Journal of Society Medicine
Research & Review Articles on Diseases
Journal of Society Medicine. 2025; 4 (3)

Management of A Preeclampsia Patient with Diabetic Ketoacidosis and Acute Pulmonary Edema : A Case Report

Osmond Muftilov Pison [1*], Arna Fransisca MP [2]

[1]*Consultant Intensive Care, Faculty of Medicine Padjadjaran University / Hasan Sadikin General Hospital Bandung,*
[2] *Intensive Care Trainee, Faculty of Medicine Padjadjaran University / Hasan Sadikin General Hospital Bandung*

***Corresponding Author**: Osmond Muftilov Pison, Email: muftilov03@gmail.com

ARTICLE INFO

Article history:
Received
28 January 2025

Revised
21 February 2025

Accepted
31 March 2025

Manuscript ID:
JSOCMED-28012025-43-1

Checked for Plagiarism: Yes

Language Editor: Rebecca

Editor-Chief:
Prof. AznanLelo, PhD

ABSTRACT

Introduction: Preeclampsia is a hypertensive disorder that develops after 20 weeks of gestation, often accompanied by proteinuria and maternal organ dysfunction, including pulmonary edema. Women with diabetes are at a higher risk for developing preeclampsia and its complications. Managing severe preeclampsia requires prompt monitoring and intervention, particularly when organ failure and metabolic disturbances are present.

Case Report: A 25-year-old pregnant woman at 30-31 weeks of gestation presented with severe dyspnea, high blood pressure (185/105 mmHg), and metabolic disturbances, including diabetic ketoacidosis (DKA) and pulmonary edema. The patient had no prior history of hypertension or diabetes. Laboratory tests revealed elevated ketones, hyponatremia, and mild proteinuria. She was admitted to the ICU, where she received nitroglycerin infusion, fluid resuscitation, and blood glucose regulation. However, her condition worsened, and she required intubation and mechanical ventilation due to severe hypoxemia and respiratory failure. After stabilization, a cesarean section was performed, leading to improvements in her condition. The patient was extubated after three days, and her blood glucose levels were stabilized.

Conclusion: This case highlights the complexities of managing severe preeclampsia complicated by pulmonary edema, diabetic ketoacidosis, and urinary tract infections. Early recognition, aggressive treatment, and multidisciplinary care are essential for improving outcomes and preventing further complications in high-risk pregnancies.

Keywords

Preeclampsia, Diabetic Ketoacidosis, Acute Pulmonary Edema

How to cite: Pison OM, Fransisca MPA. Management of A Preeclampsia Patient with Diabetic Ketoacidosis and Acute Pulmonary Edema : A Case Report. *Journal of Society Medicine*. 2025;4(3):70-75. **DOI: https://doi.org/10.71197/jsocmed.v4i3.196**

INTRODUCTION

Preeclampsia is defined as hypertension that newly occurs after 20 weeks of gestation, accompanied by proteinuria; severe maternal organ dysfunction may be associated with fetal growth restriction.[1,2] Preeclampsia with severe symptoms necessitates rapid hypertension control and requisite organ management prior to immediate delivery.[2] Patients are admitted to the intensive care unit (ICU) in cases of uncontrolled hypertension, management of complications such as seizures, stroke, pulmonary edema, liver failure, hemorrhage and coagulopathy, and for postpartum monitoring and care.[2]

Pulmonary edema is one of the severe complications of preeclampsia. The incidence of pulmonary edema complicating preeclampsia is reported at 2.9% and constitutes one of the primary reasons for admission to the intensive care unit (ICU), with more critical situations requiring mechanical ventilation.[3,4] Pulmonary edema is more likely to occur in pregnant patients due to increased cardiac output and blood volume, and

decreased plasma oncotic pressure.[5,6] The risk of preeclampsia increases two to four-fold in women with type 1 or type 2 diabetes, as well as gestational diabetes mellitus.[7,8] Diabetic ketoacidosis is a potentially life-threatening condition in pregnancy that affects 0.5-3% of diabetic pregnancies.[9] Pregnancy is also associated with physiological changes that may predispose a pregnant woman with diabetes to diabetic ketoacidosis. The physiological changes of pregnancy and diabetes are also contributing factors to the increased incidence of urinary tract infections during pregnancy.[10]

CASE REPORT

A 25-year-old woman, gravida 3, para 2 (G3P2), at 30-31 weeks of gestation, presented to Hasan Sadikin Hospital with a 3-day history of dyspnea. The patient denied any previous history of cough or fever. Her obstetric history included two cesarean sections, the first in 2018 and the second in 2022 due to hypertension during the second pregnancy. The patient denied any history of diabetes mellitus, and her antenatal care was irregular. There was no known history of hypertension in the current pregnancy.

On physical examination, the patient was conscious, with a blood pressure of 185/105 mmHg, a heart rate of 110 beats per minute, and an oxygen saturation of 95% with nasal cannula support. Her body temperature was 38.5°C. She exhibited peripheral edema but no other signs of heart failure. Laboratory findings showed significant abnormalities (Table 1).

