International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Vishal AK et al. Int J Reprod Contracept Obstet Gynecol. 2021 Mar;10(3):1188-1191
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20210758

Case Report

A case of amniotic fluid embolism and its sequelae during COVID-19 pandemic: a success story

Anish Kumar Vishal*, Dinesh Bhasin, Krishna Prasad

Department of Obstetrics and Gynaecology, Military Hospital Kirkee, Pune, Maharashtra, India

Received: 01 January 2021
Accepted: 06 February 2021

***Correspondence:**
Dr. Anish Kumar Vishal,
E-mail: anishacme@gmail.com

ABSTRACT

Amniotic fluid embolism (AFE) is an unforeseeable, life-threatening complication of pregnancy with an extremely high mortality rate. This is a complex disorder classically characterized by the abrupt onset of hypoxia, hypotension and consumptive coagulopathy. We experienced a patient who underwent caesarean delivery because of sudden cardiovascular collapse. Intra op she had DIC and was hemodynamically unstable. Surgery was able to complete with inotropes and vasopressors. In subsequent post op period, she had sepsis with MODS. The diagnosis of amniotic fluid embolism was made after other differential diagnosis had been ruled out. The successful outcome in our case is attributable to early recognition with immediate delivery of the fetus, high-grade resuscitation, timely hysterectomy and aggressive treatment of coagulopathy by blood and blood-products, involvement of multidisciplinary team, constant supervision by nursing staff with positive approach. From a grim situation of near death, the final outcome was a successful story.

Keywords: Cardiovascular collapse, Amniotic fluid embolism, Disseminated intravascular coagulation, Sepsis with multiorgan dysfunction

INTRODUCTION

Amniotic fluid embolism (AFE) is an unforeseeable, life-threatening complication of pregnancy with an extremely high mortality rate. The pathogenesis of this condition is not clear. Probably amniotic fluid enters the maternal circulation as a result of a breech in the physiological barrier that exists between maternal and fetal compartment and activate an inflammatory cascade which leads to disseminated intravascular coagulation and multiorgan failure.[1] AFE is a rare obstetrical event with highest fatality rate. The mortality rate is 60% by Gilbert et al, 90% by Weiwen et al.[2,3] A confidential enquiry in UK to find out maternal death shows increased number of deaths attributable to AFE.[4] However the mortality rate was less in the largest study from a Canadian database, but survivors have profound neurological impairment. The mortality rate associated with sepsis and multiorgan failure is 40-60%.[5,6] We encountered a case during COVID pandemic who suffered from the deadliest combination of obstetrical nightmare of amniotic fluid embolism, DIC and sepsis and yet recovered completely with neurologically intact.

CASE REPORT

A 27 years old G2P1L1 lady at 38 weeks and 05 days period of gestation with previous LSCS status was planned for elective LSCS with bilateral tubectomy on 14 May. She underwent RTPCR prior to surgery as per institutional policy which was negative. She started labour pains on the day of surgery; however, the intensity of pain was mild. In the pre-operative room, patient had rupture of membranes associated with increased intensity of labour pains with severe back ache and cough with audible wheeze. The baseline recordings showed heart rate of 100-110 beats/min, blood pressure of 100/60 mm of Hg and SpO2 of 95-96% on O2 at the rate of 5L/min. Doppler shows

fetal bradycardia (FHR- 80-100 / min). Patient was immediately shifted to the OT table and spinal anaesthesia administered. Immediately cleaning and draping done and an asphyxiated male baby was delivered at 1058 hrs. Intra op patient developed tachycardia (PR>150/min), tachypnoea (RR>38/min), hypotension (SBP <50mmHg), desaturation (SPO2 <70%) and hematuria. There were bilateral rhonchi suggestive of severe bronchospasm. The patient was immediately managed by the anaesthesia team with IV fluids, vasopressors (Injection Noradrenaline at the rate of 0.175 mcg/kg/min), inotropes (Injcetion Dopamine at the rate of 5 mcg/kg/min), steroids (Injection Hydrocortisone 100 mg IV and repeated thrice), bronchodilator (MDI Salbutamol 10 doses) and Injection adrenaline 0.3 mg IM stat and the uterine and rectus sheath suturing was completed. While suturing the skin incision, abnormal oozing was noticed which was tackled by elecro-cautery. During vaginal toileting, persistent gush of fresh blood was noticed (500 ml approximately) which gradually intensified and there was oozing from the skin incision and all other puncture sites. A bedside clotting test was done, which showed increased clotting time. The NoK was counselled and a decision of hysterectomy was taken and consent was obtained. Since the SAB didn't wear off, no other anaesthesia was given for hysterectomy. Abdomen was reopened immediately and Subtotal hysterectomy was done and bilateral intraperitoneal drains were placed. Extensive oozing of blood was present from the uterine stump and skin incision. The bleeding from all puncture sites intensified. She was immediately resuscitated (01 whole blood, 02 packed RBCs and 4 FFPs). The blood pressure stabilised (124/78 mm of Hg) thereafter and Noradrenaline drip rate was reduced to 0.05 mcg/kg/min and Injection Dopamine was stopped. The blood loss during both the surgeries was approximately 3 L. Patient was shifted in the ICU.

