Case Report

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Amniotic Fluid Embolism with Aspiration of Gastric Contents: Report of an Unusual Association

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Abstract

Amniotic fluid embolism (AFE), an exceptional, disastrous, complication of pregnancy may occur in association with aspiration of gastric contents, one of its clinical mimics. Histologic confirmation of particulate components of amniotic fluid such as fetal squamous cells, lanugo hair, mucin and debris in pulmonary microvasculature is the foundation of diagnosis and presence of partially digested vegetative material with acute inflammatory changes in terminal bronchioles is a telltale sign of aspiration of particulate gastric content. We report a case of amniotic fluid embolism with aspiration of gastric contents in a 32 year pre-eclamptic, anemic patient that occurred after induction and augmentation of labor with a vaginal administration of misoprostol and artificial rupture of fetal membranes respectively. These episodes culminated in death of mother and female fetus during labor.

Keywords: Amniotic Fluid Embolism; Maternal Mortality; Aspiration; Gastric Contents.

Introduction

Maternal deaths are routinely and medico legally investigated all over the world to find out the causes and categorize them. This exercise is done as maternal mortality is pointer of competence of health care system in a country and helps in formulating the national health care policy. The legal aspect of investigation is to find out if there was any negligence or malpractice by health care professionals' leading to death of mother [1]. Maternal mortality is also directly related to fetal mortality as the risk of fetal death increases with mother's death. It not only affects relatives and communities but also at times demoralizes the sincere health care professionals [2]. At national level, it brings substantial economic losses so as to act as stumbling block to community development.

There are direct and indirect causes of maternal demise. The former are most important and include hemorrhage, obstructed labor, eclampsia/pre-eclampsia, amniotic fluid embolism, thrombo-embolism/thrombosis, sepsis, ectopic pregnancy, anesthesia complications and unsafe abortion. The indirect causes include cardiac disease, neurological conditions, psychiatric causes, indirect malignancies and infections such as hepatitis, malaria, AIDS, and anemia [3-5]. Delayed and/or incorrect treatment and a shortage of supplies increase the maternal deaths [4-5].

Amniotic fluid embolism, one of the complications associated with pregnancy, clinically masquerades with acute myocardial infarction, anaphylaxis, air embolism, pulmonary thromboembolism, placental abruption, pre-eclampsia/eclampsia, uterine rupture, transfusion reactions, septic shock, hemorrhage, local anesthetic toxicity and aspiration of gastric contents [9-10]. Any of these entities may occur concurrently with AFE [11]. However simultaneous occurrence of aspiration with AFE has been documented as rarest of rare case reports [13-14].

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Case Report

A 32 year old, G2P1L1 female was referred from primary health center (PHC) to our hospital in the latent phase of labor at 38 weeks of gestation with probable clinical diagnosis of ANC with labor pains and pre-eclampsia. On admission the patient was conscious and oriented. Past obstetrics history revealed full term normal delivery of female baby 12 years ago. On examination, her general condition was fair, afebrile, pulse was 90 beats per minute and blood pressure 130/80 mm of Hg. Per abdominal examination revealed full term uterus, fetal heart sound 140 beats per minute and regular. Per vaginal examination revealed 1 cm dilated cervix. Per speculum examination exhibited patulous and posterior cervix. The labor was induced with per vaginal administration of tablet misoprostol 25µg. Her hemoglobin was 10 gm/dl, serum creatinine 0.6 mg/dl, blood urea 18 mg/dl, and blood group B Rh Positive (+ve). Urine examination- albumin 1+ by dipstick. Labor gradually progressed. Three hours after admission per vaginal examination revealed cervix 3 cm dilatation, 30% effacement, station -1, BOM+ (Bag of Membrane). Artificial rupture of membrane was done. Liquor was clear and it took six hours for dilatation of cervix. Fetus showed vertex presentation with station 0 to +1. Her blood pressure was 120/70 mm of Hg. Five hours after artificial rupture of membranes; she suddenly became breathless and unconscious while bearing down. Her blood pressure and pulse were not recordable. She failed to respond to all measures of cardio-pulmonary resuscitation. Both mother and fetus were declared dead 15 minutes after onset of clinical symptoms. Police was informed about the death of mother and fetus as it was a case of maternal death.

