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CASE REPORT

Use of Extracorporeal Life Support in Postpartum in a Patient with Amniotic Fluid Embolism Complicated by Left Ventricular Failure and Disseminated Intravascular Coagulopathy

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Background

Amniotic fluid embolism is a rare but catastrophic complication of pregnancy. We describe our institutional experience with extracorporeal membrane oxygenation (ECMO) use in a pregnant women who presented with amniotic fluid embolism and was complicated by disseminated intravascular coagulopathy and postpartum haemorrhage (PPH) who subsequently developed left ventricular failure requiring ECMO therapy.

In this report we describe the effective value of extracorporeal membrane oxygenation in the successful treatment of a patient with amniotic fluid embolism with uterine conserving management of Bakri balloon treatment of PPH secondary to disseminated intravascular coagulopathy (DIC).

Case Report

We report a 32-year-old Chinese lady, Gravida 1 Para 0, who was booked at 6 weeks and 5 days. She had open appendicectomy previously. She did not have any significant past medical history of note or drug allergy. Non-invasive prenatal screening (Harmony test) at 10 weeks and 5 days was low risk and her screening ultrasound at 22 weeks and 5 days showed a right pelvic kidney. The rest of the antenatal follow-up was uneventful.

She was admitted at 39 weeks and 3 days for prostin induction for social request. Oxytocin infusion was started 12 hours later when cervical os was 2 cm and partially effaced. She had spontaneous rupture of membrane at the 13th hour post prostin and her first stage of labour lasted 6 hours. Her second stage of labour lasted 54 minutes and she delivered a baby boy via vacuum assisted delivery for maternal exhaustion. Birth weight was 3140 g. Apgar was 9 at 1 minute and 10 at 5 minute. She had a second degree perineal tear which was repaired with Vicryl Rapid.

She developed postpartum haemorrhage 45 minutes post-delivery. On examination, there was a persistent ooze of dark red watery blood that seemed poorly oxygenated from her vagina but her uterus was contracted at all times. Estimated blood loss was 500 ml. She was clinically hypotensive and coagulopathic with bleeding from her nasal and oral cavities and her saturation decelerated to 69%. Patient was brought back to the operating theatre for examination under anaesthesia. Aggressive management by the multidisciplinary team including anaesthetist, cardiologist and obstetrician ensured that she was resuscitated with fluids, 6 pints packed cell transfusion, 2 litres fresh frozen plasma, 8 packs of platelet transfusion as well as 6 vials of Recombinant Coagulation Factor VIIa (Novo 7). Uterotonics such as intravenous (IV) tranexamic

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acid 1 g, intramuscular (IM) carboprost 250 mcg, and IV duratocin 100 mcg were given. IV syntocinon drip (30 units in 1 pint of dextrose saline) was started. Bakri balloon was inserted to tamponade the uterine-placental surface bleed and to monitor blood loss, with 1.5 litres of blood loss from Bakri drain after 4 hours of resuscitation.

Her coagulation profile was severely deranged with a PT of 15 seconds and APTT 84 seconds, platelets of 79 × 109/l and haemoglobin of 9.5 g/dl. Emergency transthoracic echo (TTE) on the operating table showed normal right ventricular function and severely impaired left ventricular function with ejection fraction of 10-15% consistent with a stress cardiomyopathy pattern. Intravenous adrenaline at 0.5 mcg/kg/min could only achieve a mean blood pressure of 50 mmHg and a heart rate of 140-150 beats per minute.

In view of failure to maintain hemodynamic status with IV adrenaline and dopamine and the appearance of heart failure, the decision was made to support her with extracorporeal membrane oxygenation (ECMO). 17 F arterial cannula and 21F venous cannula was inserted via left central femoral artery and vein respectively. Left superficial femoral artery reperfusion 5F cannula was inserted under ultrasound guidance. IV adrenaline was then weaned off. She was put on anti-heart failure medications. Prevention of arrhythmia with attention to electrolytes' balance was made.