Stay in ICU

Patient stayed in the ICU for 14 days. She was managed with massive transfusion protocol (A total of 42 units of blood and blood products were transfused). She was maintained on O2 inhalation, IV fluids, IV antibiotics and Noradrenaline infusion which were later discontinued. For hemodynamic monitoring a central line was placed in the right internal jugular vein. Close monitoring was done with regular ABG to identify acute respiratory distress syndrome (ARDS) and transfusion related acute lung injury (TRALI). She had urine output of 375ml post 06 hours of surgery and a Hb of 6.8 gm%, platelet of 67,000 with D Dimer >10000. The INR was unrecordable (Calibre of machine is between 1.0-60.0).

On post-operative day 1 to day 3, patient was hemodynamically better but tachycardia and desaturation persisted with adequate urinary output. The intraabdominal bleeding continued (Hemorrhagic collection 1600 ml in abdominal drains). Investigations reveals INR of 1.8, however she remained anemic (Hb 6.4 gm %), Thrombocytopenic (50,000/cmm), however LFT (total Bilirubin 6.0 mg% with raised liver enzymes) was deranged along with hypocalcaemia (6.5 gm %) and hypokalemia. She was given Injection KCl and calcium gluconate.

On post-operative day 3 to day 6 onwards patient had sepsis with MODS. She was managed empirically with upgradation of antibiotics (Injection Meropenum, Injection Clindamycin and later Injection Ticarcillin was added) as the culture and sensitivity report of blood and urine were negative, fluid restriction (2.5 L in 24 hours), lasix infusion at the rate of 2 mg IV/hr (stopped after 36 hourrs) and correction of dyselectrolytemia. Patient responded well and supportive treatment was continued further. The abdominal and subcutaneous drains and Foleys catheter were removed subsequently. On day 14 patient as shifter to general ward and discharged on post-operative day 18 after suture removal.

Table 1: Diagnostic criteria of AFE adapted from Clark SL et al.

S.no	Diagnostic criteria of AFE
1	Acute hypotension or cardiac arrest.
2	Acute hypoxia, defined as dyspnoea, cyanosis or respiratory arrest.
3	Coagulopathy, defined as laboratory evidence of intravascular consumption, fibrinolysis or haemorrhage in the absence of other explanations.
4	Onset of the above during labour, caesarean section, D&E or within 30-minute post-partum.
5	Absence of any other significant confounding condition or potential explanation for the signs and symptoms observed.

DISCUSSION

The patient had sudden onset of increased intensity of labour pains associated with rupture of membranes, backache, and cough with audible wheeze. It triggered cardiovascular collapse immediately as evident by tachycardia, tachypnoea, hypotension, reduced oxygen saturation level and fetal bradycardia. Intra operative she had DIC and was hemodynamically unstable. Surgery was able to complete with inotropes and vasopressors. In subsequent post operative period she had sepsis with MODS. In clinical obstetrics there is a handful of conditions which can trigger sudden cardiovascular collapse at this juncture. The various differential diagnoses were peripartum cardiomyopathy, myocardial infarction, deep vein thrombosis with pulmonary embolism, amniotic fluid embolism.

Deep vein thrombosis is abrupt in onset and there is pain and oedema of the lower limbs and thigh with positive Homan's sign. These symptoms were not present in the patient, however the symptoms may mask during pregnancy. The signs and symptoms of Pulmonary embolism include tachypnoea, tachycardia, dyspnea,

pleuritic chest pain, cough and low grade fever and actually patient was having most of them. However it was ruled out by compression ultrasonography of the veins of lower limbs as no venous flow obstruction was present.[7,8] Peripartum cardiomyopathy was ruled out as no symptoms and signs of heart disease were present in the late third trimester.[9,10] Myocardial infarction during pregnancy is a remote possibility and ruled out as symptoms and signs were not present and no literature is suggestive of giving rise of massive DIC in this condition.[11] Our patient had sudden onset cardiovascular collapse and the clinical condition points towards the possibility of amniotic fluid embolism.