A post mortem was performed. Post mortem examination revealed edematous right and left lungs weighing 500gms and 450gms respectively. The tracheobronchial tree was patent and congested, edematous. The heart was normal in size and weighed 300gms. Brain, liver, spleen, kidneys, gastrointestinal tract exhibited congestion. Uterus measured 31cmx 28cm x2cm weighed 1250gms and did not reveal laceration. Female fetus weighed 3kg, crown to heel length was 55 cm and there were no external abnormalities. Placenta weighed 500gms. The placental membranes showed rupture. The cause of maternal death could not be ascertained on autopsy findings. We received bits of heart, lungs, kidneys, brain, intestine and spleen for histopathologic examination in a sealed container.

Microscopic Examination

Microscopic examination of H & E stained sections of lungs revealed fetal squamous cells, lanugo hairs and amniotic debris consisting of mucin, in the pulmonary microvasculature; and pulmonary edema (Figure 1-2). Bronchioles exhibited vegetative material and eosinophilic debris within their lumina (Figure 5 & 6). The histologic diagnosis of amniotic fluid embolism with aspiration of gastric content was made.

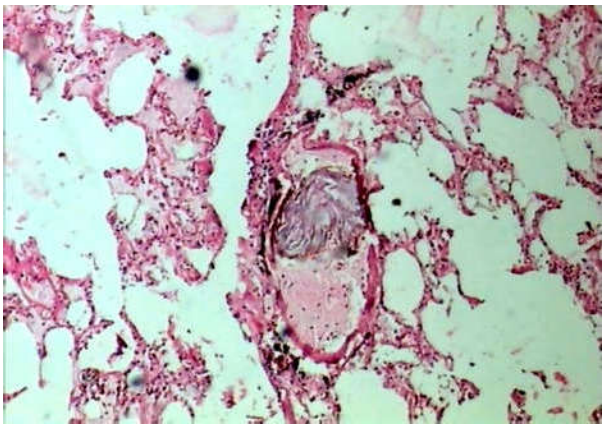

Fig. 1: Photomicrograph showing pulmonary arteriole filled with massive fetal squamous cells. (H-E; original magnification, x40).

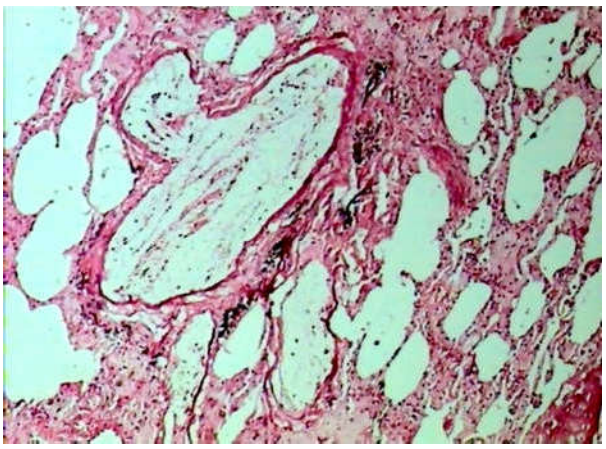

Fig. 2: Photomicrograph showing pulmonary arteriole completely filled with mucin. (H-E; original magnification, x40)

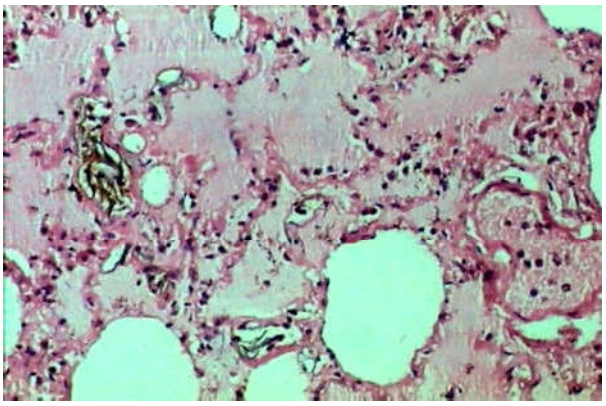

Fig. 3: Photomicrograph showing pulmonary edema and inter alveolar capillaries filled with fetal squamous cells. (H-E; original magnification, x40)

