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CASE REPORT OBSTETRIC ANESTHESIA

Fulminant peripartum pulmonary embolism leads to maternal cardiac arrest and hypoxic-ischemic encephalopathy

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ABSTRACT

Pulmonary embolism (PE) risk is greater in women during pregnancy or puerperium. If indeed the peripartum pulmonary embolism is not recognized early and treated appropriately, it could have catastrophic consequences. We therefore present a case of a pregnant woman who unfortunately went from undiagnosed peripartum pulmonary embolism and had a crisis during delivery. She eventually had cardiac arrest and hypoxic-ischemic encephalopathy as complications. We emphasize early recognition and prompt treatment of pulmonary embolism in order to prevent such serious complications in pregnant women. We discuss about the clinical condition of our patient and make contrasts based on previous literature.

Abbreviations: CPR - Cardiopulmonary Resuscitation; GCS - Glasgow Coma Scale; GDM - gestational diabetes mellitus; IVI - intravenous infusion PE - Pulmonary embolism; ROSC - return of spontaneous circulation;

Keywords: Pulmonary embolism; pregnancy, cardiac arrest; hypoxic-ischemic encephalopathy

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1. INTRODUCTION

When compared to non-pregnant women, pregnant women have a 4-5 times higher risk of having pulmonary embolism with an estimated incidence of 1-2 per 1000 pregnancies.[1] The incidence of peripartum pulmonary embolism (PE) was found to be 0.3% (0.2–0.4 %) in a systematic review and meta-analysis, which was consistent with the Nationwide Inpatient Sample (NIS) database for the United States and Germany.[2, 3, 4] With an overall in-hospital fatality rate of 3.4%, rates of in-hospital mortality were about 200 times higher among those with acute PE per 1000 pregnancies.[3,4] Hypercoagulability, venous stasis, and vascular injury, which make up Virchow's triad, all occur during pregnancy.[5] Venous stasis results from a decrease in venous return caused by the pressure of the gravid uterus on the iliac veins and vena cava, which leads to an increase in coagulation factors and a decrease in coagulation inhibitors during pregnancy, which represents the physiological preparation to protect women from the bleeding issue during miscarriage or childbirth.[5]

We present a case of a pregnant woman who went from undiagnosed peripartum pulmonary embolism and had a crisis during delivery.

2. CASE REPORT

We report the case of a 43 years old Malay lady, gravida five para four with underlying of gestational diabetes mellitus (GDM). Her body mass index (BMI) was 34 kg/m^2 with body weight 85 kg and height of 158 cm. She was admitted electively to obstetric ward in our hospital for induction of labor in view of GDM on metformin at 38 week of gestation period. She was induced with foleys catheter and tab prostaglandin E2 twice and was sent to labor suite with cervical os at 6 cm. Amniotomy was done which yielded clear liquor; 20 min after amniotomy done, she suddenly complaint shortness of breath, developed fits episode and became unresponsive.

All obstetric, anesthesia and pediatric team attended to her immediately. Intravenous (IV) magnesium sulphate 4 mg was given to cease the possible eclamptic fit, but failed. Thus, we proceeded to intubation as her Glasgow Coma Scale (GCS) dropped to 3. After 5 min of intubation, she went to asystole and first cardiopulmonary resuscitation (CPR) was commenced. Obstetric team decided to do perimortem cesarean section and the baby was successfully delivered after 3 min of CPR. Adrenaline was given twice IV. However, her uterus was atonic, despite having been given four times of intramuscular (IM) carboprost, intramyometrial carboprost, and syntocinon 5 units IV as a slow bolus.

She returned to spontaneous circulation (ROSC) after 15 min of CPR and was started on intravenous infusion (IVI) of noradrenaline and adrenaline. However, she developed asystole again after 3 min and second cycle of CPR was started. Third adrenaline was given and she regained spontaneous circulation again after 2 cycles of CPR; however, her uterus was still in intermittent atonic state. Massive transfusion protocol (MTP) was initiated with 2 pints of safe O was started. After 4 min of ROSC, she went to asystole once more and third CPR was commenced with fourth adrenaline was given together with maximum inotropic support. She regained spontaneous circulation after 3 cycles of CPR and vasopressin infusion was started.

Our initial impression was amniotic fluid embolism and obstetric team gave the patient IV methylprednisolone 1 G and started tranexamic acid infusion @ 1 G/h. Disseminated intravascular coagulation (DIC) was expected as she had an increase in vaginal bleeding, bleeding from branula site and blood-stained oral secretion. Therefore, we proceeded with bedside b-lynch compression suture, continuous warm packing and manual compression of uterus. At that time her estimated blood loss was 6 L and she had been transfused 2 pints safe O, 2 pints packed cell (PC), 4 units fresh frozen plasma (FFP), 6 units cryoprecipitate and 1 unit platelet apheresis.

