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CASE REPORT

Successful Outcome of Amniotic Fluid Embolism Complicated with Severe Postpartum Hemorrhage and Neurological Deficit

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ABSTRACT

An amniotic fluid embolism (AFE) is one of the rare obstetric emergency with a high maternal mortality rate. This condition is clinically characterized by three distinct phases: during the first phase, sudden onset of respiratory distress and cyanosis occur within seconds followed by hypotension, shock, and loss of consciousness within minutes. Of those who survive the initial insult, 40 to 50% enter the second phase characterized by coagulopathy and hemorrhage within hours. During the third phase, acute symptoms are over, and tissue injury of the brain, lung or renal is for the most part already established. The patient may succumb due to multisystem organ failure or infection acquired in the hospital.

We report an interesting case of a middle-aged pregnant mother who developed AFE during the caesarean section and complicated with severe postpartum hemorrhage, which was successfully aborted by intramyometrial prostaglandin F2 alpha injection, thus obviating the need for hysterectomy. Following that she also suffered from expressive dysphasia and have an uneventful recovery through the speech therapy. We concluded that early diagnosis and timely intervention that may be the best way to achieve the favorable outcome of amniotic fluid embolism and intramyometrial prostaglandin is the available simple drug for preventing a peripartum hysterectomy during amniotic fluid embolism.

Keywords: Amniotic fluid embolism (AFE), Coaguolopathy, Disseminated intravascular coagulation (DIVC), Intramyometrial prostaglandin, Obstetrics emergency, Postpartum hemorrhage.

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INTRODUCTION

Amniotic fluid embolism (AFE) is a rare and often fatal condition characterized by sudden cardiovascular collapse, altered mental status, and Disseminated intravascular coagulation (DIVC). The AFE was first described by Meyer in 1926.[1] It becomes a clinical entity in 1941 after Steiner and Luschbaush published a maternal mortality cases series that include eight women who had squamous cell and mucin, presumably of fetal origin within their pulmonary vasculature at post- mortem.[2] The AFE is one of the direct leading cause of maternal mortality in developed country as well as in Malaysia and majority were due to obstetric embolism which include amniotic fluid and pulmonary embolism.[3]

It is a condition of pathogenic mystery, diagnostic difficulty and therapeutic challenges.[4] It is a rare but often lethal complication of pregnancy and childbirth. Its presentation is variable, ranging from respiratory depression, cardiac arrest, sudden death through to varying degree of organ dysfunction with or without coagulopathy. The first well-documented case with ultimate survival was published in 1976, although a handful of surviving patients with presumptive diagnoses of AFE has been reported. [5]

CASE REPORT

We report a non-fatal case of AFE occur in a low-risk primigravida during a cesarean section complicated with coagulopathy and neurological deficit.

A healthy 36-years-old Malay, primigravida, teacher who presented at 38 weeks gestation for footling breech in labor. On admission, her vital signs were stable and vaginal examination showed cervical opening was 5 cm dilated with intact membrane and foot felt. Urgent ultrasound was done and showed grossly normal male fetus with normal liquor volume and placenta at the upper segment. Her vital signs were stable and term size uterus with singleton breech presentation. A decision was there-

fore made to perform a section under regional anesthesia for primigravida with the footling breech in labor.

