Amniotic Fluid Embolism - A Survival Story

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ABSTRACT

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Introduction and objective: Amniotic fluid embolism is an obstetric catastrophe which occurs in 1 in 8000-80,000 deliveries and carries 80% mortality even in developed countries. It is postulated that amniotic fluid, fetal cells, hair, or other debris enter the maternal circulation, and causes cardio respiratory collapse. A case of amniotic fluid embolism with successful recovery is described in this case report Case report: There was sudden hypotension and tachycardia 10 minutes after the delivery of the baby and uterine atony followed 2 minutes later. Coagulopathy also set in while the uterus was being closed 20 minutes after the delivery of the baby, which was proven by the lab parameters. The survival of the patient is due to the immediate measures taken to correct the hypotension metabolic acidosis, coagulation abnormalities replacement of necessary blood products Discussion: Strong clinical suspicion of amniotic fluid embolism and team work of the Anesthetists, Obstetricians, Intensivists and the blood bank officers with all the supportive staff helped in the survival Conclusion: Good antenatal care, institutional delivery, strong instinct for diagnosis and prompt supportive measures are the key factors in bringing down the mortality rate.

Keywords: Amniotic-Fluid, Embolism, AFE, Obstetric-Complications

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BACK GROUND

Amniotic fluid embolism (AFE) is an obstetric catastrophe where the women in labor suddenly collapse, which occurs in 1 in 8000-80,000 deliveries and carries 80% mortality even in developed countries.[1] It is postulated that amniotic fluid, fetal cells, hair, or other debris enter the maternal circulation, and causes cardio-respiratory collapse.[2] Steiner and Luschbaugh described AFE for the first time in 1941 after they found fetal debris in the pulmonary circulation of women who died during labor, post mortem.[1] Data from the National Amniotic Fluid Embolus Registry describes the process of AFE as similar to anaphylaxis than to embolism. The term anaphylactoid syndrome of pregnancy is now penned because fetal tissue or amniotic fluid components are not found in all women who present with signs and symptoms similar to AFE.[4] The diagnosis is essentially one of exclusion based on clinical presentation. Other causes of hemodynamic instability should not be neglected. In a critically ill patient, a blood sample obtained by aspiration from pulmonary artery catheter that contains fetal squamous cells is considered suggestive of but not diagnostic of AFE syndrome.[3] At autopsy amniotic fluid embolism is diagnosed when fetal squamous cells are found in the maternal pulmonary circulation. Fetal squamous cells are commonly found in the circulation of patients in labor but all these patients do not develop the syndrome. High degree of suspicion aids in diagnosis and treatment is generally supportive. Over the last 30 years the improvement in technology and availability of the supportive measures has brought down the mortality to around 50% in advanced nations.[1]

CASE REPORT

A case of twenty two year old primi gravida was admitted for safe confinement at 38 weeks and 6 days of gestation. She has been undergoing regular antenatal checkup from this same hospital from initial diagnosis of pregnancy onwards. She was on diet control for gestational diabetes. Her blood group was B + and Hb level was 12.9gm% in the third trimester. Her antenatal period was uneventful.

Induction of labor was done after priming of cervix with PgE2 gel at 38 completed weeks of gestation one day after admission. Labor was augmented with artificial rupture of membranes (ARM) and oxytocin. The labor was progressing normally with normal cardiotocogram. Two hours after ARM, cardiotocogram showed persistent fetal bradycardia, in spite of

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Table 1. Intraoperative events