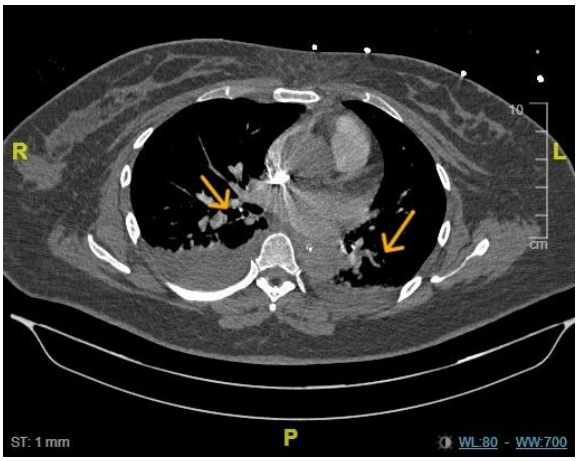

Figure 1: Computed Tomography Pulmonary Angiogram (CTPA).

Filling defects in the posterior segmental branch of both lower lobe pulmonary artery in keeping with pulmonary embolism. Bilateral pleural effusion with adjacent consolidations.

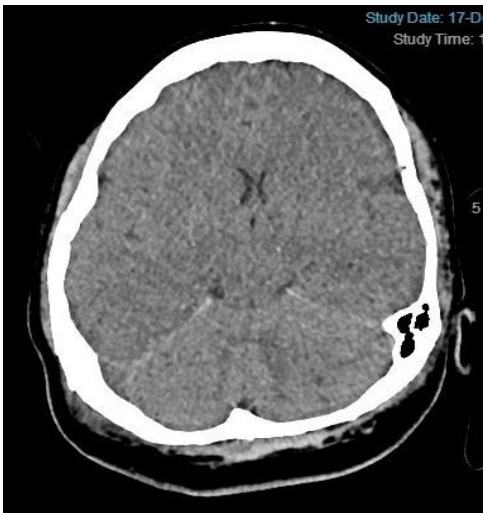

Figure 2: Computed Tomography (CT) Brain

Generalized cerebral sulcal and CSF spaces effacement with narrowing of lateral and third ventricles, representing diffuse cerebral edema. Loss of normal grey white matter differentiation. Basal cisterns are effaced.

At this stage she was rushed to the operating room (OR) for emergency hysterectomy with bilateral internal iliac ligation. During surgery, she was still in the state of DIC and was transfused with 4 pints of PC, 8 units of FFP, 12 units of cryoprecipitate and 2 units of platelets. Obstetric team had to apply Surgicell®, hemostatic powder and two small abdominal packs as patient did not achieve hemostasis even though after completion of hysterectomy and bilateral internal iliac ligation. Total blood loss in OR was 3 L. At that time, our diagnosis was

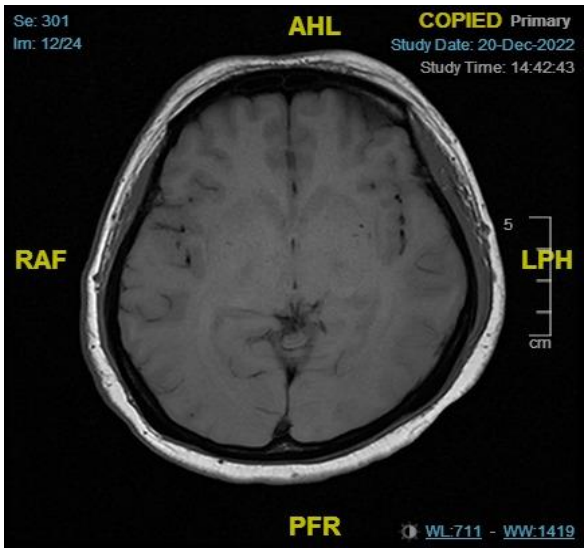

Figure 3: Magnetic resonance imaging (MRI) Brain
Diffuse swollen gyri and effacement of the bilateral cerebral sulci. Mild effacement of all ventricles. Basal cisterns are still preserved

'*massive PPH secondary to DIC secondary to amniotic fluid embolism complicated with uterine atony'*.

Postoperatively, she was sent to intensive care unit (ICU) for close monitoring, intubated and sedated. Her hemodynamic status was supported with noradrenaline infusion @15ml/h. During ICU stay, she still had ongoing bleeding with blood loss of 1 L and was transfused with 2 pints of PC, 4 units of FFP and 6 units of cryoprecipitate. In ICU, bedside scan was done with the result of ventricles kissing, ejection fraction of 57%, minimal pleural effusion and bilateral atelectasis.

