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Amniotic Fluid Embolism-Two case reports and literature review

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Amniotic fluid embolism (AFE) is a complex condition classically characterized by the abrupt onset of hypotension, hypoxia and consumptive coagulopathy.

AFE is a threatening condition, and in most cases, the outcome is fatal. It cannot be predicted nor prevented. Review of the literature reveals that there are no standardized investigations at methods or protocols to confirm the diagnosis in suspected cases. Therefore diagnosis has been accepted based on clinical grounds for those who survive and on autopsy findings in the lungs of those who die (ref).

Keywords: Amniotic fluid embolism, consumptive coagulopathy

Epidemiology:

The incidence ranges between 1 in 8,000 and 1 in 80,000 pregnancies [1, 2]. In the last confidential enquiry (1997-99) in UK there were eight deaths due to AFE, (3.7/ million maternities). AFE is responsible for 10% of all maternal deaths in the United States [2, 3]. 70 % of AFE occur during labour, 11% after vaginal delivery and 19 % during lower segment cesarean section usually after delivery of the infant[4]

Case 1

A 35 years old G4 P3 + 0 patient was admitted in spontaneous labour at term. She had no previous medical or obstetrics problems. At 5 cm cervical dilatation, her labour was augmented with an artificial rupture of membrane and oxytocin infusion. She suddenly developed acute cardiopulmonary collapse with central cyanosis in late 2nd stage of labour. The baby was delivered by forceps with very low Apgar score and was admitted to the neonatal intensive unit (NICU). Delivery of the placenta was followed by severe coagulapathy. Patient died on the following day.

Case 2

A 39 years old lady G7 p4 +2 was admitted in spontaneous labour. She had no relevant medical or obstetrics history. Labour was progressing normally until the cervix was about 5-6 cm. Because of some delay artificial rupture of membrane was performed. Soon after this, she suddenly developed cyanosis, cardiovascular collapse and cardiac arrest. She was still in the first stage of labour (5-6 cm dilated). Resuscitation failed and patient died within one hour. Baby died in utero... The fetus died in utero.

Discussion:

AFE was first described by Meyer in 1926[5]. It became an established clinical entity in 1941 after Steinr and Luschbaugh published a maternal mortality case series that included eight women who had squamous cell and mucin, presumably of fetal origin, within their pulmonary vasculature [6]. Because AFE is so uncommon no single institution has sufficient experience to assess risk factors, determine the

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pulmonary hypertension and vasospasm are the initial hemodynamic response to AFE. The resulting right heart failure and hypoxia could accounts for sudden death or sever neurological impairment. A number of mechanisms have been suggested for the occurrence of myocardial dysfunction. One hypothesis is ischemic injury to the myocardium in those presenting with acute respiratory distress and hypoxemia [16]. The cardiac out put is depressed subsequent to impaired with ventricular filling which in turn is decreased. Endothelin has been suggested to be responsible for the myocardial depression in case of AFE [17]. In AFE there is a sudden increase in the level of maternal plasma endothelin. Animal experiments have showed that endothelin has a powerful constrictor effect in coronary and pulmonary arteries and in human bronchi. In 1995 Chark[7] was the first to suggest that AFE should be called "Anaphylactoid Syndrome of Pregnancy" . He noted the clinical and hemodynamic changes which are similar between AFE and both anaphylaxis and septic shock. In all these conditions, primary and secondary mediators are released in response to the entrance of foreign body to the maternal circulation. These mediators cause myocardial depression, decrease cardiac out put, produce pulmonary hypertension and disseminated intravascular coagulation (DIC).

Coagulation:

In 1950 Warner and Reeid were the first to report the coagulopathy associated with AFE [7]. they found that this syndrome is almost always associated with disseminated intravascular coagulation (DIC), with or without significant bleeding. Although many authors have attempted to clarify the cause and the mechanism of the consumptive coagulopathy that occurs with AFE, the exact etiology remains obscure. Lock wood et al [19] suggested that tissue factors may be the responsible factor for the development of the consumptive coagulopathy and found substantial quantities of tissue factor in amniotic fluid. Potential sources include sloughed fetal skin and epithelial cells derived from the fetal respiratory, gastrointestinal, and genitourinary tract mucosa. Tissue factors activate the extrinsic pathway by binding with factor VII. This complex in turn triggers clotting by activating factor X.