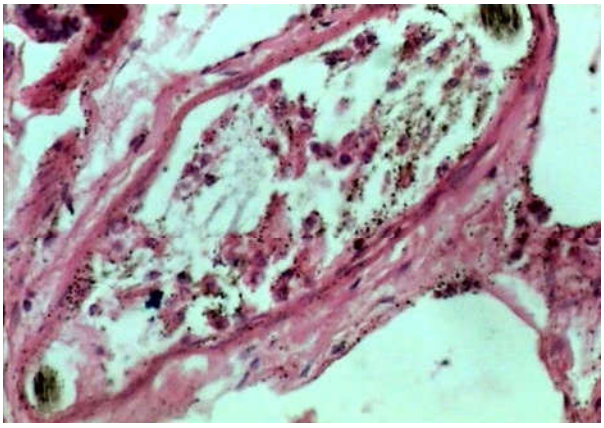

Fig. 4: Photomicrograph showing lanugo hair and amniotic fluid debris. (H-E; original magnification, x40)

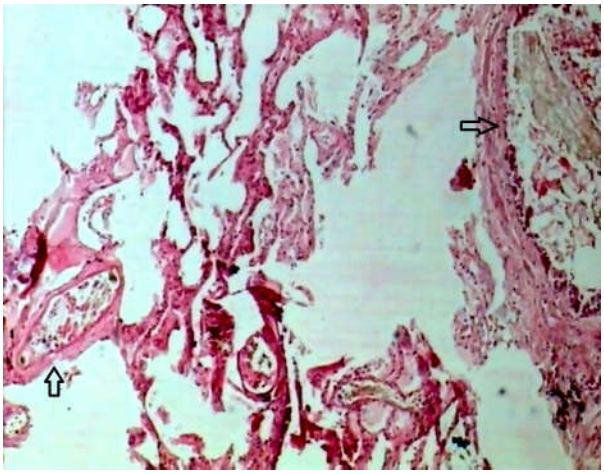

Fig. 4: Photomicrograph showing lanugo hair and amniotic fluid debris. (H-E; original magnification, x40)

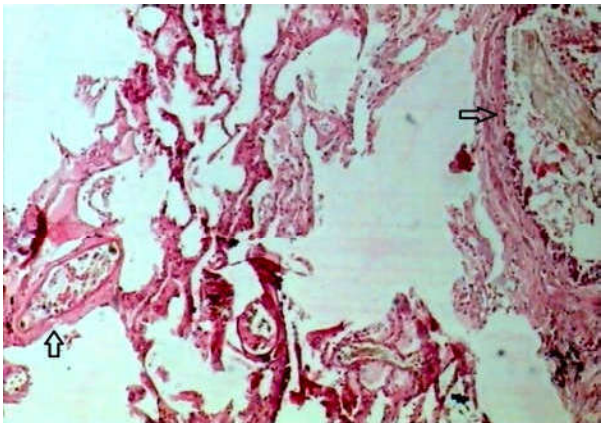

Fig. 5: Photomicrograph showing vegetative material in terminal bronchiole (Horizontal arrow) and mucin with lanugo hair (vertical arrow) in a pulmonary microvasculature. (H-E; original magnification, x40)

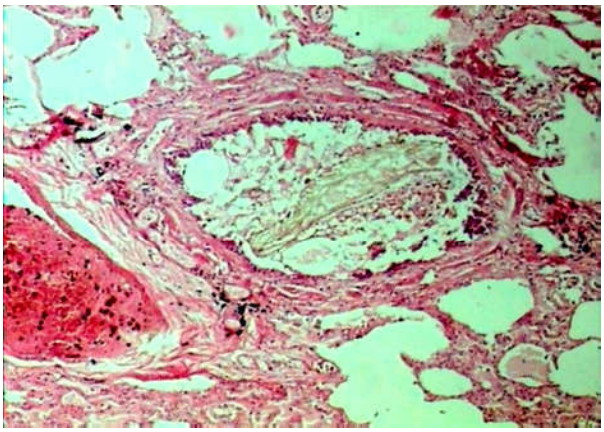

Fig. 6: Photomicrograph showing vegetative material in terminal bronchiole. (H-E; original magnification, x40)