On the same day, her computed tomographic pulmonary angiogram (CTPA) was done to rule out any possible cause of cardiac arrest such as pulmonary embolism or lung pathology (Figure 1). The result shows that her lungs had a filling defect in the posterior segmental branch of both lower lobe pulmonary arteries in keeping with pulmonary embolism and bilateral pleural effusion with adjacent consolidations in keeping with infective changes. Inj. Clexane® 60 mg BD was started subcutaneously for PE treatment, when bleeding was controlled.

After 48 h in ICU, she underwent repeat laparotomy, removal of abdominal packing and peritoneal washout. Her GCS did not improve despite the sedation was off for two days postoperatively. Therefore, serial investigation was done to exclude any possibilities. Computed tomography (CT) brain was done on day 4 post event (Figure 2), Magnetic resonance imaging (MRI) brain was done on day 7 post event (Figure 3) were done, which consistently showed severe hypoxic ischemic encephalopathy (HIE). Electroencephalogram (EEG) was also done on day 8 post event to see any improvement in brain activity, with the same result as HIE features.

Family conference was done to brief for tracheostomy to help patient wean off ventilation and prevent further complications. Tracheostomy was done by ENT team on day 16 of intubation. Subsequently, the patient gradually improved and was shifted from ICU to the general ward. She was discharged home after almost 2 months in hospital. For PE treatment, inj Clexane was changed to tablet rivaroxaban 15 mg BD.

3. DISCUSSION

The prevalence of acute PE per 100,000 pregnancies increased with advancing maternal age.[3] Acute PE patients were more likely to be black, obese, smokers, hypertensive, diabetic, had heart failure, thrombophilia, and pre- eclampsia or eclampsia but were less likely to have gestational hypertension and gestational diabetes.[3] Of those with acute PEs, deep venous thromboses (DVT) were present in 26% of patients.[3] However, our current case describes the late diagnosis of PE because this patient presenting with low risk predictive factors. Despite her advanced age, she only had gestational diabetes and was class 1 obese with a BMI 34 kg/m2. This reduces the likelihood of an early suspicion when the patient presents early.

For PE related to pregnancy, the European Society of Cardiology (ESC) recommends a two-step risk stratification.[5] High-risk PE is first recognized by the presence of cardiac arrest or hemodynamic instability. Aside from cardiac arrest, the current ESC criteria include persistent hypotension, which is defined as a systolic blood pressure (SBP) less than 90 mmHg or a decline of SBP less than 40 mmHg without another cause. Second, in hemodynamically stable patients, the presence of RV dilation, elevated troponin I or T, elevated B-type natriuretic peptide (BNP) or N-terminal-pro BNP identifies patients who may benefit from more intensive hemodynamic monitoring.[5] Since our current case is accompanied by cardiac arrest and hemodynamic instability, it meets the risk stratification for PE related to pregnancy. Pregnant women often have non-specific symptoms for PE, such as tachycardia or shortness of breath atexertion.[1]

The gold standard for diagnosing PE in pregnant women up until recently was imaging by CTPA.[1] In the present case, the diagnosis of PE was based on a CTPA which revealed an abnormally filling defect in the posterior segmental branch of both lower lobe pulmonary arteries. In a recent systemic review, both CTPA and V/Q scans were shown to be appropriate for excluding PE during

pregnancy.[1] However, it is not recommended to routinely perform CXR in all patients with suspected PE before CTPA.[1]

For the management of high-risk PE during pregnancy, there are various therapeutic options. Current recommendations include thrombolytic therapy, surgical embolectomy, extracorporeal membrane oxygenation (ECMO), and catheter-guided thrombectomy for life-threatening PE.[5] Unfortunately, this could not be performed in the current case because of the risk of bleeding expected with patient in DIC state. In such cases, anticoagulant therapy was initiated as to prevent further obstruction and dissemination by PE.

In adults, cardiopulmonary arrest is the most frequent cause of HIE.[6] Compared to CT, MRI is more sensitive and has a stronger association with the temporal and geographical distribution of clinical events following a hypoxic-ischemic insult.[6] Our patient arrested for 30 min and the risk of HIE was there as the GCS still not improve despite off the sedation. Therefore, the diagnosis of HIE related to cardiopulmonary arrest was confirmed by CT and MRI. EEG was used to further validate the ultimate diagnosis of HIE and to evaluate the survival following cardiac arrest.

4. CONCLUSION

Acute PE during pregnancy has been associated with higher rates of morbidity and mortality in women. Pregnancy-associated PE should be closely monitored by physicians, especially in high-risk patients. In the vulnerable patient population, better preventive and management techniques need to be adopted promptly. Therefore, thromboprophylaxis is recommended for women having a history of PE in subsequent pregnancies as well as in probable low to high risk patient.

Conflict of interest

Ethical issues

Written informed consent was obtained from the next of kin (NoK) of the patient for publishing for educational purposes.

Authors contribution

MRH, MFIMS: Conducted the case, literature review, manuscript writing

MMM, MSS: Review of the manuscript, final approval of manuscript

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