embolism (AFE), a rare and catastrophic event, remains a formidable problem throughout the world as a significant cause of maternal morbidity and mortality. AFE is believed to occur in 1 of 8000 to 20,000 live births and has been associated with a maternal mortality rate of 60% to 80%, with half of the survivors sustaining permanent neurologic complications [Miller 2007]. Similar fetal outcomes have also been reported. Despite a greater recognition of the problem and a better understanding of the pathophysiology, treatment is typically supportive of the complex constellation of problems, which sine qua non include respiratory failure, hypoxemia, acute right heart failure, and severe disseminated intravascular coagulopathy. We report a case of overwhelming AFE successfully treated with temporary circulatory support that allowed hemodynamic stabilization and management of the severe coagulopathy.

CASE REPORT

Our patient is a 36-year-old, otherwise healthy, gravida 5, para 2-0-2-2. She presented at 40 weeks gestation for a labor augmentation secondary to polyhydramnios with an unstable fetal lie in early labor. Because of painful contractions, an epidural catheter was placed without difficultly. Several minutes later, the patient collapsed and had no detectable blood pressure. Fetal bradycardia was apparent at 50 beats per minute. She was taken for emergent cesarean delivery. Following rapid preparation of the abdomen and a lower transverse incision, the baby was delivered. Apgar scores were 1 and 3 at 1 and 5 minutes, respectively, with cord pH values of 7.04 (venous) and 6.99 (arterial). Following closure of the uterus and the abdominal incision, a Bakri balloon (Cook Medical, Bloomington, IN, USA) was placed to control the profuse uterine bleeding. She received simultaneous cardiopulmonary resuscitation and intravenous administration of vasoactive medications. Misoprostol, oxytocin, and methylergonovine were administered but produced minimal improvement in the uterine bleeding. The unstable cardiovascular status prompted a transesophageal echocardiography evaluation, which revealed a large thrombus in the main pulmonary artery, with severe right ventricular (RV) dilation and dysfunction (Figure). The following arterial blood measurements were made: pH, 7.0; PCO_2, 53 mm Hg; PO_2, 148 mm Hg; lactate, 11 mg/dL. A hematology profile showed the following: fibrinogen, <65 mg/dL; international normalized ratio, >15; thrombin time, >120 seconds; partial thromboplastin time, >180 seconds; prothrombin time, >130 seconds. Following sternotomy and heparinization (300 U/kg), standard aorto–right atrial cardiopulmonary bypass (CPB) was initiated. The pulmonary arteries were explored, and a gelatinous clot was evacuated. The patient also had evidence of intra-abdominal hemorrhage and compartment syndrome. While the patient was on bypass, we reexplored her abdomen to evacuate her hematoma and to better control her uterine bleeding via surgery. Once these tasks were accomplished, the abdomen was closed. The patient was then weaned from bypass with moderate pharmacologic support (milrinone, 0.125 µg/kg per minute; vasopressin, 0.04 U/hour; and Levophed, 0.03 µg/kg per minute), empiric inhaled nitric oxide (40 ppm), and protamine sulfate to reverse her heparinization. The total bypass time was 127 minutes. Within 48 hours, she was weaned off all vasoactive medications and nitric oxide. After aggressive diuresis following her massive intraoperative transfusion (46 units of blood, 30 units of fresh frozen plasma, 6 packs of platelets, 6 units of cryoprecipitate, and 4 mg recombinant factor VIIa), the patient was extubated

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Correspondence: Michael S. Firstenberg, MD, Division of Cardiac Surgery, N817 Doan Hall, 410 W 10th Ave, Columbus, Ohio 43210, USA; 1-614-366-7414; fax: 1-614-293-4726 (e-mail: Michael.firstenberg@osumc.edu).

