

Amniotic Fluid Embolism

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Abstract

Amniotic fluid embolism (AFE) is a devastating obstetric complication that requires early and aggressive intervention with optimal cardiopulmonary resuscitation, as it has high morbidity and mortality. Immediate recognition and diagnosis of AFE is essential to improve maternal and fetal outcomes. The true incidence of this entity is unclear because this syndrome is difficult to identify and the diagnosis remains one of exclusion, with possible under-reporting of nonfatal cases. AFE is caused by abnormal activation of immunologic mechanisms following entry of fetal antigens into maternal circulation. AFE classically presents as a sudden cardiovascular collapse associated with respiratory compromise, fetal distress and development of a coagulopathy. Treatment of AFE is supportive and directed at treating cardiovascular, pulmonary, and coagulation derangements.

Keywords : Amniotic Fluid, Embolism

Introduction

In 1926 amniotic fluid embolism (AFE) was first recognized and reported in Brazil. In 1941 Steiner and Lushbaugh defined AFE based on post mortem findings of fetal squamous cells in vasculature. Although AFE was first identified as a clinical entity in 1941, it remains an unpredictable condition and treatment is still largely supportive. AFE still accounts for 4.7% of direct maternal deaths in the UK, 13% in France, 30% in Singapore, and up to 10% in the USA and Australia (As cited in Dedhia & Mushambi, 2007).

Normally, amniotic fluid does not enter the maternal circulation because it is contained safely within the uterus, sealed off by the amniotic sac. AFE occurs when the barrier between amniotic fluid and maternal circulation is broken and, possibly under a pressure gradient, fluid abnormally enters the maternal venous system via the endocervical veins, the placental site (if placenta is separated), or a uterine trauma site.

In Nepal, a 10 year review of maternal mortality ratio in Paropakar Maternity and Women's Hospital (PMWH) Thapathali Kathmandu showed the leading

cause of MMR was hemorrhage (30.30%) followed by eclampsia (24.24%). Sepsis, suspected cases of pulmonary embolism and amniotic fluid embolism each contributing 15.15%, 4.54% and 3.03% respectively (Upadhyaya, 2014). In developing countries little attention has been given to near miss obstetric events. The scenario is similar in Nepal. This probably is a result of persistently high level of maternal mortality that has overshadowed other severe obstetric complications.

Amniotic fluid embolism is an event that is as unpreventable as it is unpredictable (Singh, 2013) and is a catastrophic syndrome occurring during labor and delivery or immediately postpartum. This is a rare emergency in obstetrics with high mortality rate. AFE classically presents as a sudden cardiovascular collapse associated with respiratory compromise, fetal distress and the development of a coagulopathy. The AFE reported is; during labor- 70% , after C/S – 19%, after vaginal delivery – 11% ,after membranes ruptured – 78% (Dedhia & Mushambi, 2007).

The etiology of AFE remains unclear. Initially AFE was thought to be secondary to the mechanical

obstruction of the maternal circulation by amniotic fluid. More recent theories suggest that AFE is an immune mediated response to the presence of amniotic fluid in the maternal circulation. Older age, Intrauterine fetal death, Multiparity, Physiologic intense uterine contractions, large fetal size, Medical induction of labor, Meconium staining of the amniotic fluid, Instrumental vaginal delivery, Placental abruption, Prolonged gestation, Eclampsia, Cesarean section, Fetal distress, Uterine rupture, Trauma to abdomen Polyhydramnios Surgical intervention High cervical tears, Premature placental separation as risk factors for AFE (Tan & McDonnell, 2010).

There is no single laboratory finding by which AFE can be diagnosed. Ventilation perfusion scans aid in the diagnosis. ECG may show right ventricular strain while echocardiography confirms severe left ventricular failure. Various blood coagulation tests may be deranged.

Clinical management of AFE should focus on aggressive cardiovascular support, treatment of hypoxia, management of hemorrhage and coagulopathy, and delivery of the fetus. In the maternal cardiac arrest condition, basic and advanced life support should be undertaken which is essential to optimize maternal outcome. Following initial resuscitative measures, most patients will require continued monitoring and support in an ICU (Jeejeebhoy, Zilap & Lipman, 2015). Oxygen (100% face mask or bag mask) should be given to treat hypoxia and prevent further hypoxic injury. The goal should be to keep oxygen saturation greater than 90%. Intubation and advance airway support are often required, to achieve this goal.

Electrocardiogram, pulseoxymetry and blood pressure should be immediately done. Large bore intravenous access should be obtained. An arterial line should be placed in hemodynamically unstable patients. Transthoracic or transesophageal echocardiography may be done for immediate evaluation of right and left ventricular function (McDonnell, Percival, & Paech, 2013). If the patient is unstable and hypotensive volume resuscitation, vasopressors and inotropic supports are used to optimize preload, contractility, and after-load. Vasoactive drug therapy must be tailored to the clinical situation. Fetus and

placenta should be delivered as soon as feasible. The maternal mortality rate could be as high as 80 percent, with 50 percent dying within the first hour of the onset of symptoms. Survival is rare and those that do survive have increased chances of neurological impairment. The survival rate of newborn, is estimated to be about 70 percent (Singh, 2013).

Conclusion

AFE is a rare and often fatal complication of the peripartum period. The severity of its consequences, rapid onset, and limited treatment options make recognition of this syndrome a vital importance for those caring for patients in the peripartum period. The most important goal of therapy is to prevent additional hypoxia and subsequent end-organ failure. The treatment is supportive and focuses on rapid maternal cardiopulmonary stabilization.

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