Initial Management of Acute Pulmonary Edema in Pre-Eclampsia: A Case Report

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ABSTRACT

Objective: Describe the initial management for acute pulmonary edema in pre-eclampsia.
Methods: A case report.
Case: 41-year-old woman, G5P4A0, unknown gestational age, admitted with headache, dyspnea, and unconsciousness. The patient denied a history of past illness. Crisis hypertension, tachypnea, tachycardia, low oxygen saturation, audible lung crackles, pretibial edema, and 3+ proteinuria were found on examination, consistent with the diagnosis of pre-eclampsia with severe features. For initial management of acute pulmonary edema, the patient was given oxygen with Jackson-Rees bagging and furosemide injection, while nicardipine drip and MgSO4 injection were also given to treat pre-eclampsia. After the right initial management, patient was fully conscious, stable, then treated in the intensive care unit using non-invasive positive pressure ventilation. The patient underwent vaginal delivery and gave birth to male, BW 2900gram, BL 47cm, APGAR 3/8.
Discussion: Initial emergency management focuses on providing adequate oxygen is the main management of pulmonary edema in preeclampsia. A non-invasive positive pressure ventilation is required as the main flow of oxygen delivery because it's not invasive and is more convenient. Additional therapy such as diuretics, antihypertensive, and anticonvulsant are also needed. Close monitoring in the intensive care unit was also required for best results.
Conclusion: Initial management of acute pulmonary edema in pre-eclampsia patients should be done correctly, consequently decreasing maternal and perinatal morbidity and mortality.
Keywords: pregnancy, pre-eclampsia, pre-eclampsia with severe features, pulmonary edema.

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Introduction

Preeclampsia is one common complication in pregnancy and causes 10-15% of morbidity and mortality in pregnant women.[1] Based on the American College of Obstetricians and Gynecologists, severe preeclampsia or preeclampsia with severe features specifically defined as new hypertension presenting after 20 weeks with significant systolic blood pressure 160 mmHg or more or diastolic blood pressure 110 mmHg or more on two examinations at least 4 hours apart accompanied by massive or absence of proteinuria, or thrombocytopenia <100 x 10^9/L, or progressive renal insufficiency showed by serum creatinine >1.1mg/dL, or an impaired liver function showed by elevated in transaminases concentration, or the presence of pain in epigastric region or right upper

abdominal region, or the presence of pulmonary edema or neurological symptoms.[2]

One of the complications that occur in severe preeclampsia is acute pulmonary edema, with an incidence rate of only about 0.08%[3] and it's the leading cause of death in women with pre-eclampsia.[4] Pulmonary edema in patients with preeclampsia is a life-threatening condition, increasing mortality and morbidity in both maternal and perinatal, therefore the management of preeclampsia patients with pulmonary edema is important and must be carried out appropriately.[5]

Methods

This is a case report about 41-year-old woman with pulmonary edema as a severe feature of pre-eclampsia. The patient was initially treated in the emergency unit and after stabilized patient was transferred to the intensive care unit, patient then underwent vaginal delivery to a healthy baby. This case report is important because most publications on this topic only explain the maternal and perinatal outcomes without explaining the initial management that led to a good outcome.

Case Report

Woman, 41-year-old, G5P4A0, unknown gestational age, presented to the emergency room (ER) with sudden onset of headache, difficulty of breathing, and unconsciousness. The patient had no other complaints, never had a pregnancy checked, denied a history of similar complaints in a previous pregnancy, and denied a history of past illness. Initial examination, the patient appeared somnolence ($E_3M_5V_2$) and dyspneic, blood pressure (BP) was 210/120mmHg, heart rate (HR) was 104 beats/minute, respiratory rate (RR) was 32 times/minute, oxygen saturation was 56% in room air, and had normal temperature 36.6°C. On physical examination, audible lung crackles, bilateral pretibial edema, and normal patellar reflexes were found, BMI was 27.7 kg/m2 before pregnancy marked an obesity grade I based on the Asia-Pacific category. On obstetric examination, no uterine contractions were found, fundal height was 34cm, Leopold's examination showed breech presentation and pelvic examination marked 1 cm dilatation without a bloody show. Fetal heart sound was detected at 110x/minute by Doppler, marked fetal bradycardia. On laboratory examination, routine blood tests, liver function (AST and ALT), BUN, and creatinine were found normal. Urine dipstick showed proteinuria (3+). The patient was suspected with acute pulmonary edema as a severe feature of pre-eclampsia and fetal distress.

