BIUC Prevails When All Else Fail As A Successful Haemostatic Method In Uncontrollable PPH In A Case Of Amniotic Fluid Embolism- A Case Report

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ABSTRACT:
Amniotic fluid embolism (AFE) was first reported by Richardo Meyer 1926. Amniotic fluid embolism (AFE) occurs in 1/8000 to 1/80,000 deliveries with a maternal mortality ranging from 26% in a recent report to 86% in earlier ones. Neonatal outcome is generally poor with a mortality rate of 20%–25% and, of the survivors, only 50% may be neurologically intact. The presenting signs and symptoms of AFE involve many organ systems. Acute dyspnea or sudden agitation and anxiety are common premonitory symptoms. It is estimated that approximately 10–50% of patients with AFE present with seizures. Rapid decline in pulse oximetry values or sudden absence or decrease in end-tidal carbon dioxide may be apparent. Hemodynamic compromise quickly follows these prodromal signs. We present here a case of AFE with Disseminated Intravascular Coagulation (DIC) in a tertiary care setting but with a situational resource limitation. The ideal requirements for the case management were not met due to the patient’s rare blood group as well as unavailability of fresh frozen plasma or other sophisticated tools. The baby could not be saved, but with the simple and effective technique of a persistent and sustained Bimanual Internal Uterine Compression (BIUC) over almost an hour the haemorrhage was controlled and the patient could be saved.

KEY WORDS: Amniotic fluid embolism, Bimanual Internal Uterine Compression, Disseminated Intravascular Coagulation.
INTRODUCTION:

Amniotic fluid embolism (AFE) occurs in 1/8000 to 1/80,000 deliveries with a maternal mortality ranging from 26% in a recent report to 86% in earlier ones. In the USA, this condition is the most common cause of peri-partum maternal death and is responsible for roughly 10% of all maternal deaths[1]. The fetal mortality rate although lesser than the maternal mortality rate, is a dismal 21%, and 50% of the surviving neonate experience permanent neurological injury. No data are available from India for comparison.

CASE REPORT:

A 25 years old lady, a primigravida with Rh negative pregnancy with severe PIH (Pregnancy Induced Hypertension) at 38wks gestation was admitted in the antenatal ward with complaints of bilateral pitting pedal edema for one wk. She was booked in a private hospital and there were no features suggestive of imminent eclampsia. She maintained optimal urine output and she perceived fetal movements well. Her obstetric history and past medical history were otherwise unremarkable.

On physical examination She was conscious oriented, her BP was 150/110mm Hg, bilateral pitting pedal edema was evident and knee jerks were normal. Haemogram, Renal Function Tests, Liver Function Tests, Serum uric acid were within normal limits. Non-stress test was reactive indicating that the foetal condition was satisfactory. At zero hour, induction of labour was initiated, indicated by severe PIH as the Bishop score was 3/13, intracervical ripening with foley’s catheter was done. She responded to antihypertensives [Nefidipine].

At 12th hour, foley catheter was removed; further ripening with PGE2 was done. At 18th hour, amniotomy and at 20th hour, oxytocin induction was done. Labour progressed satisfactorily with hypertension under control with calcium channel blocker, however with no imminent symptoms of eclampsia. At 22nd hour of induction, she had sudden onset and progress of dyspnoea and collapsed within five minutes. She was pulseless and suffered cardiopulmonary arrest like a bolt from the blue.

Cardiac massage was initiated, intubation was done, ionotrophic support started and patient was shifted to the ICU and put on ventilator support.

In the post resuscitation phase she was comatose and tachycardic (160 beats/minute) with a blood pressure (BP) of 90/60 mm of Hg. Her systolic BP failed to rise and fluctuated at a low level even with vigorous resuscitation and the foetal heart sounds (FHS) disappeared. Spontaneous bleeding from i.v. puncture sites and vagina ensued within 30 minutes of collapse. At 24th hour, she was in early active phase of labor. Labour progressed with oxytocin, with 3 ionotropes on maximum dose [dopamine, dobutamine and norepinephrine]. Clotting time and bleeding time were prolonged. Evidently disseminated intravascular coagulation was triggered and vaginal bleeding actively commenced.

At 26th hour, She delivered a fresh stillborn male 3.1kg by vacuum assisted forceps delivery and atonic postpartum haemorrhage >800ml followed. She continued to bleed vaginally with uterine atony. Haemoglobin dropped from 10gm% prenatally to 4gm% postnaturally.