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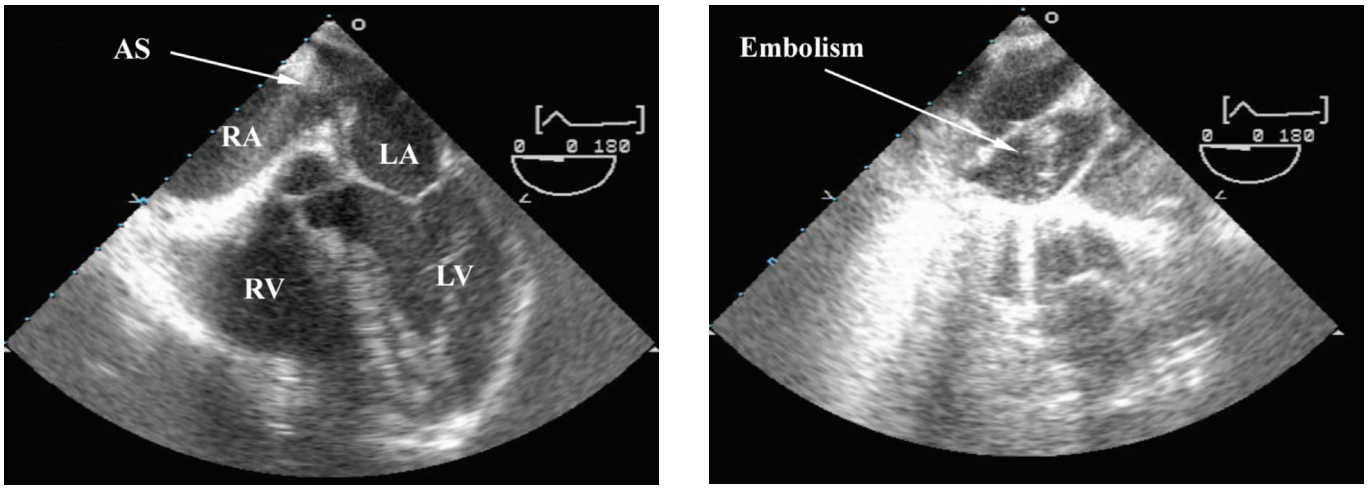

A, Four-chamber view showing a dilated right atrium (RA), a dyskinetic right ventricle (RV), bowing of the atrial septum (AS), and underfilling of the left ventricle (LV) consistent with RV failure and pulmonary hypertension. LA indicates left atrium. B, Further imaging shows what appears to be fresh "embolism" in the main pulmonary artery.

on postoperative day 6. The results of postoperative venous duplex scanning of the upper and lower extremities were unremarkable. Despite brain magnetic resonance imaging results that showed a small posterior fossa infarction, she had no detectable neurologic deficit. On postoperative day 21, the patient was discharged home. At 6 months postoperatively, her PaO_2 in room air was 89 mm Hg, her pulmonary function testing and diffusion capacity results were normal, and a transthoracic echocardiogram showed an improved RV function, a moderate tricuspid function, and an estimated RV systolic pressure of 29 mm Hg. Both mother and baby are well.

DISCUSSION

AFE was first associated with maternal-fetal mortality by Meyer [1926]. Despite extensive research and clinical experience, it is still associated with considerable maternal and fetal morbidity and mortality. Amniotic fluid contains a variety of vasoactive cytokines, including thromboxane and bradykinin, as well as procoagulants, such as platelet-activating factor and arachidonic acid. Even small amounts of amniotic fluid can produce intense pulmonary vasoconstriction and acute right heart (and subsequently left heart) failure [Benson 2007]. Simultaneously, a profound disseminated intravascular coagulopathy usually precipitates uncontrolled hemorrhage from the highly vascular, engorged, and recently gravid uterus [Clark 1995]. The challenge is to successfully address these 2 complex and catastrophic problems.

The hemodynamic instability is treated with vasopressor and inotropic support while blood and blood products are used to control the coagulopathy and bleeding. Use of recombinant factor VIIa and antifibrinolytics has also been described but is associated with further thrombotic complications [Gist 2009]. In severe cases, however, rapid progression to further end-organ damage, shock, and hemorrhage can lead to an ongoing and potentially refractory physiological deterioration—and death. As we experienced, and advocate, early recognition of AFE with implementation of temporary circulatory support can assist in the management of these problems.

