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Amniotic fluid embolism-Vagaries of presentation

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ABSTRACT

Entry of amniotic fluid into the maternal circulation leads to dramatic sequel of clinical events, characteristically referred to as amniotic fluid embolism.It is the fifth most common cause of maternal mortality in the world. It's onset can neither be predicted nor prevented. First described in 1941,approximate incidence is 1 in every 20,646 deliveries, exact pathophysiology is still unknown. Some authors have proposed the term anaphylactoid syndrome of pregnancy. In a short span of weeks we have encountered 3 such cases. First case developed breathlessness and cyanosis within 5 minutes of rupture of membranes following misoprostol induced labour. Second case presented with breathlessness and progressed to DIC following outlet forceps delivery. Third case presented with breathlessness following rupture of membranes. All the 3 patients could not be saved inspite of all resuscitative measures. No definitive clinical or laboratory test is available for its diagnosis. Criteria for diagnosis of amniotic fluid embolism as per Clark's national registry are cardiac arrest, dyspnoea and cyanosis, coagulopathy, onset during labour or C-section or within 30 minutes post partum and absence of other significant pathology causing such clinical features. A team approach among obstetrician, anaesthesiologist and intensivist is necessary for successful outcome. Despite early intervention maternal and fetal mortality remains high.

INTRODUCTION

The disastrous entry of amniotic fluid into the maternal circulation leads to variety of clinical events , characteristically referred to as amniotic fluid embolism. These clinical events are respiratory distress, cyanosis, hypotension, coagulopathy, neurological manifestations like confusion, seizures and coma. It is a rare catastrophic obstetric emergency occurring during pregnancy, labour or within few post partum hours. Most experts agree that amniotic fluid embolism cannot be predicted nor prevented .Incidence varies widely because of lack of sensitive and specific diagnostic studies leading to both over and under reporting. According to Gilbert [1] incidence is 1 in 20,646 deliveries. Passage of amniotic fluid was first reported by Meyer[2] in 1926 and later identified as syndrome in 1941 by Steiner and Lushbagh[3]. It is estimated to be fifth most common cause of maternal mortality rate in the world. Maternal mortality rate is 61%, many surviving mothers have neurological deficits and fetal mortality is 21% and 50% surviving neonates have permanent neurological injury[4].The pathophysiology of amniotic fluid embolism is still not completely understood. It might result from immune activation. Early recognition and aggressive resuscitive efforts by multidisclipenary approach enhance the probability of maternal and neonatal survival. Herewith, we are reporting 3 such cases with vagaries of presentation.

CASE REPORTS

Case 1:

Mrs. XY,28 years aged, primigravida with full term pregnancy, was admitted with labour pains and breathlessness since 5 hours. She had pre-mature rupture of membranes since 8 hours. On examination patient was conscious and oriented ,pulse rate was 130 bpm,blood pressure was 90/60 mmHg ,mildly anaemic, oedema was absent. Respiratory system examination revealed bilateral crepts and rhonchi , respiratory rate was 40 cycles per minute .On per abdomen examination uterus was contracting once in 3 minutes for 40 seconds, head was engaged, fetal heart sound was absent. On per vaginal examination cervix was fully dilated , membranes were absent , vertex at +1 station,caput was present, meconium stained liquor was seen.

Investigations showed her haemoglobin as 7.1 gm%, total count - 24,000cumm,SpO2 70%, chest x-ray showed consolidation of right lower lobe, patchy consolidation of left para hilar and paracardiac region. Other investigations were within normal limits. Injection ceftriaxone 1 gm intravenously stat and 100cc metronidazole intravenously stat was given. She was delivered by outlet forceps with episiotomy and fresh still born female baby was extracted. Because of persistent tachycardia ,hypotension and falling oxygen saturation ,physician's and anaesthesiologist's opinion was taken and she was put on ventilator. Dopamine drip was started .Injection effcorlin and deriphylline were given. She developed cardiac arrest and could not be saved inspite of Cardio-pulmonary resuscitation.