Time	PR	SBP	DBP	SpO2	IVF	BldProducts	Drugs	Remarks
Birth of baby	88	140	70	98	NS		Oxytocin 20U IV Methergin, IV Atracurium IV Midazolam	Delivered male baby
After 10 min	144	90	60	94	RL	RBC	IV phenyl ephrine IVhydrocortisone 200 IV Pg F2α	Bleeding persisting
+10 min	140	90	68	96	6% hes-tryl	PRP		Left radial artery line
+10 min	132	94	70	98		WB	Inj methergin	IJV line with CVP monitoring
+10 min	130	96	70	98	NS	PRP		
+20 min	120	98	70	98		FFP PRP		Plt count 75 000 Bt 8 min Ct 12 min
+10 min	110	100	70	98	NS	FFP		Hysterectomy
+10 min	110	100	70	98	NS	WB PRP	Ca Gluconate,	ABG analysis Met acidosis Hyperkalemia
+10 min	110	98	68	98		FFP	Inj Vit K	
+20 min	108	100	70	98	NS	PRP		
+20 min	110	96	66	96		FFP		
+20 min	112	100	68	98		PRP	Inj Dexamethasone	GRBS-138
+20 min	106	102	70	99	NS	FFB		
+20 min	110	106	72	98		WB PRP	Inj Frusemide 40 mg	
+20 min	100	110	70	99		FFP PRP		Transfer to intensive care unit with ventilatory support

PR- Pulse rate, SBP-Systolic blood pressure,DBP-Diastolic Blood pressure,SpO2 Oxygen saturation, IVF- Intra venous fluid NS- Normal saline, RL- Ringer lactate, Bld products- Blood products, RBC- Packed Red blood concentrate,PRP- platelet rich concentrate FFP- Fresh frozen plasma

oxygen inhalation, left lateral position, stopping of oxytocin. Fetal heart dropped to 60 beats per minute (normal range 120-160 beats/min). Exactly 15 minutes after the fetal bradycardi, decision for emergency caesarian section was taken and the patient shifted to theater. Clinically there was no evidence of abruption as uterus was relaxing in between contractions. There was no bleeding per vagina. Emergency lower segment caesarian section was done under general anesthesia and delivered a male baby at 11.00 AM with a birth weight of 3.045kg. The liquor was clear and mature, and there was no evidence of abruption.

Patient developed hypotension and tachycardia while the uterus was being closed. Within ten minutes time the blood pressure dropped to 90/70 mm of mercury. The radial arterial line was established for infusion. Atonic postpartum hemorrhage started two minutes later and it was not getting controlled with the uterotonic agents, Inj methyl ergometrin and Inj Pg F2α and Inj Oxytocin. Hence, bilateral uterine arteries and the uterine branches of ovarian arteries were ligated, and started closing the abdomen after ensuring hemostasis. Half way through while closing the rectus sheath we noticed generalized oozing from the rectus muscle and needle prick sites. Coagulation profile showed that there was prolongation of bleeding time (BT) and clotting time (CT) and platelet count was 75000/cmm (normal range, 1.5Lakhs to 3.0 lakhs per cmm). Disseminated Intravascular coagulation was diagnosed and decided to do obstetric hysterectomy. The consent was taken and obstetric hysterectomy was done. After hysterectomy, abdomen closed with intra peritoneal and sub rectus sheathdrain. Catheter draining blood stained urine, and the patient was on antibiotics (piperacillin tazobactum combination).

Meanwhile supportive measures were given by the anesthetic team as mentioned in **Table 1.** Arterial blood gas analysis (ABG) two hours after fetal bradycardia showed metabolic acidosis. In total intra-operatively 1 unit of packed cell concentrate, 3 units of whole blood, 6 units of fresh frozen plasma and 8 units of platelet concentrates were given. The patient shifted to post operative intensive care unit with assist control mode ventilatory support three hours and forty minutes after the delivery of baby.

Table 2. Coagulation profile on the day of surgery

Time	BT-sec	CT-sec	PT-sec	Control-sec	INR	APTT Sec	Fibrinogen levels	Platelet Permm3
+1hr	8	12	20	12	1.7	40	60	75000
+2hrs	7	10	16	12	1.3	36	66	76,000
+4 hrs	6	8	15	13	1.2	35	72	1,00,000
+7 hrs	3.20	5.45	15	13	1.2	34	96	1,10,000

Time Delivery of the baby (+) Time after delivery of the baby BT-Bleeding Time, CT-Clotting Time
PT-Prothrombin Time, APTT-Activated Partial Thromboplastin Time

In post operative intensive care unit on day one, 3 units of cryoprecipitate and 2 units of fresh frozen plasma (FFP) were given, and the urine output was normal. X-ray chest taken at two hours post surgery showed clear lung fields. Blood culture has been send on the day of surgery .The report was obtained on day 3 as having no growth. On second post operative day, patient was extubated from ventilator. One unit of packed cell and two unit whole blood were given on day 2 to improve hematocrit values.

