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Acute postpartum pulmonary edema in severe preeclampsia

Fernando Nathaniel[1], Freddy Dinata[2]

[1] Faculty of Medicine, Tarumanagara University, Jakarta, Indonesia

[2] Departement of Obstetrics and Gynecology, Ciawi Regional General Hospital, West Java, Indonesia

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ABSTRACT

Pulmonary edema is a feature of preeclampsia's severe end of the disease spectrum. It refers to excessive fluid accumulation in the pulmonary interstitial and alveolar spaces. We report a case 50-year-old G3P3 Sundanese woman who presented to the emergency department of Ciawi Regional General Hospital with progressive dyspnea after five days post-Cesarean section. On physical examination, her blood pressure was severely elevated. Her clinical manifestations, along with investigations, were consistent with pulmonary edema and preeclampsia was likely the cause. The patient started receiving antihypertensive, salt and fluid restriction, and diuretic. The patient improved clinically after five days of treatment. Acute pulmonary edema is a life-threatening event. Therefore, appropriate and adequate management and multidisciplinary interventions were necessary to manage this case.

Corresponding Author:

Fernando Nathaniel,
Faculty of Medicine,
Tarumanagara University,
Jakarta, Indonesia,
Email: fernandonathaniel24@gmail.com

INTRODUCTION

Pulmonary edema (PE) is an uncommon but severe pregnancy complication. This condition exacerbated the physiological changes of pregnancy and the contributing effect of poorly understood pathophysiology of pregnancy-related diseases like preeclampsia, which is correlated with significant morbidity and mortality for patient (Ellyzabeth, 2018; You et al., 2018). Pre-pregnancy conditions (e.g., hypertension, cardiomyopathy, arrhythmias, obesity, endocrine disorders, increased maternal age), specific pregnancy conditions (e.g., preeclampsia, sepsis, embolism), pharmacological agents (e.g., corticosteroids), iatrogenic (e.g., fluid overload), and fetal conditions (e.g., multiple gestations) are all risk factors for the development of acute pulmonary edema (E. et al., 2018; Vaught et al., 2018). Upon reviewing the patient's chart, her total fluid consumption throughout the postpartum period was roughly 2.5 liters per day. If all subsequent fluid losses were considered, iatrogenic fluid overload was an improbable cause. A combination of various risk factors (e.g., hypertension, preeclampsia, obesity, increased maternal age, diabetes) are demonstrated in this case that could have a role in acute pulmonary edema (Macedo et al., 2020; Sukmawati, 2017; Sukmawati et al., 2021; Vaught et al., 2018).

Structural or functional cardiac defects predispose to develop pulmonary edema. Preeclampsia is a known risk factor for cardiomyopathy, both peripartum and years after delivery (Behrens et al., 2016; Sukmawati, 2018). Women with severe preeclampsia have abnormal cardiac remodeling, with echocardiographic evidence of diastolic dysfunction and LV remodeling in some patients (Vaught et al., 2018). Although there may be some overlap between preeclampsia and peripartum cardiomyopathy, some evidence suggests that both of these disorders have similar pathogenesis, though unified theories should be investigated (Parikh & Blauwet, 2018). The patient was not assigned to an echocardiographic examination in this current case, making it difficult to assess for any structural or functional abnormalities of the heart (American Heart Association, 2021; Perhimpunan Dokter Hipertensi Indonesia, 2021).

Established risk factors for preeclampsia in women with type 1 and type 2 diabetes comprise nulliparity, advanced maternal age, preceding preeclampsia, hypertension, a longer duration of diabetes, poor glycemic control, microalbuminuria, nephropathy, and retinopathy. Obesity is a shared risk factor for both preeclampsia and type 2 diabetes (Weissgerber & Mudd, 2015). More research is needed to examine the relation between pregestational diabetes and preeclampsia sequelae, particularly pulmonary edema (Honardoost et al., 2021; Rollo et al., 2020; Santosa & Baharuddin, 2020).

The mechanisms involved in preeclampsia-induced pulmonary edema are largely undefined. This complication could have been associated with several mechanisms. Elevated plasma volume, reduced plasma oncotic pressure, increased capillary permeability, and pulmonary capillary hydrostatic pressures are elements of these. Cardiac functions were shown to be changed with pulmonary edema. Most patients had preserved systolic function, which could have been the case with our patient (Mudrikatin, 2020; Santosa & Baharuddin, 2020).

The clinical manifestation of severe preeclampsia- or eclampsia-related pulmonary edema is substantially indistinguishable from that of other causes of pulmonary edema: clinical symptoms (dyspnea, orthopnea, restlessness, palpitation, and cough) and signs (tachycardia, tachypnea, rales, and wheeze, S3 gallop rhythm, and heart murmurs, decreased oxygen saturation) (Pordeus et al., 2018). In this case, a combination of dyspnea, chest discomfort, and/or low (93 percent) oxygen saturation by pulse oximetry is linked to a poor maternal outcome (maternal death and hepatic, renal, central nervous system, cardiorespiratory, and hematologic morbidities) (Millman et al., 2011).

