AMNIOTIC FLUID EMBOLISM: A CASE REPORT

Dr. K. Sudheesh M.D, DNB. *Senior Resident, Dept of Anesthesiology, MMC & RI, Mysore*
Dr. K.A. Rasheed D.A. *Consultant Anaesthesiologist, Orchid hospital, Malappuram, Kerala*
Dr. Sajna Thayyil D.G.O. *Consultant Gynaecologist, Orchid hospital, Malappuram, Kerala*

INTRODUCTION:

Amniotic fluid embolism is a rare catastrophic and life threatening complication of pregnancy (Delivery?) that occurs in setting of a disrupted barrier between amniotic fluid and maternal circulation.

Onset of signs and symptoms are dramatic and abrupt in form of dyspnoea, hypotension, loss of consciousness, and arterial hypoxemia. Foetal distress is also present at the same time. More than 80% of parturients experience cardio pulmonary arrest. Coagulopathy resembling DIC is common. There is a dearth of publications reporting non-fatal episodes of AFE presumably because mortality for both mother and fetus is usually high despite aggressive therapeutic interventions. Here we report anaesthetic management in a case of suspected amniotic fluid embolism.

CASE REPORT:

A 30 year old 5th gravida in active labour presented (Peripheral Location of the Hospital?) with sudden onset chest pain, breathlessness and giddiness, 15minutes following artificial rupture of membranes. She had an uneventful ante natal period and previous pregnancies were uneventful. Patient was hemodynamically stable before artificial rupture of membranes with blood pressure being 120/70 mm of Hg. Patient was resuscitated with supplemental oxygen and 1 Litre of i. v. fluids (RL) and, the patient was rushed into operation theater for “crash LSCS” in view of sudden onset maternal and fetal distress.

On pre anaesthetic examination, patient was drowsy and agitated, was in severe respiratory distress (shallow rapid breaths with active accessory muscles) and was profusely sweating. She had tachycardia and blood pressure was 130/90 mm of Hg. Her basal saturation was 92% which increased to 95% with 100% oxygen supplementation through mask . Cardio vascular examination revealed loud P_2 with a systolic murmur at the tricuspid area. Respiratory system revealed rhonchi in right infraaxillary and subscapular regions. Investigations during her previous visit was, Hb% - 10 gm%, BT – 45 sec, CT – 3min 20sec. Monitors connected were SpO_2, NIBP,& 5 lead ECG. Premedication with Inj. Ranitidine 50 mg i.v and Inj. Metoclopramide 10 mg i.v was given before shifting the patient to O.T.

Patient was preoxygenated in left lateral position and rapid sequence induction with Inj. Propofol 75 mg, Inj. Ketamine 50 mg (Why 2 Induction Agents?) and intubation facilitated with Inj. Succinylcholine 75 mg. Patient was intubated with 7.0mm cuffed endotracheal tube. Inj. Deriphylline 2ml and inj. Hydrocortisone 100 mg was given just before induction.

She was maintained on ketamine infusion(10-15 µg / kg / min(? Wt of the Patient)) and intermittent doses of vecuronium.(Actual Dose of Drugs?) Intermittent positive pressure ventilation was done with 100% O_2 initially, N_2O was added towards the end stages of surgery. Oxytocin 10 units was administered by infusion after extraction of baby. Intra operative monitoring consisted of SpO_2, NIBP, ECG, $EtCO_2$, Paw, PIO_2 (What are the intraoperative $EtCO_2$ & PIO_2 ?)

Intra operatively, patient had SpO_2 varying between 94% - 96% in the initial 30 min inspite of continuing positive pressure ventilation with 100% O_2. Rhonchi gradually disappeared after 30 min and SpO_2 stabilized permitting addition of N_2O. Blood pressure which was near normal preinduction dropped to 70 mm of Hg systolic (Was it due to Propofol 75 mg?) with diastolic pressure being unrecordable, in the post induction period and persisted so for initial 20 - 30 min and slowly stabilized to 110/70 mm of Hg towards the end of surgery with fluid resuscitation and blood transfusion. (Quantity of Blood?) *Interestingly,* there was persistant bleed from the uterus and also from the incision sites. Uterine bleed did not respond to oxytocics, massage and hemostatic sutures, hence an emergency decision to go for hysterectomy was taken. Bleeding was controlled only after transfusion of 2 units of fresh blood and administration of styptic agents (tranexemic acid 1 gm i.v). Abdomen was closed after complete hemostasis. Surgery was completed by 2 hours. Patient was extubated after complete reversal of neuromuscular blockade with Neostigmine and atropine at the end of surgery.

Baby when extracted was in severe distress with an APGAR of 4/10 which improved to 8/10 at 5 min with thorough suctioning, bag and mask ventilation and chest compressions.

