Amniotic Fluid Embolism

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(Obstet Gynecol 2014;123:337–48)

Question 1:
How would you counsel a patient about a future pregnancy if she has been lucky enough to survive an amniotic fluid embolism (AFE)? Would there be any special precautions she would need to take for her next pregnancy?

Response from Dr. Clark:
The available data in this area consist only of several very small series and case reports. These data suggest that the risks of recurrence are low. In addition, a pathophysiologic mechanism of disease that hinges on a maternal reaction to a specific set of fetal antigens would suggest that recurrence ought to be uncommon. On the other hand, having dodged one bullet, is it really wise to spin the wheel again? My counseling goes something like this: “Available data suggest that the risk of recurrence is low, and there are a number of reports of successful pregnancy outcome after AFE survival. However, given the potential severity of AFE if it does recur, and a lack of really good data regarding risks, I advise you to undertake another pregnancy only if you are willing to accept a small risk of catastrophic outcome including death.” If a patient chooses to undertake pregnancy, I do not alter my management in any way, other than delivery in a tertiary center.

Question 2:
Although AFE is the most common cause of acute cardiovascular collapse in a peripartum patient, what other disease processes would be in your differential diagnosis?

Response from Dr. Clark:
1) Standard postpartum hemorrhage from atony, retained placenta or accreta, or laceration; 2) Anesthetic accident; 3) Pulmonary thromboembolism; and 4) Myocardial infarction.

Question 3:
Have there been any developments or treatments for systemic inflammatory response syndrome that have improved the treatment of patients with suspected AFE?

Response from Dr. Clark:
No. Treatment remains supportive.
Question 4:

Given that the passage of some fetal tissue into the maternal circulation appears to be ubiquitous during delivery, and the findings of these in the maternal lungs at autopsy are not diagnostic of an AFE and can be found in other settings, should AFE be the “default” diagnosis of death if all other causes have been ruled out? If so, are we not making the diagnosis of AFE a “wastebasket” again?

Response from Dr. Clark:

No. the clinical triad of hypotension, hypoxia, and coagulopathy, with onset during labor or within minutes of delivery, remain the hallmarks of most cases of AFE. Although not all three of these components are always present with AFE, their absence necessitates thought of a different diagnosis. Sometimes no diagnosis can be reached with confidence, as with some cases of unexpected sudden death in any adult patient.

Question 5:

Do you think in the future there may be ways to test or detect abnormal immunologic susceptibility to the fetal antigens that activate the proinflammatory response in AFE? Might there be something like a “skin test” or in vitro test for reactions to these antigens as there are for molds or pollen? Would this test be applicable to all women prior to conceiving or just after the diagnosis of pregnancy? How useful would such tests be for a condition with an incidence of 1:40,000 deliveries?

Response from Dr. Clark:

I hope so. This is one principle focus of the Baylor study. Whether such a test is cost effective or not is another issue altogether, particularly given the absence of a preventative measure. Ideally, identification of such at-risk patients would lead to a more thorough understanding of pathophysiology and lead to a more specific treatment. I believe we are still some distance from achievement of this goal – a long, basic science slog is going to precede effective clinical prevention.

Question 6:

You mention that the clinical triad of hypotension, hypoxia, and coagulopathy is not always encountered. Is there one part of this triad that classically occurs more often than the others? Should one of these events make us more likely to call the process an AFE?

Response from Dr. Clark:

No good data exists to answer this question, since many patients in large population-based studies without all three legs of this triad probably did not have AFE. While disseminated intravascular coagulation (DIC) may be the component that is most commonly clinically absent in AFE (usually due to rapid death before DIC can be assessed), it may be the most specific. In truth, only two conditions in obstetrics result in acute, life-threatening DIC and hemorrhage: massive placental abruption and AFE. In the absence of obvious clinical evidence of the former, the sudden appearance of massive hemorrhage due to DIC strongly suggests AFE, regardless of other signs or symptoms. This must of course be distinguished from massive hemorrhage that results in coagulopathy.

Question 6:

Since most general obstetrician–gynecologists will likely transfer their patient whom they suspect is having an AFE to the intensive care unit and their critical care colleagues, what do you believe would be the most important aspect of the disease process that the intensivists would need to know to best assist in caring for the patient?

Response from Dr. Clark:

1) After delivery, the source of the illness has been eliminated; 2) Treat this patient like you would treat any individual with this degree of cardiac dysfunction, lung injury, or coagulopathy; and 3) Be aware of previously published norms for central hemodynamic parameters in pregnancy, and consider them in your management plan.