Amniotic Fluid Embolism: Two Cases, Two Outcomes

Abstract # 133

A 34 yo G2P1 presented for elective repeat cesarean delivery. Spinal anesthesia was initiated, and the surgery proceeded uneventfully. 1 minute after delivery she experienced a 30s tonic-clonic seizure and endotracheal intubation was performed. 3 min later the patient developed PEA and ACLS protocol was initiated. Administration of epinephrine and atropine resulted in recovery of her blood pressure. Additional IV access and invasive monitoring were placed. She remained hemodynamically stable and was transferred to the ICU. She was discharged on POD#3 without neurologic deficits.

The 2nd case was a 29 yo G2P1 who underwent fetal intracardiac injection of KCl for fetal anomaly. Labor was induced with misoprostol, and she received a CSE for analgesia. 14 hrs later, she experienced a 30s tonic-clonic seizure. During this event, she acutely desaturated which recovered with nasal cannula (NC) oxygen; CXR and EKG were unremarkable. Vaginal delivery occurred at 15:15 without placental delivery. 2 min later the patient developed SOB and chest pain accompanied by desaturation, which recovered with NC. The patient also became acutely hypotensive; additional IV access and an arterial line were placed, and a phenylephrine gtt started. At 17:15, the patient was taken to the OR for removal of retained placenta; she continued to require a phenylephrine gtt and NC oxygen. Despite modest EBL, DIC was detected prompting transfusion; a Bakri was placed and additional uterotonics were given. She was transported to the PACU where she remained coagulopathic yet hemodynamically stable with no obvious bleeding, but had increasing oxygen requirements. At 22:30, she was emergently intubated for acute respiratory failure. Her abdomen became acutely distended, and hypotension worsened requiring increasing vasopressor support. During transport to the OR for exploratory laparotomy, she had PEA arrest. ACLS protocol was initiated and epinephrine was administered. Central venous access was obtained, and she soon required maximum vasopressor support. Ongoing bleeding and coagulopathy necessitated massive transfusion, and emergent hysterectomy was performed. Despite these interventions, she developed asystole and difficult to ventilate due to pulmonary edema. At 23:56 the patient was pronounced dead. Maternal autopsy confirmed AFE.

Discussion: Classically, AFE presents with sudden onset of cardiovascular (CV) collapse, respiratory arrest, and coagulopathy. However, 20% of AFE’s present with seizure, which occurred in both of our cases1. While CV collapse immediately occurred in the 1st case, AFE symptoms developed insidiously during the 2nd case. Treatment of AFE is primarily supportive, but novel treatments (ECMO, inhaled NO, leukotriene inhibitors) have been described3. It is unclear whether the use of these treatments would have led to improved outcomes.

Ref.
2. Roberts C. BJOG 2010;117