Initial emergency management focuses on providing adequate oxygen to the patient. We first used a non-rebreathing mask with 15 l/m, but the oxygen saturation did not increase so it was replaced with a bag-valve-mask, the oxygen saturation increased to 69-70% but the patient's consciousness remained somnolence $E_3M_5V_2$, which we then replaced with Jackson-Rees bagging, oxygen saturation increased to 92-93%. While the patient was receiving adequate oxygenation, a Foley catheter placed, 250 ml of urine was collected and an intramuscular injection of 5 g $MgSO_4$ was administered into the right and left buttocks.

After intravenous (i.v.) access was obtained, furosemide 40 mg injection was administered to reduce fluid overload, maintenance of 6 g $MgSO_4$ in Ringer's lactate solution was administered to prevent convulsions, 6

mg of dexamethasone injection was given to fetal lung maturation, and nicardipine at a 5 mg/hour was also administered using a syringe pump to reduce the blood pressure.

Following 30 minutes of Jackson-Rees bagging and i.v. injection therapy, oxygen saturation stabilized at 93-94%, patient's level of consciousness was raised to apathetic $E_3M_6V_4$, and oxygen administration was replaced using a 15 m/l non-rebreathing mask. The patient was stabilized for another hour in the ER, the patient was then concious $E_4M_6V_5$, BP was 147/89mmHg, HR was 97x/m, RR was 30x/m, and O_2 saturation was 91%. Urine collected was approximately 700ml, which showed urine output was exceeded 0.5/hour. The patient was transferred to the intensive care unit (ICU) for further stabilization using non-invasive positive pressure ventilation (NPPV). After 30 minutes in the ICU, a cardiotocography (CTG) was performed and showed a non-reassuring category II result. The patient was then positioned on her left side and an emergency cesarean section was planned. CTG was not repeated after that.

Due to the limited number of obstetricians, the emergency cesarean section has been postponed. Seven hours later, the patient complained of strong and regular contractions with a discharge from the birth canal. Obstetric examination revealed an 8 cm dilatation, a non-intact amniotic membrane with clear amniotic fluid, and bloody mucus. She gave birth to a male, weighing 2900 grams, 47cm body length, APGAR 3/8/9, New Ballard Score equal to 36 weeks of physical maturity, and treated to level II special care nursery room.

One day after delivery, the patient underwent an echocardiography and chest X-ray. Her systolic ejection function was 52.6%, and she had moderate pulmonary edema. She was treated in the ICU using NIV for the next 2 days. While in the ICU, vital signs, fluid balance, and urine output were closely monitored. A negative fluid balance was established by restricting the patient's drinking and intravenous fluids to remove excess fluid while maintaining a urine output of > 0.5ml/hour. Treatment of furosemide intravenously was continued 20mg twice/day with additional administration of candesartan 8mg/day, amlodipine 10mg/day, cefadroxil 500mg 2x/day, mefenamic acid 3x/day, while the use of dexamethasone, $MgSO_4$, and nicardipine were stopped. Afterward, the patient was treated in the hospital ward for another 2 days, then in the outpatient care with a stable condition.