Because of the acute nature of AFE and cardiovascular collapse, immediate fetal delivery can be ongoing during simultaneous maternal resuscitation and, as in our case, preparation for pulmonary embolectomy and CPB. CPB and pulmonary embolectomy have each been described previously [Esposito 1991], as has the use of extracorporeal life support and RV assist devices [Nagarsheth 2008; Ho 2009]. The unique application in our case was not only to support the hemodynamics, correct the acute metabolism acidosis, and stabilize the acute pulmonary hypertension and RV failure, but also to allow for greater maternal stability during the acute postcesarean intra-abdominal hemorrhage. While having complete extracorporeal control of oxygenation and flow, we were able to resuscitate the patient actively and rapidly with blood and blood products. This control also permitted abdominal reexploration and technical control of the bleeding. Furthermore, unlike previous cases in which mechanical support was used, we pursued therapy immediately, thereby limiting the potential long-term complications of pulmonary hypertension and RV dysfunction [Nagarsheth 2008].

Although extracorporeal support can be obtained via peripheral cannulation, control and cannulation of profoundly vasoconstricted femoral arteries is very difficult in an arrest situation, particularly in a young patient, and is associated with a high incidence of limb-threatening complications [Firstenberg 2010]. If a sternotomy is planned, we avoid the femoral approach unless absolutely necessary and prefer central access (aortic–right atrial) whenever reasonable.

Another important component of our management was the pulmonary artery exploration and embolectomy. Surgical management of acute pulmonary embolism is an established and successful treatment option [Kadner 2008]. Although

the primary diagnosis in our case was AFE, a venous-based embolism must be considered, particularly given the known hypercoagulable state of pregnancy. We performed, and advocate, pulmonary embolectomy, not only to remove the "thrombus" burden so as to minimize the potential long-term consequences of chronic pulmonary thromboembolic disease, but also to remove any mass that might contain vasoactive and procoagulant compounds. Unfortunately in our case, the gelatinous mass seen on TEE was rapidly aspirated into a "discard" suction canister upon opening of the pulmonary arteries and so was not available for pathologic analysis. Nevertheless, the potential lifesaving benefits of evacuating any abnormal clot should outweigh the minimal risk of exploration.

CONCLUSIONS

AFE, despite aggressive medical management, is associated with a prohibitive risk of maternal and fetal mortality. On the basis of our recent success, we advocate early stabilization with temporary circulatory support and pulmonary embolectomy. Despite the rarity of AFE events, clinicians should be prepared for and consider early and invasive interventions that could be lifesaving.

REFERENCES

Benson MD. 2007. A hypothesis regarding complement activation and amniotic fluid embolism. Med Hypotheses 68:1019-25.

Clark SL, Hankins GD, Dudley DA, Dildy GA, Porter TF. 1995. Amniotic fluid embolism: analysis of the national registry. Am J Obstet Gynecol 172:1158-67.

Esposito RA, Grossi EA, Coppa G, et al. 1990. Successful treatment of postpartum shock caused by amniotic fluid embolism and cardiopulmonary bypass and pulmonary artery thromboembolectomy. Am J Obstet Gynecol 163:572-4.

Firstenberg MS, Abel E, Blais D, et al. 2010. The use of extracorporeal membrane oxygenation in severe necrotizing soft tissue infections complicated by septic shock. Am Surg 76:1287-9.

Gist RS, Stafford IP, Leibowitz AB, Beilin Y. 2009. Amniotic fluid embolism. Anesth Analg 108:1599-602.

Ho CH, Chen KB, Liu SK, Liu YF, Cheng HC, Wu RS. 2009. Early application of extracorporeal membrane oxygenation in a patient with amniotic fluid embolism. Acta Anaesthesiol Taiwan 47:99-102.

Kadner A, Schmidli J, Schönhoff F, et al. 2008. Excellent outcome after surgical treatment of massive pulmonary embolism in critically ill patients. J Thorac Cardiovasc Surg 136:448-51.

Meyer JR. 1926. Embolia pulmonary amino-caseosa. Brasil Med 2:301-3.

Miller RJ, Dean DE, Hamilton S, Gerhardt MA. 2007. Amniotic fluid embolism. Fed Pract 24:17-32.

Nagarsheth NP, Pinney S, Bassily-Marcus A, Anyanwu A, Friedman L, Beilin Y. 2008. Successful placement of a right ventricular assist device for treatment of a presumed amniotic fluid embolism. Anesth Analg 107:962-4.

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