As with typical preeclampsia, the patient may exhibit various symptoms, including headache, dyspnea, visual symptoms, nausea, vomiting, epigastric pain, seizure, edema, and oliguria. Prior to the presentation, our patient displayed the majority of the symptoms listed. Severe hypertension and pulmonary edema indicate critical conditions requiring immediate attention.

The mainstay of treatment was the treatment of hypertension, salt, and fluid restriction, as well as diuresis with furosemide. Fluid balance (input against urine output Including predicted insensible losses [typically 30 to 50 mL/hour]) should be evaluated carefully. A maintenance infusion of an isotonic saline solution of around 80 mL/hour is frequently sufficient for a patient who is nil by mouth and has no continuing abnormal fluid losses (Anthony & Schoeman, 2013). Supplemental oxygen was used as needed for hypoxia. Our patient recovered quickly and was scheduled for debridement of her diabetic ulcer and discharged within a few days as a result of proper diagnosis and management.

RESEARCH METHOD

Here we discuss the case of a 50-year-old woman who has developed severe preeclampsia complicated by acute pulmonary edema in the early postpartum period. A literature review regarding pulmonary edema is discussed.

RESULT AND DISCUSSION

A 50-year-old P3A0 Sundanese woman, with a history of recent Cesarean section 5 days before, attended the emergency department on RSUD Ciawi with a chief complaint of shortness of breath for the last two days. She stated that she experienced this complaint about the first time. Other complaints include pain or discomfort in the epigastric, nausea, vomiting, bloating, persistent dry cough, fatigue, and swelling of extremities. On the first toe of the left foot, there was a small ulcer with reduced sensation.

The patient was in relatively good health until approximately seven years ago, when she was diagnosed with hypertension and diabetes mellitus. She also had obesity. There is no family history of preeclampsia or diabetes mellitus. Patients often visit the doctor every month, taking metformin 500mg b.i.d. and amlodipine 5mg q.d. to control blood sugar and blood pressure. Unfortunately, the patient had not been monitored in the last two years due to the COVID-19 Pandemic.

The patient has three children. The first and second were born spontaneously, full-term, with average birth weight, with the help of a midwife. She denied any symptoms and severe conditions during both previous pregnancies. During the obstetrical check-up of her third pregnancy at 20 weeks gestation, her blood pressure was elevated, reaching 160/100 mmHg, with laboratory tests resulting in proteinuria without any sign of renal or hepatic disorder. Given her ongoing preeclampsia, a cesarean section was scheduled for 34 weeks of gestation. One day before the cesarean section, an examination showed ureum at 95.4 mg/dL and creatinine at 3.07 mg/dL.

On physical examination, the woman was orthopnoeic. She had a respiratory rate of 32/min, a heart rate of 120/min, and regular, SPO2 of 92% room air, along with a high blood pressure of 190/100 mmHg. Elevated jugular venous pressure, and auscultation revealed fine crackles were detected bilaterally with decreased basilar breath sounds. Bilateral pedal pitting edema was also noted. She had a gangrenous open wound on the first toe of her left foot. Chest X-ray at admission had features of cardiomegaly and increased bronchovascular markings (figure 1). In addition, sinus tachycardia with a transition point at lead V5-V6 suggests clockwise rotation in ECG examination (figure 2).

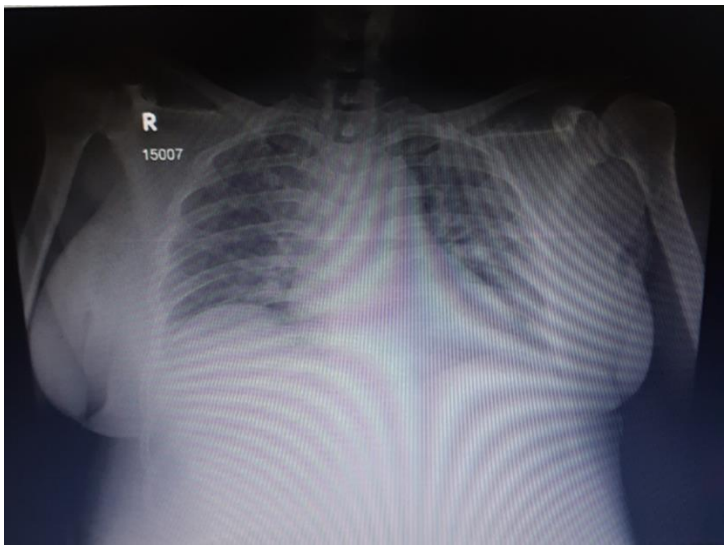

Figure 1 Chest X-ray showed increased bronchovascular markings and cardiomegaly.