She underwent CT thorax, abdomen and pelvis which reviewed no pulmonary embolus or other etiology to account for postpartum collapse. No heparin administration was started in view of highly deranged coagulopathy. Her coagulopathy resolved over the following 3 days and she was started on IV heparin on day 4 of ICU/ECMO and was converted to fraxiparine on day 7. Serial TTEs revealed gradual recovery of heart function and ECMO was successfully weaned off on day 6 of ICU stay with Bakri balloon removed later in the afternoon of the same day. ECMO was complicated by deep vein thrombosis on her left upper femoral superficial vein which resolved on day 13 of admission after anti-coagulation therapy.

She was monitored in intensive care unit (ICU). She developed fever on day 7 of ICU stay but blood and urine culture were negative and she was covered with prophylactic antibiotics, subsequently fever resolved. Chest X-ray on day 8 of ICU stay showed pulmonary edema, which was treated aggressively with diuretics and hence she was extubated to the 9th ICU day. Patient woke up without neurological deficits. She was started on after load reduction in view of her persistent high blood pressure. Nasogastric tube was taken off on day 12 of ICU stay before being transferred to general ward. Left ventricular ejection fraction was 50% on pre-discharge TTE. Her brain natriuretic peptide (ProBNP) levels decreased from 14000 s to 1400 s. She underwent physical rehabilitation and titration of medication since her extubation and was eventually discharged well on post-delivery day 15.

Both she and her baby were well at 6 weeks postnatal. She did not require further cardiac medications. Her Pro BNP returned to normal level.

Discussion

AFE is a rare, unpredictable and life-threatening complication in obstetrics. CAMECH [1] reports it as the 4th direct cause of death, 0.57/100000 between year 1985 to 2008 and Knight, et al. [2] reports an incidence rate of 2.0 per 100000 maternities in the UK. It is a condition associated with poor prognosis, a leading cause of maternal mortality of approximately 85%, with half of the deaths occurring within the first hour after diagnosis and an additional 25% to 50% of mortality occurring during the ensuing 4 to 5 hours [3] and more so with the ethnic minority [2].

According to Clark, et al. [4], amniotic fluid embolism may be the result of anaphylactic reactions to fetal antigens. Mechanical, humoral and immunological factors are implicated. In addition to insoluble fetal components, amniotic fluid also contains numerous vasoactive substances (bradykinin, histamine, and others) and procoagulant substances that can lead to endothelial activation and a massive inflammatory reaction. Thus it is commonly called an anaphylactoid syndrome of pregnancy [5].

When amniotic fluid or fetal cells enter the maternal circulation, it will produce a biphasic hemodynamic response. In the first stage, the spasm of pulmonary vessels results in pulmonary hypertension, bronchoconstriction, and severe hypoxia. The second stage involves left ventricular failure resulting from myocardial ischemia, resulting in pulmonary edema, hypotension and shock. After which, uterine atony and DIC sets in. Consumptive coagulopathy is often the earliest sign of amniotic fluid embolism. The hemodynamic decompensation may be transient and recoverable within a few hours [6-8].

A definitive diagnosis is usually made by demonstration of amniotic fluid material in the maternal circulation and in the pulmonary vessels by identification of lanugo or fetal hair and fetal squames in an aspirate of blood from the right heart but this is hardly used in clinical practice [8].

Diagnosis of AFE is often based on clinical symptoms after other causes/diagnoses have been excluded as in our case. There is no universal criteria for AFE but the criteria most frequently cited in the current literature are those of the UKOSS (UK Obstetric Surveillance System) and those of Benson [5]. McDonnell noted that the risk factors associated with AFE are advanced maternal age, multiple pregnancy, caesarean birth,

instrumental delivery, placenta praevia, placental abruption, eclampsia, fetal distress, polyhydramnios, uterine rupture and ethnic minority [9].

AFE has a wide spectrum of presentation, it could present as mild PPH, coagulopathy and sometimes undiagnosed; to very severe cases presenting as cardiorespiratory arrest [2] Clinical symptoms of AFE are disseminated intravascular coagulation (DIC) was commonly seen [1] along with hypotension, hypoxia, pulmonary edema and cardiopulmonary arrest. Other features include that of altered mental state, dyspnea, non-reassuring fetal status, seizures and vomiting [1,4].

Fetal outcome is reported to be poor if the onset of AFE is before delivery. Clarke, et al. reported 79% of fetuses survived but only 50% of these were neurologically normal. In the UK registry 87% of the fetuses survived, with 29% of survivors developing hypoxic ischemic encephalopathy [8].

