Amniotic Fluid Embolism – A Case Report

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Abstract: An amniotic fluid embolism (AFE) is a rare childbirth (obstetric) emergency in which amniotic fluid enters the blood stream of the mother to trigger a serious reaction. This reaction then results in cardiorespiratory (heart and lung) collapse and massive bleeding (coagulopathy).[1] A 32 year old lady elderly primi with 39 weeks of gestation with cephalic presentation with precious pregnancy with polyhydramnios brought by relative’s to the local hospital. Patient was taken to LSCS and live male baby was extracted by gynecologist. Liquor clear and excess, placenta and membranes removed manually, uterus feel flabby. No evidence of post-partum hemorrhage or tear. HISTO-PATHOLOGY REPORT: lungs show features of pulmonary oedema, interstitial inflammation and foci of hemorrhagic infarct. AFE is diagnosed when all other causes have been excluded. The presence of fetal squamous cells or other fetal tissues, including meconium have been found in the maternal circulation after the event. Diagnosis is also based upon the signs and symptoms observed during the birth or procedures.

Keywords: amniotic fluid embolism, caesarian section, polyhydramnios, pulmonary edema, interstitial inflammation, hemorrhagic infarct.

1. INTRODUCTION

An amniotic fluid embolism (AFE) is a rare childbirth (obstetric) emergency in which amniotic fluid enters the blood stream of the mother to trigger a serious reaction. This reaction then results in cardiorespiratory (heart and lung) collapse and massive bleeding (coagulopathy).[1]

Amniotic fluid embolism is suspected when a woman giving birth experiences very sudden insufficient oxygen to body tissues, low blood pressure, and profuse bleeding due to defects in blood coagulation. Though symptoms and signs can be profound, they also can be entirely absent. There is much variation in how each instance progresses. [2, 3]

AFE is very rare and complex. The disorder occurs during the last stages of labor when amniotic fluid enters the circulatory system of the mother via tears in the placental membrane or uterine vein rupture. [4]

Upon later analysis, fetal cells are found in the maternal circulation. When the fetal cells and amniotic fluid enters the bloodstream, reactions occur that cause severe changes in the mechanisms that affect blood clotting.

Disseminated intravascular coagulation occurs and results in serious bleeding. The condition can also develop after elective abortion, amniocentesis, cesarean delivery or trauma. Small lacerations in the lower reproductive tract are associated with AFE.

The use of drugs to induce labor, such as misoprostol, nearly doubles the risk of AFE. A maternal age of 35 years or older is associated with AFE.[5]
Amniotic fluid embolism is very uncommon and the rate at which it occurs is 1 instance per 20,000 births. Though rare, it comprises 10% of all maternal deaths.

1.1. Risk Factors
Many factors are associated with increased risk, but evidence is inconsistent. As with exposure to fetal antigens, many of the risk factors are commonplace or at least much more likely than amniotic fluid embolism, and there is no good pathophysiologic understanding of why only a few women with risk factors develop the syndrome. Nonetheless, risk is generally thought to be increased by the following:

- Cesarean Delivery
- Polyhydramnios

2. Case Report
On April 2019 we received a deceased body of 32 year old female, for post mortem examination at department of forensic medicine & toxicology, Adichunchanagiri Institute of Medical Sciences, B G nagar.

3. History
A 32 year old lady elderly primi with 39 weeks of gestation with cephalic presentation with precious pregnancy with polyhydramnios brought by relative’s to the local hospital. Patient was taken to LSCS and live male baby was extracted by gynecologist. Liquor clear and excess, placenta and membranes removed manually, uterus feel flabby. No evidence of post-partum hemorrhage or tear.

On examination patient blood pressure drops to 70/30 mmhg, pulse 52/minute, spo2 65% and patient was non-responsive. CPR initiated with adrenaline and intubated. Then patient was referred to higher centre accompanied by gynaecologist and anesthetist. But on the way to higher centre patient was declared dead.

Medico-legal-case intimation was sent to nearest police station and inquest report was prepared by concerned police under section 174 cr.p.c.

4. Postmortem Findings
The deceased body is of a female, aged about 33 years, moderately built and nourished, measuring 5’5” in length with black colored scalp hair.

Both the eyes are closed and pupils are dilated and fixed.

5. Discussion
AFE is diagnosed when all other causes have been excluded. The presence of fetal squamous cells or other fetal tissues, including meconium have been found in the maternal circulation after the event. Diagnosis is also based upon the signs and symptoms observed during the birth or procedures.

The most significant pathologic findings at autopsy are limited to the lungs. Grossly, the lungs show evidence of pulmonary edema (in 70% of the cases). Alveolar hemorrhage and pulmonary embolism of amniotic fluid materials are present; the presence of embolic particles is essential for diagnosis, but on histology they may be missed because of their small size.

Lung Frequent findings at autopsy included pulmonary edema, congestion, and focal atelectasis in AFE.

The clinical manifestation of AFE resembles both embolism and anaphylaxis. A reliable diagnosis could be made only upon histological evaluation; however, it is still nothing more than one of the necessary conditions for the diagnosis of AFE.

6. Conclusion
Amniotic fluid embolism syndrome is an infrequent, unpredictable, and catastrophic complication of pregnancy. It is virtually impossible to predict which patients are at risk for AFE. Diagnosis must be based on a spectrum of clinical signs and symptoms and by exclusion of other causes. Most cases of AFE
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are associated with dismal maternal and fetal outcomes, regardless of the quality of care rendered. Improved understanding of the pathophysiology of AFE may lead to the development of preventive measures and more effective and specific treatment.

REFERENCES