Discussion

United Kingdom Obstetrics Surveillance System (UKOSS) program has been studying AFE and aspiration of gastric contents occurring as separate entities during pregnancy and labor [15]. However as in our case, both of these conditions may occur simultaneously. Aspiration of solid, semi-solid gastric contents in a tracheobronchial tree is easy to visualize and pose no diagnostic problem. However aspiration of aparticulate gastric acid juice in the respiratory system is very difficult to diagnose as it is liquid in nature and disappears rapidly [16]. In case of AFE aspiration of aparticulate acid gastric juice may not be noticed on histological examination as acid gastric juice gets miscible with pulmonary edema fluid caused by both AFE as well as aspiration. A tell-tale sign of aspiration is presence of a particulate material (vegetative material as in our case) in terminal bronchioles and alveoli. Recognition of aspiration with AFE is important as simultaneous occurrence of AFE and aspiration of gastric contents will speed up the development of hypoxia which may culminate in instantaneous death of mother.

Pulmonary aspiration is defined as the inhalation of foreign material into the lower respiratory tract beyond vocal cord [15]. It can cause severe maternal morbidity including acute respiratory distress syndrome (ARDS)/ acute lung injury (ALI), associated organ failure and maternal death. Emergency general anesthesia and pregnancy are the risk factors for gastric content aspiration. Pregnancy increases the risk of aspiration of gastric content due to delayed gastric emptying which may be attributed to effects of progesterone on lower esophageal sphincter and gastric smooth muscle. It also causes airway edema with congestion resulting in difficult prolonged intubation thus prompting for aspiration. The patient may aspirate stomach contents during labor. The labor itself and lithotomy position are responsible for this. The consequences depend on the period of survival after the aspiration. If patient subsists often develop aspiration

pneumonia and abscess [42]. However there are no epidemiological data on maternal pulmonary aspiration in pregnancy and its effects on the affected women. Also the data on the management of aspiration in pregnancy, as well as the subsequent maternal, fetal and neonatal morbidity and mortality, is sparse [15,43].

Pulmonary aspiration may be clinically asymptomatic (silent aspiration/unwitnessed) or clinically manifested by any combination of bronchospasm, hypoxia, cough, breathlessness and hypotension [43-55]. Witnessed aspiration event is characterized by presence of food particles in tracheobronchial tree [54]. Unwitnessed gastric aspiration is very difficult to diagnose and therefore it is difficult to estimate true incidence of aspiration induced lung injury [42].

The lung injury caused by aspiration of gastric contents ranges from mild subclinical pneumonitis to severe respiratory failure. The extent and progression of lung injury is determined by composition of aspirate [42]. Aspiration events lead to either aspiration pneumonitis or aspiration pneumonia. The aspiration pneumonitis evolves into two phases, phase-1occurs immediately after aspiration and is characterized by intense coughing and bronchospasm. The Phase-2 is characterized by onset of acute inflammation in the pulmonary parenchyma over next 4-6 hours [56].

In cases of sudden death, gastric contents are commonly found in upper respiratory tract at autopsy. It may be either agonal or early postmortem spill or antemoretem (true vital aspiration). It is not possible to distinguish between these entities unless clinical or witnessed evidence and/or histological findings of acute lung injury is available as there is no gold standard for diagnosis of aspiration induced lung injury [42-57]. Aspiration should be differentiated from regurgitation, The latter is characterized by reflux of gastric contents into oropharynx or esophagus without entry into lungs [42]. Agonal regurgitation of gastric contents occurs during pumping of chest and abdomen, commonly practiced resuscitation attempts. Thus finding of gastric contents in upper respiratory tract is medico legally less significant and could not be ascribed as a sole cause of death [57]. A death due to aspiration can only be attributed if the air passage beyond the level of vocal cord is completely occluded by food and most commonly seen in patient who have compromised central nervous system [58]. On other hand, presence of aspirated particulate gastric contents in smaller bronchi and terminal bronchioles with minimal inflammation substantiates the aspiration to be antemoretem in cases of sudden death [59-60].

Conclusion

Aspiration of gastric content may occur simultaneously with AFE particularly in an unconscious state leading to acute cardio-respiratory collapse and death thereby reducing the interval between the onset of symptoms of AFE and death.

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