Result from blood investigations revealed moderate anemia (hemoglobin 9.9 g/dL) [reference value: 11.7-15.5 d/dL], hematocrit 27% [reference value : 35-47%], total leucocyte count of 17,100/µL (1% eosinophils, 0% basophils, 83% neutrophils, 9% of lymphocytes, and of 7% monocytes) [reference value: 4,000 – 11,000/µL], thrombocyte 477,000 [reference value: 150,000-440,000/µL]. Biochemistry investigation revealed blood sugar 85 mg/dL [reference value: 80-120 mg/dL], high ureum at 81.5 mg/dL [reference value: 10-50 mg/dL], high creatinine 2.28 mg/dL [reference value: 0.6-1.3 mg/dL], serum glutamic oxaloacetic transaminase 22 U/L [reference value: 0-35 U/L], serum glutamic pyruvic transaminase 11 [reference value : 0-35 U/L], albumin 1.79 [reference value: 3.5-5.5 g/dL], high NT-proBNP 2,031 pg/mL [reference value: <125 pg/mL]. Whereas urine investigation revealed protein 4+ and erythrocyte sedimentation 10-15/HPF.

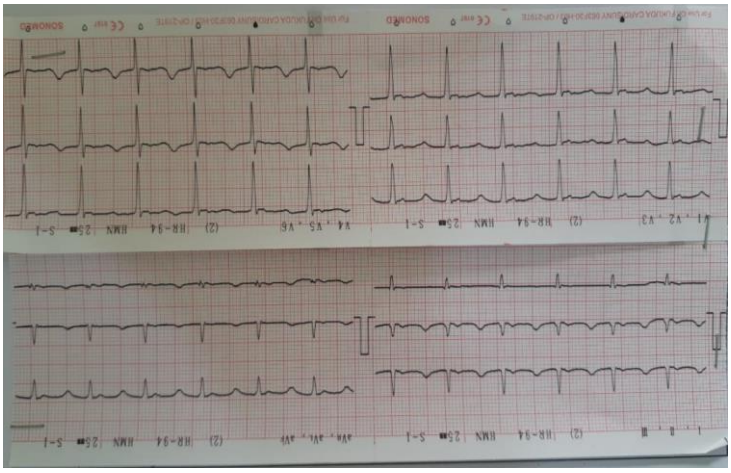

Figure 2 The ECG at admission shows sinus tachycardia and clockwise rotation.

In terms of patient management, the patient was transferred to the High Care Unit (HCU) for further monitoring and to receive joint care from an obstetrician, cardiologist, and general surgeon. The patient was managed conservatively, including a propped-up position, administration of oxygen using nasal cannulae (5 lpm oxygen), an intravenous infusion of NaCl 500cc every 24 hours, and furosemide 5mg IV every hour. Oral medications were ramipril 5 mg b.i.d., spironolactone 25 mg q.d., and bromocriptine 2,5 mg b.i.d.. The patient's blood pressure remained high after the third day of initial treatment, so the ramipril dose was increased to 10 mg b.i.d., and oral bisoprolol 2.5 mg q.d. was added. The patient improved clinically after three days of treatment and was scheduled for debridement of her left toes

Based on the patient's presentation, various illnesses may result in acute postpartum dyspnea. Conditions unrelated to pulmonary edema include pneumonia, pulmonary embolism, and amniotic fluid embolism. Conditions related to pulmonary edema can be either cardiogenic or noncardiogenic causes can cause pulmonary edema during pregnancy or peripartum.

Based on the medical history, the patient was diagnosed with chronic hypertension superimposed with severe preeclampsia and pregestational diabetes mellitus. Clinical symptoms of orthopnea, dyspnea on exertion, jugular venous distention, pitting edema, high NT-proBNP, abnormal ECG, and abnormalities on chest radiograph on hospitalization are consistent with pulmonary edema with noncardiogenic pulmonary edema was the likely cause.

Acute pulmonary edema is a rare but potentially fatal condition that causes significant morbidity and mortality in both the mother and the fetus. It affects between 0.08 and 1.5 percent of pregnant and postpartum women (Sciscione et al., 2003). Its primarily associated with hypertension. In a prospective case-control study, 10% out of 63 cases of preeclampsia with severe features developed pulmonary edema (Vaught et al., 2018). Acute pulmonary edema can occur in antenatal,

intrapartum, and postpartum periods. In a case series, 70 percent of patients with pulmonary edema acquired it during the postpartum period (a mean of 71 hours following birth) (Sibai et al., 1987).

CONCLUSION

Acute pulmonary edema is one of the primary causes of death in women with severe preeclampsia. It is a rare entity; Identifying the at-risk patient, recognizing indicators of acute disease, and managing with a professional multidisciplinary team is vital. Risk reduction approaches should include attention to the significance of fluid balance and documenting regular clinical observations. Appropriate long-term follow-up is essential to limit the possibility of developing complications in later stages of life.

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