Her antenatal follow-up visits were uneventful and no underlying medical and surgical problems before. Intraoperatively, clear liquor was noted, and baby boy 3.9 kg was delivered with breech extraction. The baby's apgar score was 9 at one minute and attended by a pediatrician immediately. A few minutes after delivery of the baby, patient complaints of sudden onset of difficulty in breathing and became cyanosed. Subsequently, she was unconscious, and oxygen saturation was dropped from 98 to 68%. There was hemodynamically instability with the heart rate of 40 beats per minute and blood pressure was unrecordable. She was given intravenous atropine and intubated immediately. Cardiopulmonary resuscitation was commenced. She developed ventricular tachycardia and defibrillation was started with 50 J, 100 J, 150 J until achieving sinus tachycardia. At this stage, blood pressure was 90/50 mmHg, and carotid pulse was felt. She looked pale and profuse bleeding from the surgical wound and raw area of the placenta bed was noted. Manual massage was done and intravenous oxytocin 10 units bolus which is followed by 40 units oxytocin in 500 mL normal saline infusion was given. She was transfused one unit of whole blood; four units of packed cells, four units FFP and six units cryoprecipitate. Uterus became contracted and closed in two layers with estimated blood loss about 1000 mL at that time. The hypotension responded to fluid administration and a blood transfusion but excessive oozing from the vagina continued and almost 2000 mL loss within a few minutes. The uterine massage was continued and we decided to give intramyometrial carboprost, a prostaglandin F2 alpha 1 mg to arrest the bleeding. Bleeding was stopped within a few minutes after given intramural prostaglandin F2 alpha as it is an uterotonic agent that helped for uterine contraction and arrest bleeding. Subsequently she was transferred to ICU for cerebral resuscitation and continue further supportive treatment. She was received massive blood transfusion including blood components, and total blood loss was 4 liters. In ICU, she recovered well and extubated on day six postpartum.

Postoperatively, she was noted to have expressive dysphagia but responded to command. She had computed tomography brain scan (CT brain scan) twice, two weeks apart and showed no infarct and no residual damage. She continued with physiotherapy and speech therapy. There was no residual neurological damage, and she was discharged well with the baby on day 16 postpartum. Subsequently, she continued physiotherapy until full recovery at 6 to 8 weeks postnatal follow-up.

Table 1 showed the result of full blood count, coagulation profile and renal function tests postoperative day one to postoperative day six with improving biochemical parameters.

DISCUSSION

We reported a survival case of an amniotic fluid embolism at the cesarean section for 38-year-old primigravida, footling breech presentation in labor. AFE is a rapidly lethal condition. Multiple and simultaneous interventions are needed for acutely ill patients regardless of the cause of illness.[5] The AFE is a rare event, ranging from 1 in 8,000 to 1 in 80,000 deliveries. [3]

The variance in the incidence is explained by dissimilar case definitions and possibly improvements in intensive care management of affected patients. In Malaysia, AFE is one of the common direct cause of maternal mortality.[6] There are no consistent, identifiable predisposing factors for AFE apart from the timing of the onset. As a consequence, no single institution has sufficient experience to access risk factors, determine the pathophysiology and clinical course or evaluate management strategies.

The classical description is sudden onset of dyspnoea, cyanosis, and hypotension out of proportion to the blood loss, followed quickly by cardiorespiratory arrest. This initial episode present with sudden onset of dyspnea, cyanosis, hypotension which usually followed in survivors by DIVC.[7]

Unanswered questions remain about this condition where the microscopic fetal debris particles or microem-

Table 1: Blood investigation results from POD 1 to POD 6 (ICU)

	POD 1	POD 2	POD 3	POD 4	POD 5	POD 6
HB	6.2 gm/dL	7.8	8.1	8.8	9.0	12.2
platelet	92	132	102	103	129	274
INR	1.55	1.23	1.20	1.01	0.93	1.0
APTT	55.5	33.0	30.3	30.6	30.1	30.2
Urea	4.1 mmol/L	3.2	5.3	6.4	5.2	3.2
Creat	61 micromol/L	67	82	47	60	59