On third post operative day, all the drains were removed and also the arterial lines were removed. Oral feeds were started, and she began to start breast feeding her baby. Slowly the patient came back to normal. She had spikes of temperature on post operative day 6, 7 and 8, and was given only supportive care. The patient was discharged on 10th post operative day in reasonably good general condition with haemoglobin level of 7.1 gm%. The Histopathological report obtained was consistent with post partum uterus.

DISCUSSION

There was terminal fetal bradycardia in our case and there was persistent fetal heart deceleration lasting for 5 minutes when the decision for caesarian section was taken. There was sudden hypotension and tachycardia that lasted 10 minutes after the delivery of the baby and uterine atony followed 2 minutes later. Hypoxia was not pronounced as the patient was under general anaesthesia. Coagulopathy also set in while the uterus was being closed 20 minutes after the delivery of the baby, which was proven by the lab parameters mentioned in the **Table 2**. The other probable symptoms discussed later in the article here could not be appreciated in our case as the patient was under general anesthesia.

All the criteria suggested by United States and United Kingdom AFE registry[2] were satisfied in this case and clinical diagnosis of amniotic fluid embolim (AFE) was confirmed. The laboratory investigation reports obtained were also suggestive of AFE there was a coagulation profile abnormality **(Table 3).** The arterial blood gas analysis showed metabolic acidosis **(Table 2).** The haematocrit drop was also pronounced as shown by both blood gas analysis and the lab investigation. There were no ECG changes and the X-ray chest taken did not show any changes of pulmonary embolism.

The survival of the patient is due to the immediate measures taken to correct the hypotension metabolic acidosis, coagulation abnormalities replacement of necessary blood products. Strong clinical suspicion of the diagnosis and team work of the anesthetists, Obstetricians, Intensivists and the blood bank officers with all the supportive staff helped in the survival. The central line blood obtained immediately did not demonstrate any fetal cells. This again is not mandatory for the diagnosis of the condition.

Pathophysiology of Amniotic fluid embolism

The pathophysiology of AFE has not been clearly understood. It is believed that amniotic fluid and fetal cells enter the maternal circulation, and triggers an

Table 3. Arterial Blood Gas analysis

Sample time	+ 2 hrs	2 hrs post operative
Sodium meq/l	136.6	141.5
Potassium meq/l	6.81	3.39
Calcium meq/l	1.030	0.846
Chloride meq/l	106.9	102.9l
pH	7.287	7.452
Partial Pressure Of Oxygen mmof Hg	264.4	289.9
Partial pressure of Carbon dioxide mmof Hg	34.7	35.6
Bicarbonate meq/l	16.2	24.3
Hematocrit%	20.4	13
Sulphurdioxide	99.8	99.9
Corrected CO2 mm of Hg	17.2	25.4l
Base excess	10.4	0.4
Anion Gap	20.2	17.7
Osmolarity mosm/kg	272.5	281.8
Base excess mmol/l	9.4l	0.8
Temperature corrected pH	7.252	7.420

Table 4. Post Operative Investigations

Investigations- units	POD1	POD2	POD3	Reference values
Hemoglobin g/dl	4,8	6.5	7.1	12-15
Packed cell volume%	15	20	23	36-42
Urea mg/dl	64	58	50	20-60
Creatinine mg/dl	2	1.7	1.4	0.6-1.2
Prothrombin Time sec	16	14	12	
Control sec	13	12	12	
International numerical Ratio	1.2	1.1	1	1
platelet count	1.2	1.23	1.28	1.5-4.5
Activated partial thrombo-plastin time sec	40	36	34	<35
Total bilirubin mg/dl	2.9	2.0	1.6	.4-1.2
SGOT(aspartate amino tran-ferase) IU/L	57	50	50	<35
SGPT(Alanine amino trans-ferase) IU/L	31	30	30	<35
Alkaline Phosphatase IU/L	74	72	70	20-70
Total protein mg%	4.1	5.6	5.8	4-6
Albumin gm/dl	2.5	2.8	3.1	3.2-5

anaphylactic reaction to fetal antigens. Fetal material is not always found in the maternal circulation in patients with AFE, Fetal cells are often found in women who do not develop AFE also.