Discussion

Preeclampsia is a multi-system disorder in pregnancy characterized by new-onset hypertension and organ failure after 20 weeks of gestation. Until now the pathophysiology of pre-eclampsia is very complex and unknown. The main hypothesis for the cause of pre-eclampsia is the failure of re-modeling of the spiral arteries which was initiated due to impaired penetration of the cytotrophoblast into the spiral artery muscle layer, causing placental dysfunction that releases pro-inflammatory factors and angiogenic proteins in the maternal circulation and leads to systemic endothelial disturbance.[6] Several risk factors such as nulliparous patients, aged > 40 years, multiparas with a previous history of pre-eclampsia or a history of chronic disease, multiple pregnancies, obesity before pregnancy, or pregnancies > 10 years apart are often associated with pre-eclampsia.[7]

Preeclampsia is also characterized by an organ failure,

including the cardiovascular system. Complications in the cardiovascular system that can be found are heart failure, stroke, and pulmonary edema. Acute pulmonary edema is a rare complication in pre-eclampsia with an incidence rate of only 0.08%.[5] Some factors that increase the risk of pulmonary edema in preeclampsia include pregnancy at an advanced age, obesity, receiving tocolytic therapy, having a history of heart problems before pregnancy, and administering too much fluids in the treatment of pre-eclampsia.[3]

Acute pulmonary edema caused by preeclampsia is defined as acute respiratory distress that occurs during pregnancy or within the first 45 days after delivery, and more often occurs before birth compared to after birth. It is characterized by sudden shortness of breath, moist crackles, desaturation, and an X-ray examination that describes pulmonary edema. The pathophysiology underlying the development of acute pulmonary edema in preeclampsia is due to increased plasma volume or cardiac output and decreased plasma colloid osmolarity or osmolality during pregnancy.[8] There is also an increase in peripheral vascular permeability and peripheral vascular resistance in preeclampsia which increases after-load.[9] Diagnosis of acute pulmonary edema requires echocardiography which is later used to differentiate between cardiogenic and noncardiogenic causes. In noncardiogenic acute pulmonary edema, echocardiography results usually do not reveal any abnormalities and preeclampsia is usually the cause.[8]

The most important initial management of acute pulmonary edema caused by preeclampsia is giving adequate oxygen, fluid restriction, and lowering blood pressure with intravenous antihypertensive agents, such as beta-blockers (labetalol), diuretics (furosemide), vasodilators (nitroglycerin, hydralazine), or using calcium channel antagonists (nicardipine, nifedipine).[5,9] Additionally, fluid therapy in preeclampsia is still needed to maintain fluid and electrolyte balance or replace lost intravascular volume, but it should be restricted. The recommended administration is approximately 60-80 ml/hour, with urine output, stool, and insensible loss calculated and monitored based on clinical observations.[9]

According to ESC guidelines for acute pulmonary edema, three commendable treatments exist. First, oxygen should initially be administered as continuous positive airway pressure, non-invasive positive-pressure ventilation (NPPV), or high-flow nasal cannula (HFNC). Second, i.v. diuretics is needed. Last, if systolic BP is high, an i.v. vasodilators can be given.[10] NPPV (non-invasive positive pressure ventilation) is required as the main flow of oxygen delivery because it's not invasive, compared to HFNC, NPPV/NIV is more convenient because of its ability to heat and humidify the gas.[11] In several studies that have been conducted, administration of NPPV during pregnancy can prolong gestation with better neonatal outcomes and its use improves oxygenation and pH, decreases the work of breathing and partial pressure of carbon dioxide, and also prevents intubation.[12,13]

Giving MgSO4 in preeclampsia with acute pulmonary edema is still a debate. MgSO4 has benefits as anti-eclampsia and neuroprotective for the fetus, but the use of MgSO4 in pre-eclampsia patients increases the risk of acute pulmonary edema. However, if the administration of MgSO4 provides benefits outweigh the consequences,

then MgSO4 can be administered under close monitoring.[14]

Acute pulmonary edema causes acceleration of birth, which causes an increase in the rate of prematurity, neonatal morbidity and mortality, and neonatal asphyxia with the need for neonatal resuscitation.[8] Termination of pregnancy is the only treatment for preeclampsia. Patients with preeclampsia with severe features are delivered immediately to prevent maternal and fetal complications.[4] As per ACOG, preeclampsia with severe features at or beyond 34 0/7 weeks must undergo delivery after maternal stabilization and should not be delayed to allow steroid administration.[13] Although in the case of preeclampsia with severe features, labor induction can be performed using prostaglandin or an osmotic dilator. However, if vaginal delivery is not successful or attempts failed, then cesarean delivery is indicated.[4]