Case 2 :

Mrs. AB, 33 years aged gravida 4 para 3,was admitted at 41 weeks period of gestation for induction of labour. She was induced with vaginal misoprostol 25mcg and repeated after 6 hours .Membranes ruptured spontaneously 6 hours after the second dose. After 5 minutes of spontaneous rupture of membranes patient complained of breathlessness and was restless. On examination patient was confused ,pulse rate was 110 bpm, blood pressure was 180/100 mmHg ,not anaemic and oedema was absent. On respiratory examination there was bilateral crepts ,respiratory rate was 40 cycles per minute and cyanosis was present. On per abdomen examination uterus was contracting once in 4 minutes for every 35 seconds, cephalic presentation ,and fetal heart rate was 140 per minute and regular. On per vaginal examination cervix was fully dilated ,membranes were absent, vertex at +1 station. Investigations were within normal limits.SpO2 was 90%. Oxygenation was started, Injection hydrocortisone 200 mg intravenously stat was given, Injection Deriphylline intravenously stat and duolin nebulisation was given. Patient delivered a live female baby and collapsed within 5 minutes of expulsion of placenta. Physician's and anaesthetist's advice was taken and patient was intubated and dopamine drip was started. Injection adrenaline 1cc intravenously stat and injection atropine 1cc intravenously stat was given. Cardio-pulmonary resuscitation was continued ,but inspite of all measures patient could not be revived.

Case 3 :

Mrs. CD, 30 years aged primi para, presented with complaints of breathlessness after 1 hour of delivery. She had delivered a live female baby by outlet forceps delivery outside the institution .On examination patient was disoriented and confused, pulse rate was 140 bpm, thready, blood pressure was not recordable, she was severely anaemic and oedema was absent. On respiratory system examination there was bilateral crepts and respiratory rate was 38 cycles per minute. On per abdominal examination uterus was well contracted and retracted, gaseous distension was present .On per vaginal examination there was high vaginal wall tear in left lateral vaginal wall.It was friable vagina and minimal bleeding was present. Investigations showed her haemoglobin level as 5.9 gm %,total count was 41,000 cumm, clotting time was more than 10 minutes, PT INR was 100, control : 14.8. and test :5.Her blood urea ,serum creatinine ,uric acid and liver function tests were increased . Other investigations were within normal limits. Physician's and anaesthetist's opinion was taken and patient was intubated and oxygenation was started , dopamine

Table 1. Clinical associations with amniotic fluid embolism

Maternal risk factors	Advanced maternal age
	Pre eclampsia/eclampsia
	Trauma
	Diabetes
Neonatal risk factors	Intra uterine death
	Fetal distress
	Fetal macrosomia
Complications of pregnancy that have been linked to amniotic fluid embolism	Placenta previa
	Placental abruption
	Operative delivery
	Recent amniocentesis Meconium stained amniotic fluid Uterine overdistension Chorioamniotis
	Induction of labour
	Rupture of amniotic membrane Uterine rupture
	Cervical laceration
	Saline amnioinfusion
	Cell salvaged blood transfusion.

Table 2. Clinical features of Amniotic fluid embolism

Signs and symptoms	Frequency (%)
Hypotension	100%
Acute dyspnoea and/ or cyanosis	83%
Fetal distress	100%
Pulmonary oedema or ARDS	93%
Cardio pulmonary arrest	87%
Cyanosis	83%
DIC	83%
Seizures	48%
Mental status changes	50%
Uterine atony	23%
Cough ,headache and chest pain	7%

Table 3. Differential diagnosis of amniotic fluid embolism

Respiratory distress
Pulmonary embolism
Pulmonary oedema
Anaesthesia complications
Aspiration
Hypotension and shock related symptoms
Septic shock
Hemorrhagic shock
Anaphylactic reaction
Myocardial infarction
Cardiac arrhythmias
Haemorrhage and bleeding disorders
Disseminated intravascular coagulation
Placental abruption
Uterine rupture
Uterine atony
Neurological and seizure–related conditions
Eclampsia
Epilepsy
Cerebrovascular accident
Hypoglycaemia

and dobutamine drip was started. She was put on ventilator.3 units of packed cells were given along with intra venous fluids .Patient deteriorated and developed acute cardiopulmonary arrest .Injection adrenaline 1cc intravenously and injection atropine 1cc intravenously was given but patient expired inspite of all resuscitative measures taken to save her.

