

SUCCESSFUL RESUSCITATION OF AN OBSTETRIC PATIENT WITH SUSPECTED AMNIOTIC FLUID EMBOLISM

- Case Report -

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We describe the successful resuscitation of a parturient who developed cardiovascular collapse immediately after cesarean delivery most likely due to amniotic fluid embolism, based on the clinical presentation and later confirmed by exclusion. Aggressive therapy was instituted right away including early administration of vasopressin. Care continued in a critical care setting and she recovered completely.

Amniotic fluid embolism (AFE) is a rare, but potentially life-threatening complication of obstetric patients. Mortality remains high, despite improvements in diagnosis, monitoring and treatment. This report describes sudden development of dyspnea, coagulopathy and cardiovascular collapse in a young woman shortly after delivery of a healthy baby. Based on a high index of suspicion, therapy was instituted immediately with successful results.

Case Description

A 34 year-old woman, gravida 3, para 1, was scheduled for caesarean section (c/s). She had a history of hypertension and was morbidly obese (139.7kg/1.626cm). She had developed pneumonia after the last cesarean section several years previously. She had undergone laparoscopic gastric banding without anesthetic complication. No drug allergies were reported. Combined spinal epidural anesthesia with hyperbaric bupivacaine 7.5mg, fentanyl 15µg, and morphine 200µg was selected as the anesthetic technique. Blood pressure remained stable throughout. Adequate analgesic level was documented at T6. Ten minutes after skin incision, a female baby was delivered with Apgar score of 9/9. Infusion of oxytocin (20u/1000cc lactated Ringer's solution) was started, followed by a test dose of Unasyn®. While the recommendation to administer antibiotics prior to skin incision has been advocated, we believe that because of the extremely low infection rate in this procedure, the risk of possible fetal compromise outweigh the benefit. Following a five minute period during which there was no negative response to the antibiotic, Unasyn® 1.5gm was given slowly intravenously. Five minutes later, the patient complained that she felt "sick", and became acutely dyspneic. Motor power and sensation was intact above T6. No skin erythema was seen and there was no wheezing. Systolic blood pressure decreased from 120 to 70. Heart rate remained at 110-120 bpm. The antibiotic infusion was stopped. Several doses of phenylephrine 50µg were given intravenously followed by intravenous epinephrine 50µg, 100µg and 200µg. The patient was arousable but lethargic. Her gag reflex remained intact. Oxygen saturation fell to 80% and end tidal CO₂ measured by side stream capnography decreased to about 20. Nasopharyngeal

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and oropharyngeal airways were inserted to facilitate respiration which was decreased due to her obesity. Mask ventilation with O₂ 100% was started. Oxygen saturation remained at 88-95%. However, it was very difficult to maintain her systolic blood pressure >90 mmHg. Vasopressin 20u was given intravenously. The differential diagnosis at this point, in descending order of likelihood was amniotic fluid embolism, antibiotic anaphylaxis, latex allergy, venous air embolism, thrombotic pulmonary embolism. Decadron 4mg was given, followed by an intravenous infusion of phenylephrine (10mg/100cc NS). Eventually, infusions of epinephrine (2mg/100cc in normal saline) and vasopressin (20u/100cc in normal saline) were also added. Surgery was completed with adequate hemostasis. The patient remained lethargic and was transferred to the ICU. She became more alert and the nasopharyngeal and oropharyngeal airways were removed within an hour. However, her blood pressure remained around 90mmHg with epinephrine and vasopressin infusions. The coagulation profile showed PT of 18, PTT of 48.7, fibrinogen of 196 and D-Dimer of 1482. Chest CT was negative for pulmonary embolism. The diagnosis of amniotic fluid embolism was established. The patient received several units of fresh frozen plasma and cryoprecipitate. Her coagulopathy resolved the next day. She continued to recover and was discharged home on postoperative day #4.

Introduction

AFE is a rare but potentially catastrophic complication of obstetric delivery.¹ It is believed that AFE is not an all-or-none phenomenon. Rather, it is a spectrum of disease that ranges from a subclinical entity to one that is rapidly fatal.² The true incidence of AFE is unknown, with great variation among reports, ranging from 1 in 8,000³, to 1 in 20,646⁴, even to 1 in 80,000 pregnancies¹. However, the mortality remains high in symptomatic patients. Morgan reported a mortality rate of 86%¹. Later, in Clark's registry, the mortality was 61%⁵. Among survivors in Clark's registry, only 15% of patients were neurologically intact⁵.

There were no predisposing maternal risk factors identified throughout the review studies.^{1,5} The onset of symptoms is highly variable. It can occur during vaginal delivery⁶, Caesarean section^{7,8}, or other uterine surgical

procedures during pregnancy, such as, first trimester curettage abortion⁹, second trimester termination¹⁰, and cervical suture removal¹¹. The presentation of AFE can be highly variable and depends on the predominant physiological aberration. It can present as an isolated coagulopathy⁶, pulmonary edema¹¹, seizures⁸, hemodynamic collapse¹⁰, or combination of the above clinical signs¹². However, disseminated intravascular coagulopathy (DIC) develops in up to 83% of patients⁵.