As the literature says that the diagnosis of AFE is essentially clinical and of exclusion. As AFE presentation can be variable and non-specific, a high index of suspicion is required for its diagnosis. The pathophysiology of AFE is poorly understood. It is suggested that the response of the body to this condition is biphasic. In the first phase the vasoactive substances in the amniotic fluid enters the maternal circulation by a breech in the physiological barrier and causes pulmonary vasospasm and hypoxia leading to cardiovascular collapse. In the second phase there DIC with heart failure and acute respiratory distress syndrome. The first phase lasts for about half an hour and responsible for fatality in most of the cases.[12-14]

The onset of DIC was rapid and it was evident by the time the skin incision was being closed and vaginal toileting was being done. It was confirmed clinically while performing hysterectomy when there were multiple bleeding points which were difficult to control. The INR was also unrecordable for 10-12 hours in post op period. Later on, the D-dimer was more than 10,000 and FDP was more than 20 ng/dl. The management of DIC is supportive. Timely hysterectomy and transfusion of blood and blood products were other turning events. Immediate administration of blood had prevented further hypoxia and restored circulation and Hysterectomy prevented further loss of platelets and coagulation factors. Continuous transfusion of blood and components had provided clotting factors and as a result the INR was 1.8 from unrecordable range. Clark et al proposed model to diagnose AFE comprising five diagnostic criteria (Table 1), has been widely adopted since 1995, and our patient's presentation met every diagnostic criteria.

Patient had sepsis with MODS in post op day 3 as manifested by bradycardia, raised TLC with predominantly neutrophils, positive CRP, raised serum procalcitonin, left to right shift in PBS with presence of toxic granule, elevated serum urea and creatinine levels with decreased urine output, deranged LFT, bilateral pleural effusion on chest x-ray, however the culture report of blood and urine were negative. The patient was treated conservatively by fluid restriction, intermittent lasix injection, upgradation of antibiotics, correction of Dyselectrolytemia and other supportive therapy. Patient had responded to the conservative management and her renal parameters improved and sepsis was controlled in subsequent days.[5,6]

The successful outcome in our case is attributable to early recognition with immediate delivery of the fetus, high-grade resuscitation, timely hysterectomy and aggressive treatment of coagulopathy by blood and blood-products, involvement of multidisciplinary team, constant supervision by nursing staff with positive approach. From a grim situation of near death, the final outcome was a successful story.

CONCLUSION

Amniotic fluid embolism is a life-threatening condition. There is no diagnostic tool available and its diagnosis is of exclusion. A collaborative multidisciplinary approach is mandatory for effective management and favorable outcome.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Knight M, Tuffnell D, Brocklehurst P. Incidence and risk factors for amniotic fluid embolism. Obstet Gynecol. 2010;115(5):910.
2. Gilbert WM, Danielsen B. Amniotic fluid embolism: decreased mortality in a population based study, Obstet Gynecol. 199;93:973.
3. Weiwen Y. Study of the diagnosis and management of the amniotic fluid embolism: 38 cases of analysis. Obstet Gynecol. 2000;95:385.
4. Saving mother's lives 2003-2005 Report: A Report of UK confidential enquiries into maternal deaths. London: RCOG Press; 2007. Confidential Enquiry into Maternal and Child Health (CEMACH).
5. Angus DC, Vander Poll T. Severe sepsis and septic shock. N Eng J Med. 2013;369:840.
6. Munford RS. Severe sepsis and septic shock: Introduction. In Longo DL, Fauci AS, Kasper DL. Harrison principle of Internal medicine. 18 edition. 2012;22-23.
7. Antiphospholipid syndrome. American college of Obstetrician and Gynecologists. Practice Bulletin No,132. 2012
8. Guyatt GH, Akl EA, Crowther M. Executive summary: Antithrombotic therapy and prevention of thrombosis. In: American college of chest physicians evidence based clinical practice guidelines, chest. 9th edition. 2012;141:7S.
9. Deans Charlotte L, Uebing A, Steer J. Cardiac disease in pregnancy: John stud. Progress in Obstet gynaecol. 2006;17:164-82
10. Thaman R, Varnava A, Hamid MS. Pregnancy related complications in women with hypertrophic cardiomyopathy. Heart. 2003;89:752.

11. American college of Obstetrician and Gynecologists. Critical care in pregnancy. Practice bulletin No 100. 2009;2011a13.
12. Clark SL, Montz FJ, Phelan JP. Hemodynamic alterations associated with amniotic fluid embolism: A reappraisal. Am J Obstet Gynecol. 1985;151:617-21.
13. Clark SL. Amniotic fluid embolism. Crit Care Clin. 1991;7:877-82.
14. Howes LJ. Anaphylactoid reaction possibly caused by amniotic fluid embolism. Intl J Obstet Anesth. 1995;4:51-4.

Cite this article as: Vishal AK, Bhasin D, Prasad K. A case of amniotic fluid embolism and its sequelae during COVID-19 pandemic: a success story. Int J Reprod Contracept Obstet Gynecol 2021;10:1188-91.