In the treatment of AFE, ECMO can be considered [10]. It is possible that the widespread use of ECMO during the 2009 H1N1 pandemic, and the publication of the CESAR trial have contributed to its worldwide growth [11]. Cara, et al. [9] reported that eighteen patients were treated with ECMO during the study period from January 2009 to June 2015 of which 2 were for AFE. Cardiopulmonary bypass plays a major role by obtaining a few critical hours to stabilize the cardiovascular status while providing supportive therapy in terms of correcting coagulopathy, plasma exchange or exchange transfusion can also be considered [2,6]. There is no consensus on the optimal range of clotting time for maintaining anticoagulation in pregnant or post-partum patients [12,13]. Early institution of ECMO is important as it will prevent secondary organ failures e.g. liver, kidney, brain which will adversely affect prognosis [6]. In our case, venous-arterial (VA) support is required for circulatory support as she had poor ejection fraction of 10-15% with high doses of adrenaline. Venous-venous (VV) ECMO only provides gaseous exchange support and no circulatory support. While there was cardiovascular support with VA ECMO, there was also active heart failure management that was geared towards fluid management, inhibition of the renal-angiotensin-aldosterone system (RAAS), anti-inflammatory markers and agents that promote coronary microvascular flow. All these were done with various pharmacological agents, trans-thoracic echo surveillance, and close monitoring of renal, hepatic and hematologic parameters.

Nirmal [14] performed a literature review and reported that the indications for ECMO in pregnancy use includes severe acute respiratory distress syndrome (ARDS), cardiac arrest, postpartum cardiogenic shock, status asthmaticus and amniotic fluid embolism. There is little evidence to indicate the optimal duration of ECMO support and the best time for weaning. Early application of ECMO can play a role in cardiovascular support in patients with AFE and prevent end organ damage like hypoxia induced neurological damage [4,6].

Chang, et al. [6] reported that complications such as hemolysis, renal function impairment, intracranial hemorrhage, sepsis, and limb ischemia are directly related to the duration of extracorporeal life support (ECLS). To avoid these complications, patients should be weaned from ECMO as soon as possible. Moore [15] suggested that other ECLS-related complications e.g. cannula dislodgment, nosocomial infections, including bloodstream, respiratory, urinary tract, and line-related infections.

In our case, NT ProBNP remains elevated despite echocardiogram showing improvement of left ventricular ejection fraction at 50%. Brain natriuretic peptide levels are approximately twice as high in pregnant versus non-pregnant women, but do not vary significantly during a given normal pregnancy or postpartum [16]. Tanous, et al. [17] explained that women with ventricular dysfunction correlates with high BNP during pregnancy. We postulate that her elevated BNP is attributed to renal impairment and persistently mild left ventricular impairement [18]. Long term prognosis for mortality, HF, and new ischemic events is described to be better stratified with a combination of NT ProBNP with LVEF [18,19].

Prompt recognition of PPH secondary to AFE, use of Bakri balloon to conserve uterus and early activation of ECMO for support and myocardial recovery while treating underlying AFE and its complications is important. Bakri balloon was inserted as massive blood loss and hypotension can result in uterine atony. Massive transfusion and PPH protocol was activated but the patient continues to be unresponsive to high dose inotropes, therefore ECMO was activated. Continuous 'flogging' of the heart after 'anaphylactoid syndrome' from AFE is usually futile. The heart needs time to recover and early commencement of ECMO can prevent on-table maternal mortality and allow good recovery if maternal downtime is minimized.

Notably, PPH is not a contraindication to ECMO but special precautions has to be given to postpartum patients with AFE complicated by coagulopathy and bleeding who require ECMO as it could complicate titration of anticoagulation parameters and transfusion protocols and worsen the postpartum haemorrhage [3,6,7,20]. There are only a few case reports on peripartum patients going on ECMO with PPH in current literature [3,12].

In summary, key factors in the management of AFE in pregnancy in this case are early recognition, prompt resuscitation and supportive therapy, delivery of the fetus and early input of an experienced multidisciplinary team of consultants (anesthesiologist, obstetricians, cardiothoracic and vascular surgeons, cardiologists

and intensivists) to reduce mortality as patients can deteriorate rapidly.

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