Post operatively, patient was monitored in post operative care unit for 24 hrs. she was asymptomatic and saturation maintained at 98% - 99% with O_2 supplementation. Chest auscultation revealed scattered crepitations in the lower lung fields. There were no rhonchi. Post operative X- ray chest revealed mild haziness in right lower zone with prominent bronchovascular markings bilaterally. Post operative Hb% was 7.8 gm% and Prothrombin time was 16 sec (control 14 sec). Post operative treatment included steroids, antibiotics, nebulization, IV fluids and blood transfusion. Patient was shifted from the post operative ward after 24 hrs. recovery was uneventful from then onwards.

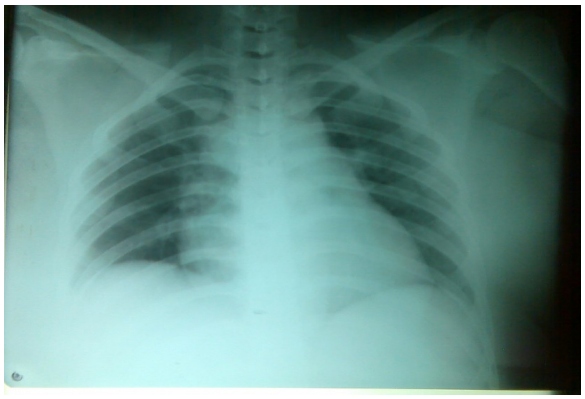

Figure 1. post operative Chest X ray AP View

DISCUSSION:

Amniotic fluid embolism was first described by Meyer in 1926 but it was only brought to prominence in 1942 when Lushbaugh and Steiner reviewed the unexpected deaths of 8 patients and found material consistent with amniotic fluid debris in the pulmonary vasculature. Though incidence varies from 1:8000 to 1:80000, there has been increase in reporting due to increased awareness[1]. A recent report by Clark *et al* [2] cited a 61% maternal mortality rate (MMR), with 15% of the survivors remaining neurologically intact. In addition, the demise rate of fetuses *in utero* at the time of a symptomatic AFE event was 61% (2). Analogous to our report, documentation of survival from AFE has appeared in the peer-reviewed literature very recently [3,4,5,6]. Patient can present with symptoms varying from mild dyspnoea and tachycardia to seizures and loss of consciousness depending upon the size of embolus.

The signs and symptoms are not only due to mechanical obstruction but also due to release of undefined chemicals such as prostaglandins, leukotrienes, serotonin or histamine leading to pulmonary hypertension, intrapulmonary shunting, pulmonary edema, bronchoconstriction, and severe hypoxia. Hypoxemia is due to V/Q mismatch and circulatory instability can occur due to right ventricular obstruction. Coagulopathy is thought to be precipitated by several procoagulant components of amniotic fluid, most notably thromboplastin, which initiate the extrinsic pathway of the clotting cascade and result in excessive fibrinolytic activity [7]. Predisposing factors once considered to be associated with AFE include placental abruption, uterine overdistention, fetal death, trauma, tumultuous or oxytocin-stimulated labor, multiparity, advanced maternal age, and rupture of membranes.[8] Our patient presented with abrupt onset of symptoms following increase in intensity of contractions, 15 min after rupture of membranes.

The presenting signs and symptoms in Morgan's series were respiratory distress in 51%, hypotension in 27%, coagulopathy in 12% and seizures in 10% (9) However, our subject typically presented with respiratory distress, desaturation and coagulopathy.

Though blood pressure was normal initially largely owing to the resuscitative efforts in the labour room and also probably due to sympathetic response to anxiety, it dropped to 70 mm of Hg immediately after induction of anaesthesia.

There is unfortunately no routine or standardized diagnostic scheme to confirm AFE and hence it remains a diagnosis of exclusion. A review of the largest case-series to date concluded that the physiologic and haematologic sequelae of AFE resemble septic or anaphylactic shock rather than an embolic phenomenon ([10]) and hence this obfuscates matters. The plethora of differential diagnoses include air or thrombotic pulmonary emboli, septic shock, acute myocardial infarction, cardiomyopathy, anaphylaxis, aspiration pneumonitis, placental abruption, eclampsia, uterine rupture, transfusion reaction and local anaesthetic toxicity ([11]). There are no laboratory investigations which are pathognomonic of AFE but the following may support the diagnosis: complete blood count, coagulation profiles, arterial blood gases, serum tryptase, chest x-ray, ventilation-perfusion lung scans, ECG and echocardiography (12). Masson *et al.* (11) also described a method based on blood obtained from the pulmonary microvasculature where components of amniotic fluid i.e., squamous cells and mucous strands can be identified. Other novel studies which involve testing maternal serum for TKH-2 antibodies ([13]) and zinc coproporphyrin ([14]) seem promising, though further studies are needed to elucidate their practical utility and reliability (12).

Our patient was awake through out progress of labour and had not received any labour analgesics. She was not a known case of bronchial asthma nor had any pre existing respiratory symptoms or infections prior to the onset of labour. No drug had been administered without test dosing to suspect drug hypersensitivity and there was no bleeding P/V following rupture of membranes or evidence of abruption during cesearean. This provoked us to suspect amniotic fluid embolism.