There is yet no specific laboratory test or image study to confirm the diagnosis of AFE². The classic triad includes acute hypoxia, hemodynamic collapse and coagulopathy without an obvious precipitating cause¹³. In this case report, a high index of suspicion played an essential role in the initial diagnosis. Prompt recognition and rapid institution of aggressive resuscitative efforts, especially early introduction of vasopressin, contributed greatly to successful resuscitation.

Discussion

The clinical symptoms and signs in this patient included dyspnea, hypotension, and coagulopathy which led to the initial differential diagnosis of amniotic fluid embolism, anaphylactic shock from antibiotics or latex, thrombotic pulmonary embolism or venous air embolism. In an awake patient the initial manifestations of anaphylactic shock include skin erythema, pruritis, a generalized feeling of warmth, and fear of impending doom as the patient experiences a sensation of closure of her throat. Urticaria is very common. Between 25-50% of fatal anaphylaxis have pathologic changes consistent with severe asthma. Cardiovascular collapse occurs later.¹⁴ The presentation of this patient included coagulopathy and cardiovascular collapse without any urticaria, which makes the diagnosis of anaphylaxis highly unlikely.

Also, in an awake patient, the presentation of thrombotic pulmonary embolism or venous air embolism includes dyspnea, wheezing, chest pain, agitation, confusion, tachycardia, and hypotension.¹⁵ The additional finding of coagulopathy in our patient makes these diagnoses unlikely. Although it was difficult to confirm the diagnosis of AFE histologically, secondary to the patient's survival, the clinical course of cardiovascular collapse and coagulopathy during

the caesarean section suggested AFE as the most likely cause of this intra-operative event.

There is as yet no specific laboratory test, image study or protocols available to confirm the diagnosis of AFE in suspected cases². At autopsy, Roche and Norris confirmed the presence of mucin and fetal debris, in maternal lung sections¹⁶. The classic triad of AFE includes acute hypoxia, hemodynamic collapse and coagulopathy without an obvious precipitating cause¹³. AFE should be suspected in any pregnant patient, specifically those with ruptured membranes, who develop sudden onset of dyspnea with hypoxia, acute hypotension and/or cardiac arrest followed by coagulopathy¹⁰.

Review of relevant literature suggests that the historical concept of an actual embolism of amniotic fluid producing the clinical syndrome is not accurate¹³. An analysis by Clark reported a striking similarity between AFE and anaphylaxis, suggesting a common underlying pathophysiologic mechanism⁵. He proposed that AFE be renamed as *Anaphylactoid Syndrome of Pregnancy*. Other reports confirmed that it is very difficult to differentiate between anaphylaxis and AFE, especially among survivors¹⁷. Nevertheless, this hypothesis helps to direct more attention to the management of AFE, rather than attempting to confirm a diagnosis. In this way, the management of AFE is logically similar to that of anaphylaxis.

Our patient presented the triad of AFE including acute hypoxia, hemodynamic collapse and coagulopathy shortly after the fetal delivery during caesarean section. The management of AFE remains primarily supportive with emphasis on resuming the stability of oxygenation, cardiac output and coagulation². A high index of suspicion plays an essential role in the initial diagnosis. Prompt recognition and institution of resuscitation may improve maternal and fetal outcomes¹⁰. Management must be aggressive and supportive initially¹³. If the patient becomes unconscious or hypoxia is refractory to mask ventilation with 100% O₂, endotracheal intubation and mechanical ventilation is indicated. For parturients who are undelivered at the time of the event, prompt delivery of the fetus is necessary for the

success of resuscitation. Adequate intravenous access must be established with central venous cannulation if possible. Aggressive volume and vasopressor support is essential. Hemodynamic management may be facilitated by placement of a pulmonary artery catheter. Development of coagulopathy should be anticipated and treated with cryoprecipitate, fresh frozen plasma and blood. An intensive care setting is crucial for the postoperative management of left ventricular failure, adult respiratory distress syndrome (ARDS) as well as disseminated intravascular coagulopathy¹³.

In this case report, the patient was conscious, maintaining oxygen saturation between 88-95%, facilitated by oropharyngeal airway, with mask O₂ 100%. Since her gag reflexes were intact, endotracheal intubation which would have required additional agents which might further increase hypotension was delayed. Attempts at hemodynamic restoration were aggressive, including placement of additional intravenous access, catheters, replenishment of pre-load with crystalloid, and combination of vasopressors. Initially, a phenylephrine infusion, a direct α -agonist, was started. As the dosage of phenylephrine was increased, the heart rate decreased, although not significantly. Epinephrine infusion was added to further stabilize the patient's blood pressure and heart rate, through its positive inotropic and chronotropic action. Systolic pressure remained at around 80. Vasopressin infusion was added at this time, which restored and maintained systolic pressure around 90 mmHg.

Although there is no algorithm or guidelines for the management of anaphylaxis that includes the use of vasopressin, Hussain reported a case of a 24-year-old woman who developed severe anaphylactic shock at induction of anesthesia while undergoing laparoscopic cholecystectomy. Shock was refractory to epinephrine and high doses of pure alpha-agonists, phenylephrine and norepinephrine. A single intravenous dose of two units of vasopressin re-established normal circulation and blood pressure¹⁸. In our case, we believe that the combination of multiple vasopressor infusions, especially adding vasopressin in catecholamine-resistant shock, contributed greatly to the successful outcome.

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