The initial triggering event in AFE is also poorly understood, usually during labor or any other procedure during antenatal period like artificial rupture of membranes, amniocentesis, or amnioinfusion, amniotic fluid and debris, or some as yet unidentified substance, enters the maternal circulation. This triggers a massive anaphylactic reaction, and activation of the complement cascade. The progression of AFE occurs in two phases.[4] Phase 1, pulmonary artery vasospasm with pulmonary hypertension and increased right ventricular pressure causeshypoxia. This hypoxia causes myocardial capillary damage and pulmonary capillary damage, left heart failure, and acute respiratory distress syndrome. Women who survive these events may enter phase II. Phase 2 (Hemorrhagic phase), this is characterized by massive hemorrhage with uterine atony and DIC .Fatal consumptive coagulopathy may be the initial presentation also.

Clinical feature

The syndrome typically occurs during labor, soon after vaginal (70%)[10] or caesarean delivery (19%).[10] It can occur during second trimester abortion, following abdominal trauma, and during amnioinfusion. A patient in the second stage of labor becomes acutely dyspneic with hypotension. She may develop a seizure which is quickly followed by cardiac arrest. Disseminated intravascular coagulation sets in with massive hemorrhage and death follows. All the events progress very rapidly. Currently no definitive diagnostic test exists for AFE. The United States and United Kingdom AFE registries recommend the following 4 criteria, all of which must be present to make the diagnosis of AFE;[1] Acute hypotension or cardiac arrest, acute hypoxia and coagulopathy or severe hemorrhage in the absence of other explanations, all of these occurring during labor, LSCS, dilation and evacuation, or within 30 minutes postpartum with no other explanation of findings.

Laboratory investigation needed

The laboratory investigations needed for diagnosis and monitoring includes; arterial blood gas levels (hypoxia/hypoxemia), complete blood count with platelets, prothrombin time and activated partial thromboplastin time, activated partial thromboplastin time (APTT) may be within normal ranges or shortened, fibrinogen level, blood grouping and RH typing done for cross matching and blood transfusion to be given when required, chest Xray may show features of pulmonary embolism, a 12-lead ECG may show tachycardia, ST segment and T-wave changes, and findings consistent with right ventricle strain etc.

TREATMENT

Treatment is mainly supportive.[4] Administer oxygen to maintain normal saturation, intubate if necessary. Initiate cardiopulmonary resuscitation (CPR) if the patient has cardiac arrest. Treat hypotension with crystalloid and blood products. Use pressor agents when necessary. Pulmonary artery catheterization is done in patients who are hemodynamically unstable. Continuous fetal monitoring and prompt delivery. Coagulopathy is treated with FFP when APTT is prolonged, cryoprecipitate given for fibrinogen level less than 100 mg/dL. Platelet transfusion is indicated for platelet counts less than 20,000/μL.

CONCLUSION

Amniotic fluid embolism is caused by the entry of fetal components into maternal circulation that produces severe systemic reaction similar to anaphylactic shock. The maternal and fetal outcome is grave and hence prompt diagnosis is mandatory. There is no one test which clinches the diagnosis. High degree of suspicion and knowledge of symptoms and signs helps in diagnosis. If prompt treatment is instituted in the form of supportive care with multidisciplinary approach

involving anaesthetists, obstetricians, hematologists and intensivists, the survival rate is improved The advanced countries have brought down the mortality in last 30 years with adequate and prompt supportive care. Good antenatal care, institutional delivery, strong instinct for diagnosis and prompt supportive measures are the key factors in bringing down the mortality rate.

END NOTE

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