G109 A Case of Anaphylactoid Syndrome of Pregnancy

Jennifer L. Shuttlesworth, MD, Ana E. Lopez, MD, and Luis A. Sanchez, MD, Harris County Medical Examiner's Office, 1885 Old Spanish Trail, Houston, TX 77054*

The goal of this presentation is to explain the use of the term anaphylactoid syndrome of pregnancy and the difficulties one can encounter in making a diagnosis of amniotic fluid embolus.
This presentation will impact the forensic science cimmunity by illustrating a case of anaphylactoid syndrome of pregnancy and will include a discussion of the autopsy procedures necessary for intrapartum deaths.

A 19-year-old G2P1 Hispanic female presented to the emergency room with spontaneous rupture of membranes at 33-6/7 weeks gestation. She had no prior medical history and had undergone routine prenatal care. Upon admission, fetal heart rate monitoring showed evidence of fetal distress, and a decision was made to deliver the fetus via Cesarean section (the decedent had undergone a Cesarean section for a prior delivery). During the C-section delivery with an epidural anesthetic, the patient suddenly became bradycardic and hypoxic at the point of fascial closure, following delivery of the fetus and placenta. Cardiopulmonary resuscitation efforts were unsuccessful, and she died in the operating room. The male fetus survived and had no complications.

At autopsy, she had an intact surgical site with no evidence of cardiac disease or pulmonary embolus related to deep venous thromboses. Microscopically, there were platelet and fibrin thrombi with admixed neutrophils filling the small pulmonary vasculature. Thorough sampling and special stains of the lungs failed to reveal squamous cells in the pulmonary vasculature, necessary for the diagnosis of amniotic fluid embolus. Examination of the placenta showed acute chorioamnionitis.

Even though the clinical features in this case pointed towards an amniotic fluid embolus (i.e., sudden intrapartum bradycardia and cardiopulmonary arrest), the diagnosis could not be made because squamous cells were not identified in the pulmonary vasculature. A review of the decedent's medical records indicates that intraoperatively her hemoglobin decreased from 9.0 to 5.2 to 4.5 gm/L. No source of hemorrhage was identified at autopsy; therefore, the decrease in hemoglobin and the pulmonary platelet and fibrin thrombi were likely related to disseminated intravascular coagulopathy (DIC). Instead of classifying the cause of death as "amniotic fluid embolism", the cause of death was classified as "intrapartum maternal demise with diffuse pulmonary fibrin and platelet thrombi complicating Cesarean section for fetal distress, with acute chorioamnionitis."

The clinical and hemodynamic manifestations of amniotic fluid embolism have been noted to be similar to those that are manifested in anaphylaxis and septic shock. The signs and symptoms include hypotension, fetal distress, cardiopulmonary arrest, coagulopathy, cyanosis, dyspnea, and seizures. The pathophysiological mechanism for the development of the amniotic fluid embolism begins with maternal intravascular exposure to fetal elements, when there is a breach in the barrier between amniotic fluid and maternal circulation. This in turn initiates an endogenous mediator response similar to an allergic reaction, with mast cell degranulation and activation of the complement pathway.

The diagnosis of amniotic fluid embolism has been traditionally made by identifying squamous cells in the pulmonary vasculature; however, fetal tissue or amniotic fluid components are not always found in the women who present with the clinical signs and symptoms of amniotic fluid embolism, as was the case in our autopsy. In light of the apparent pathophysiological mechanisms involved and because squamous cells may not always be identified in the pulmonary vasculature, the term "anaphylactoid syndrome of pregnancy" has been used to describe the syndrome of acute peripartum hypoxia, hemodynamic collapse, and coagulopathy, which we believe this case represents.

The postmortem diagnosis of amniotic fluid embolism can be challenging to forensic pathologists. The gross findings are usually nonspecific and can include pulmonary edema and atelectasis, evidence of DIC, and pulmonary hyperinflation. Autopsy findings include fetal squamous cells in the pulmonary vasculature and masses of neutrophils and fibrin thrombi in the small pulmonary vessels. Special stains such as cytokeratin and mucin may be helpful. The autopsy should include a thorough sampling of the lungs, a proper evaluation of the uterine body looking for the possibility of wall tears as well as examination of the placenta. Thorough toxicology testing and a tryptase level are also important procedures in the evaluation of intrapartum deaths when an amniotic fluid embolism is suspected because the diagnosis is essentially one of exclusion, based on clinical presentation.

Amniotic Fluid Embolism, Anaphylactoid Syndrome of Pregnancy, Intrapartum Death

 ** Presenting Author*