DISCUSSION

Amniotic fluid embolism remains a diagnosis of exclusion and should always be considered early in the clinical management of any obstetric emergency involving cardiovascular collapse. These 3 cases presented with breathlessness and cyanosis followed by hemodynamic instability and cardiovascular collapse. Normally amniotic fluid does not enter maternal circulation because it is contained safely within the uterus sealed off by the amniotic sac [5].Amniotic fluid embolism occurs when the barrier between amniotic fluid and maternal circulation is broken and amniotic fluid enters the maternal venous system via the endocervical veins, placental site (if separated), or a uterine trauma site. Amniotic fluid embolism more closely resembles an anaphylactoid reaction to fetal debris (squames,hair,vernix) than an embolic event and the term “anaphylactoid syndrome” is proposed by many authors [6].

Three phases in the clinical course of amniotic fluid embolism have been described. Phase I is characterised by respiratory distress, cyanosis, altered mental status and hemodynamic collapse.Phase II involves coagulopathy and haemorrhage (DIC) and Phase III includes tissue injury and end organ failure.

Diagnosis

The finding of fetal debris in the maternal pulmonary circulation, once considered pathognomic is neither specific nor sensitive. Therefore diagnosis must be based on clinical features and should not be confused with other pregnancy related complications or medical conditions.

Increased serum tryptase and urinary histamine concentrations as well as significantly lower serum complement concentrations suggest anaphylactoid process. More studies are also needed to determine the utility of both monoclonal TKH-2 antibodies and zinc co-proporphyrin as rapid diagnostic markers[7,8]. However in cases of maternal death after suspected Amniotic fluid embolism, post mortem histopathologic and histochemical analysis may further support the clinical diagnosis of Amniotic fluid embolism.

Management

The initial management of Amniotic fluid embolism relies on early suspicion and early aggressive hemodynamic support in intensive care unit.1) Oxygenation the goal of oxygen therapy is to maintain arterial oxygen saturation at 90% or more[9].Endotracheal intubation and positive pressure ventilation is required.2) Circulation trendelenburg position to improve venous return, supportive measures include fluid therapy, pharmacological agents like methergin,oxytocin and prostaglandins, electro-cardiographic monitoring, volume replacement with isotonic crystalloid solution and ionotrophic agents like dopamine and epinephrine for maintaining cardiac output and blood pressure. 3) Control of haemorrhage and Coagulopathy- uterine massage, pharmacological agents, blood transfusion and blood components and hysterectomy if necessary. As demonstrated in this , Cardio pulmonary resuscitation may be ineffective. It has been reported that immediate Caesarean section

will improve neonatal neurological recovery and overall maternal outcome if performed within 5 minutes of maternal cardiovascular arrest[10] .Maternal resuscitative efforts are also enhanced by relief of aorto-caval compression at delivery. More invasive approaches to resuscitation have been reported including exchange transfusion, extra corporal membrane oxygenation, cardiopulmonary bypass , a right ventricular assist device and uterine artery embolisation. Recombinant factor VII is also found to be effective. Both aerolized prostacyclin and inhaled Nitrous oxide act as direct pulmonary vasodilator and have been successfully used to treat the acute pulmonary vasoconstriction of amniotic fluid embolism.

CONCLUSION

Amniotic fluid embolism is an unpredictable, unpreventable and for the most part an untreatable obstetric emergency. Management of this condition includes prompt recognition of the signs and symptoms , aggressive resuscitation efforts and supportive therapy. Any delay in diagnosis and treatment can result in increased maternal and/ or fetal impairment or death. A team approach among obstetrician, anaesthesiologist and intensivist is necessary for successful outcome. Despite early intervention, maternal and fetal mortality remain high. Whereas once the invariable outcome of the amniotic fluid embolism was death of the mother; today the prognosis is somewhat brighter, thanks to increased awareness of the syndrome and advances in intensive care medicine.

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