Her pulse rate was 190/minute, BP-80 to 90 mm of Hg systolic, and she showed marked pallor, with Hb-4gm%. Fresh whole blood transfusion with A1neg blood was initiated two bags consecutively. PGE1 rectal administration 800 +400 micro gram ,PGF2alpha- 250mg -2 doses was also given. Still uterus was inconsistently contracting and was flabby.

Bimanual internal uterine compression (BIUC) with one hand in the anterior fornix and the other on the posterior aspect of uterus was sustained for 50 minutes. In view of her negative blood
group, a ‘witch-hunt’ for blood products continued. Until three hours postnatal uterine atony was persistent. AT 6th hour postnatal, another bout of atonic PPH ensued. At 7 hours postnatal she continued to be comatose and anuric, and frank haematuria was noted suggestive of acute tubular necrosis. Two bags of whole blood was all that could be procured and administered within 7 hours postnatal & 2 bags Fresh Frozen Plasma started at 7 hours postnatal.

Postnatal day 1. She was comatose with a GCS score of <8, maintained on 3 ionotropes, and ventilator support. Anuria-50ml/24hours continued. Diuretics were administered. APPT-was prolonged. Platelets were 60,000/mm³.

Postnatal day 2. Urine output was 300ml/24hours; with administration of mannitol consciousness was regained and she responded to commands appropriately. Inotropic support and ventilatory support were weaned gradually and then she was extubated. Postnatal day 3, serum urea and creatinine was rising. Urine output 300ml/24hours. Patient ambulated, conscious coherent and free of any neurological deficit. She was discharged and was referred for dialysis, to a hospital of relatives choice where she responded well to dialysis as we followed her progress telephonically.

DISCUSSION:

AFE is a rare but often a fatal complication of pregnancy and its onset can neither be predicted nor prevented [2]. Diagnosis is primarily clinical by exclusion. Clark et al. found, the most common presenting signs and symptoms were hypotension and signs of non-reassuring fetal status (100%), pulmonary edema or respiratory symptoms (93%), cardiac arrest (87%), cyanosis (83%), coagulopathy (83%) and DIC in 83% of patients with AFE [3]. Early recognition of AFE with prompt intervention is critical to successful outcome. Panchabhai et al in his study found that 25%–50% of the parturients die within the first hour of clinical presentation. [4]

Various newer strategies of management have been described-Intra aortic balloon counter pulsation, Extra corporeal membrane oxygen, Serum protein inhibitors, Inhaled NO2, Prostoclylines and high dose corticosteroids. But then the question is how available or accessible are these sophisticated modalities of therapy.

This case has been a typical presentation of amniotic fluid embolism (AFE). Rh negative pregnancy is known to be one of the risk factors for AFE as in this case. The occurrence of sudden onset dyspnea quickly progressing to cardio pulmonary arrest with the other causes excluded, the onset of disseminated intravascular coagulation within an hour of collapse and its aftermath all conform with classical presentation of AFE.

The drop in haematocrit of 6grams%(from Hb 10 to 4gms%) qualifies for massive PPH. Ideally, blood products required for this patient to raise haematocrit to 8gm% would require minimum of 4 bags of whole blood and in the absence of cryoprecipitate, the requirement of fresh frozen plasma would be 15ml/kg bodyweight (here 750ml/4th hourly) until coagulation profile returned to normal. For want of frozen plasma and its kind, the fetus the womankind as well as the professionals interests are at stake. In this case all that she could receive was only 2 bags of whole blood and 7 hours later she received about 200ml of fresh frozen plasma that is about 25% of the requirement in 4 hours. The patient was already hemodynamically labile with fluctuating BP between 60-80 mm Hg systolic for first 4 hours post natal, with maximum dose of 3 ionotropes on Iv infusion. Bimanual internal uterine compression done for 50 minutes for atonic massive PPH has been life saving in this case even in the deficiency of blood products and failure of uterotonics. Multiple doses of uterotonics-PGE1,PGF2 alpha, methyl
ergometrine had proven ineffective in altering the uterine tone.

Based on consistent and good quality patient-oriented evidence, BIUC has proven to be of efficacy as an effective intervention in preventing as well as treating PPH [5]. BIUC here has afforded an effective uterine haemostat and does not need expensive equipments or other accessories and is purely a non pharmacological technique only necessitating the will power and commitment of the gynecologist to pursue this with perseverance.

CONCLUSION:

BIUC could be an effective alternative uterine hemostat and a simple life saving technique and needs due consideration by the care provider when put in a difficult situation with resource limitations.

REFERENCES:


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