A proper cardiac evaluation could not be done in the immediate pre operative period owing to the emergency nature of surgery. However, intraoperative 5 lead ECG monitoring did not reveal any arrhythmias or ischemia. Ketamine was used as co induction agent as intra operative hemodynamic instability was anticipated and also in view of bronchodilation. Central line was not attempted because of lack of time and facilities. NIBP and urine output gave a fair idea of circulatory status. Post operative elective ventilation was planned but since the patient showed good recovery towards end of surgery a trial of extubation was given and it was successful. Being a remote place, FFp and cryoprecipitate was not readily available in our hospital. Availability of fresh blood annulled the problem to a certain extent, but could not prevent hysterectomy.

There is no definitive treatment in amniotic fluid embolism. The management of AFE is essentially empirical and supportive, directed towards the maintenance of oxygenation, ventilation, circulatory support and correction of coagulopathy (12). Circulatory support with fluids and inotropes, correction of coagulation abnormalities with blood, FFP, cryoprecipitates and pharmacological agents is recommended[2]. Bleeding not amenable to conservative measures have to be corrected with hysterectomy as was in our case.

Recently, the successful application of extracorporeal membrane oxygenation and intraaortic balloon counterpulsation (7), as well as cell-salvage combined with blood filtration ([15]) has been documented. Other proposed modalities which allegedly have salutary effects on AFE outcome include serine protease inhibitors, cardiopulmonary bypass, nitric oxide, inhaled prostacyclin and high-dose corticosteroids (12).

CONCLUSION:

To conclude , Amniotic Fluid Embolism continues to be one of the most feared and devastating complications of pregnancy. Despite recent documented evidence that attendant mortality rates may be on the wane (1), it yet remains an enigmatic, unpredictable, unpreventable and for the most part, untreatable disease with generally dismal clinical outcomes. We presented a multiparous lady with no premorbid history who presented with symptoms and signs suggestive of amniotic fluid embolism, the outcome in our case is uncommon and underlines the need for high index of suspicion and prompt resuscitation to save the lives of both mother and baby.

References:

1. Kian-Hian Tan, Yen-Yen Sim, Jen W. Chiu, Chee-Chuen Loo, Seow-Woon Yeo: Maternal-fetal Survival Following Amniotic Fluid Embolism: 2 Case Reports. *The Internet Journal of Anesthesiology.* 2002. Volume 5 Number 4.
2. Clark SL, Hankins GDV, Dudley DA. Amniotic fluid embolism: analysis of a national registry. Am J Obstet Gynecol 1995; 172:1158-69
3. Fletcher SJ, Parr MJ. Amniotic fluid embolism: a case report and review. Resuscitation 2000;43:141-6
4. Green BT, Umana E. Amniotic fluid embolism. South Med J 2000;93:721-3
5. Awad IT, Shorten GD. Amniotic fluid embolism and isolated coagulopathy: atypical presentation of amniotic fluid embolism. Eur J Anaesthesiol 2001;18:410-3

6. Hsieh YY, Chang CC, Li PC, Tsa HD, Tsai CH. Successful application of extracorporeal membrane oxygenation and intraaortic baloon counterpulsation as lifesaving therapy for a patient with amniotic fluid embolism. Am J Obstet gynecol 2000;183:496-7

7. Katherine J. Perozzi, RN, MSN Crit Care Nurse 2004 Aug; 24(4): 54-61

8. Sisson MC. Amniotic fluid embolism. *Crit Care Obstet*. 1992;4:667–673.

9. Morgan M. Amniotic fluid embolism. Anaesthesia 1979;34:20-32.

10. Locksmith GJ. Amniotic fluid embolism. Obstet Gynecol Clin North Am 1999;26:435-44.Clark SL. New concepts of amniotic fluid embolism: a review. Obst Gynechol Surv 1990; 45:360-8.

11. Masson RG. Amniotic fluid embolism. Clin Chest Med 1992;13:657-65.

12. Davis S. Amniotic fluid embolus: A review of the literature. Can J Anesth 2001;48:1:88-98.

13. Oi H, Kobayashi H, Hirashima Y, Yamazaki T, Kobayashi T, Terao T. Serological and immunohistochemical diagnosis of amniotic fluid embolism. Semin Thromb Hemost 1998;24:479-84.

14. Kanayama N, Yamazaki T, Narusc H, Sumimoto K, Horiuchi K, Terao T. Determining zinc coproporphyrin in maternal plasma- a new method for diagnosing amniotic fluid embolism. Clin Chem 1992;38:526 - 9.

15. Waters JH, Biscotti C, Potter PS, Phillipson E. Amniotic fluid removal during cell salvage in the cesarean section patient. Anesthesiology 2000;92:1531-6