A retrospective case-control study found in a referred hospital in Indonesia concluded that pulmonary edema increased the morbidity and mortality for the maternal and perinatal, and major cases needed intensive care and even mechanical ventilation support[15]. Another literature review found also concluded that acute pulmonary edema is a medical emergency so an emergency response must be initiated[16]. NPPV should be tried as an initial treatment before tracheal intubation to maintain adequate oxygenation and ventilation. An i.v. furosemide (20-40 mg) is also used for venodilation and diuresis, and an i.v. antihypertensive agent such as nitroglycerin starting 5 μg/min is necessary to reduce high blood pressure. High-dependency care and close observation are essential and should be carried out by a multidisciplinary team.

Conclusion

Acute pulmonary edema as a severe feature of preeclampsia is quite rare but it's the leading cause of death in preeclampsia. It also has a high risk of maternal and neonatal morbidity and mortality. Giving an adequate oxygenation is the most important initial management, and additional treatment such as diuretics, anti-hypertensive, anti-convulsion, and fluid restriction can provide a good outcome which consequently reducing morbidity and risk of mortality on the patient and perinatal.

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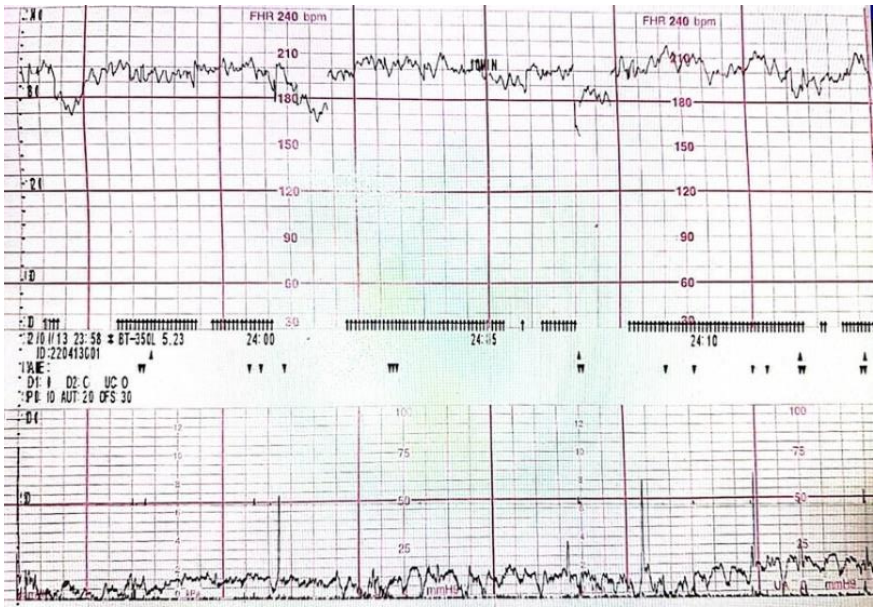

Figure 1. Cardiotocography in the ICU showed a non-reassuring category II result

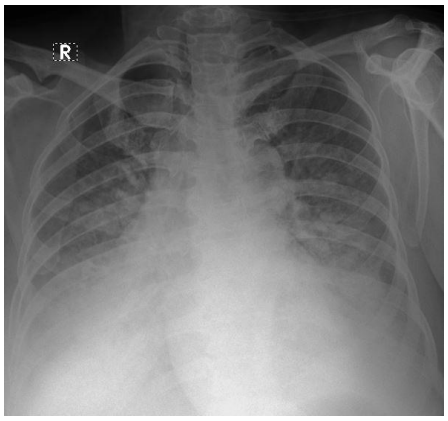

Figure 2. Postpartum Chest X-ray showed moderate pulmonary edema