Diagnosis:

Due to the abrupt events of the condition, there are no routine diagnostic measurements to confirm the diagnosis of AFE. It is a diagnosis of exclusion. Any condition that presents as acute cardio respiratory collapse or massive hemorrhage in the peripartum period must be systematically evaluated. The differential diagnosis includes air or thrombolic pulmonary emboli, septic shock, myocardial infarction, cardiomyopathy, anaphylaxis, aspiration, abruptio palcentae, eclampsia, uterine rupture and local anaesthetic toxicity [4, 13]. When central venous access has been obtained, blood from the pulmonary vasculature should be collected using the method described by Mason [5]. He suggested that in order to minimize the possibility of maternal or exogenous contamination, blood should be obtained from the distal lumen of a wedged pulmonary artery catheter. After discarding the first 10 ml of blood, an additional 10 ml is drawn, heparinized and analyzed utilizing papanicolaou's method [22]. The diagnosis of AFE is confirmed if the squamous cells are coated with neutrophils or if they are accompanied by other fetal debris such as mucin or hair.

Similarly, Lee et[23] al suggested that a more

reliable method of confirming the diagnosis might center on the identification of other amniotic fluid elements in the maternal pulmonary vasculature as a opposed to squamous cells. Recently, Kobayashi et al, [24] studied maternal serum sialyl Tn antigen levels in women with clinical AFE and compared them to both normal pregnant and non pregnant controls. Sialyl is a mucin type glycoprotein synthesized in the fetal and adult intestinal and respiratory tracts. This glycoprotein present in both meconium and clear amniotic fluid. Using a sensitive antimucin antibody, TKH-2, the authors found no difference in the serum levels of pregnant patients through out gestation or in the early post partum period when compared to healthy non-pregnant controls. In comparison, the antigen levels in the AFE group were elevated. Therefore, the authors concluded that the test was a promising simple, non- invasive method for diagnosing AFE. Another recent study, measured maternal plasma concentrations of zinc coproporphyrin a characteristic meconium component. The worker found that the level of this compound higher in women with AFE, than in healthy pregnant and non pregnant controls.[25]

Management

Care of the mother with amniotic fluid embolism is non specific, but mainly supportive. It should include high concentration of oxygen, circulatory support and correction of the coagulopathy. However, most patients with this syndrome die, and most of the survivors are neurologically impaired despite appropriate resuscitative measures. If the fetus is sufficiently mature and is undelivered at the time of maternal cardiac arrest, cesarean section should be instituted as soon as possible. Vasopressors are useful in restoring aortic perfusion pressure [20]. Pulmonary artery catheterization can be instituted to help guide therapy. Blood component is given to correct the coagulopathy. Packed red blood cells are considered the first line in the management to replace the blood loss in order to maintain oxygen delivery to the tissues. Group specific or "O" negative blood can be used if cross-matched blood is unavailable. Consumption of the platelets, clotting factors and anti thrombin III can be replaced by plasma and platelets transfusion. In patients with low fibrinogen levels, cryoprecipitate is a useful therapy. Cryoprecipitate in opsonic alpha 2 surface binding glycoprotein known as fibronectin which aids the reticuloendothelial system in the fitration of antigenic and toxic particles. Depleted levels of this glycoprotein have been reported in severely ill patients, with marked improvement in the clinical status following repletion of fibronectin levels [21]. High doses of epinephrine and corticosteroids are useful therapeutic measures [8].

Conclusion

Although the number of deaths from AFE is low, it remains frustrating that we still have no clear idea how to prevent or treat this condition. The spectrum of the disease can vary from subclinical entity to one that is rapidly fatal. Rates of obstetrics intervention in the form of amniocentesis and induction and augmentation of labour should be kept as low as possible. Further research into the condition is very necessary. We must develops national and international registries and encourage collaboration between research center. Without a specific method to confirm the diagnosis and, standardized diagnotic protocols and registries to collect data, our understanding of the disease and its treatment will continue to be hampered .

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