POD = postoperative day ICU = Intensive care unit

bolization can cause acute occlusion of a large part of the mother's pulmonary microvasculature. It was characterized by respiratory distress and cyanosis, which was a manifestation of acute cor pulmonale. Furthermore, the initial reaction of the pulmonary vasculature to amniotic fluid exposure may be transient vasospasm, pulmonary hypertension, and profound hypoxia. However, there is no direct evidence has been documented till today.[4] It is currently believed that the presence of fetal debris in the maternal circulation is merely evidence of fluid passage within the maternal circulation. The contribution, if any, of these fetal particles to the pathophysiology of AFE may be through the release of arachidonic acid metabolites at lung level, induced by the capillary damage of micro-emboli.[4] Alternatively the symptoms are fairly vague and non-specific, that make the diagnosis presumptive. In particular, hemorrhage and fetal distress have been neglected as presenting symptoms that sometimes precedes other manifestations. High index of suspicion for AFE should be considered whenever encountered with any pregnant patient who has a sudden onset of respiratory distress, cardiac collapse, seizures, unexplained fetal distress, and abnormal bleeding.[8] Anaphylaxis may play a role in the syndrome as it is conceivable that the toxins responsible for the anaphylactoid reaction of AFE it has, and no single mediator pl ays a more significant role than another with uncertainty.

In our case report, two distinctive features were noted which are sudden onset of dyspnea, cyanosis and intensity of the coagulopathy disorder with postpartum hemorrhage. At present, diagnosis is made clinically on the basis of sudden onset of signs and symptoms after excluding the possible differential diagnosis. There is no diagnostic test to confirm AFE; however, there are few supportive tests to diagnose as AFE after excluding other possibilities. There is no specific therapy and only supportive and managed symptomatically. Maintenance of oxygenation, circulatory support, and correction of disseminated intravascular coagulation are the main stay of treatment. We have now treated our case of massive postpartum hemorrhage following an AFE with intramyometrial prostaglandin. In our case, we had decided to use intramyometrial prostaglandin before performing a cesarean hysterectomy for massive severe postpartum hemorrhage due to uterine atony intraoperatively though there was a dilemma in which concerning drug side effects in relation to suspicion of AFE. However, it was successful in controlling the hemorrhage as well as preventing inevitable, intrapartum cesarean hysterectomy. Thus, we concluded that intramyometrial prostaglandin could be administered as a life and uterus-saving therapy in women with life-threatening primary postpartum hemorrhage. This is consistent with the literature review by Bruse.[9] The authors consider the transvaginal intramyometrial application of PGF2 alpha to be an effective method to circumvent an emergency hysterectomy. However, this should occur sufficiently early and in a dose of not less than 1 mg.

We believe that in situations of intractable PPH, and where a hysterectomy is otherwise not indicated, administration of intramyometrial prostaglandin ought to be contemplated before performing a hysterectomy. Therefore, we would like to suggest that intramyometrial prostaglandin can be used as adjunct for life-threatening massive hemorrhage that has failed to respond to conventional intervention before deciding for definite surgery. It should be a caution to give the patient with a known case of bronchial asthma, hypertension, and cardiac disease because of the risk of severe hypertension and cardiac arrest inadvertently injection into the vessels.[10] However, it is safe to administed at those healthy young woman without underlying medical problem as there is no adverse reaction has been reported before. We also recommend intramyometrial administration of haemabate as soon as possible in few special situations like when no blood is available; sudden onset of massive hemorrhage and before considering surgical procedures like peripartum hysterectomy or laparotomy

CONCLUSION

We present the case of 36 years, primigravida with coagulopathy and hypotension during emergency cesarean section. The presentation and clinical course were consistent with a diagnosis of amniotic fluid embolism. A high index of clinical suspicion is necessary to make an early diagnosis to prevent many maternal deaths and improve the outcome of AFE in the future. With supportive care, she recovered and returned to normal life. Early premortem diagnosis, timely and appropriate intervention with supportive treatment may improve the outcome of AFE and reduce the current high maternal mortality rate. This patient with severe postpartum hemorrhage which is mainly due to uterine atony is treated successfully with intramyometrial injection of 1 mg of prostaglandin F2 alpha with excellent results. This was an effective, safe, and rapid therapy in these cases of severe postpartum bleeding.

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AUTHORS CONTRIBUTIONS

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