Table 1. Laboratory Data and Abnormal Findings in the Patient

Laboratory Test	Result	Normal Range	Comments
KGD (Ketone Glucose Derivative)	505 mg/dL	N/A	Elevated, indicative of metabolic disturbance
Anion Gap	20.8 mEq/L	8–16 mEq/L	Elevated, suggests metabolic acidosis
Sodium (Na)	129 mEq/L	135–145 mEq/L	Hyponatremia
Liver Function Tests (LFTs)	Elevated	ALT: 7–56 U/L, AST: 8–48 U/L	Specific values for ALT/AST/Bilirubin could be listed if available
Proteinuria	+1	Negative	Mild proteinuria, potentially indicative of preeclampsia
Hemoglobin	10.2 g/dL	12–16 g/dL	Mild anemia
Platelets	Normal	150,000–450,000/µL	No platelet abnormalities

The patient was admitted to the maternal high care unit and started on NTG (Nitroglycerin) infusion at 100 mcg per minute, with normal saline (0.9%) fluid resuscitation of 2L over 3 hours. Blood glucose regulation was initiated with intravenous rapid insulin, and symptomatic management was provided. A termination of pregnancy was planned.

However, a few hours later, the patient's condition worsened. She became severely dyspneic, with a respiratory rate of 40 breaths per minute, tachycardia of 145 beats per minute, and desaturation. She exhibited bilateral rhonchi and worsening respiratory distress. Blood pressure remained controlled with NTG. Arterial blood gas (ABG) results showed Table 2.

Tabel 2. Arterial Blood Gas (ABG) Results and Interpretation

Parameter	Result	Normal Range	Comments
pH	7.26	7.35–7.45	Acidotic state
pCO2	26.9 mmHg	35–45 mmHg	Low, indicative of metabolic acidosis
pO2	52.6 mmHg	75–100 mmHg	Hypoxemia
HCO3	12.2 mEq/L	22–28 mEq/L	Low, suggests metabolic acidosis
Base Excess (BE)	-12.8	-2 to +2 mEq/L	Severe metabolic acidosis
SaO2	80%	95–100%	Low, reflecting severe hypoxemia

The arterial blood gas (ABG) results indicate severe metabolic acidosis with compensation through respiratory alkalosis. The low pH (7.26) and decreased bicarbonate (HCO3 12.2 mEq/L) confirm a metabolic acidosis state, while the low pCO2 (26.9 mmHg) suggests compensatory hyperventilation. Additionally, the low pO2 (52.6 mmHg) and SaO2 (80%) reflect severe hypoxemia, indicating impaired oxygenation. The negative base excess (-12.8) further emphasizes the severity of the metabolic acidosis. These findings point to a critical condition, requiring immediate intervention to correct the acid-base imbalance and improve oxygenation.

Chest X-ray revealed acute pulmonary edema without cardiomegaly. A diagnosis of respiratory failure was made, and the patient was intubated and placed on continuous diuretic therapy.

For Outcome and Follow-up, subsequent to stabilization, a cesarean section was performed, and the patient was placed on ventilator support with continued diuretic therapy, NTG, and blood glucose regulation. Postoperative symptomatic management was maintained. The patient's condition gradually improved, and the chest radiograph findings exhibited corresponding enhancement. Extubation was performed on the third day of intensive care unit management, and the patient continued to show clinical improvement.

The patient was diagnosed with G3P2A0 gravida at 30-31 weeks gestation, with a history of two cesarean sections, preeclampsia, partial HELLP syndrome, respiratory failure due to acute pulmonary edema, diabetic ketoacidosis, and urinary tract infection. Upon admission to the ICU, the patient was administered midazolam sedation at 5 mg/hour and placed on ventilator support with P-SIMV mode (R 12, PC 14, PS 12, PEEP 10, FiO2 60%), resulting in a tidal volume of 400-450 mL, minute ventilation of 6.8-7.5 L/minute, and SpO2 of 97-98%. The patient's blood pressure was 117/78 mmHg (with nitroglycerin at 100 mcg/min), heart rate was 138 beats/minute, and temperature was 38.9°C. Additional therapy included intravenous insulin at 4 units/hour (blood glucose 505 mg/dL) with monitoring to maintain a target range of 180-200 mg/dL, intravenous furosemide at 20 mg/hour, intravenous paracetamol at 1 gram every 6 hours, fluid loading, and maintenance with 0.9% NaCl. Anion gap measurement, blood, sputum, and urine cultures, and echocardiography consultation were ordered. The patient was temporarily placed on nil per os status pending consultation for pregnancy termination.

On the second day of care, blood glucose levels remained unstable, necessitating insulin titration to achieve the target range and hemodynamic stabilization. A central venous catheter was inserted. Echocardiography results revealed: normal chamber sizes, normal left ventricular systolic function (LV EF 60-65%), normokinetic at rest, normal anatomy and function of all valves, low probability of pulmonary hypertension, normal right ventricular systolic function, absence of pericardial effusion, and no evidence of thrombus. Pregnancy termination was performed with the patient under deep sedation (midazolam 5 mg/hour), with vital signs as follows: blood pressure 124/78 mmHg, pulse 135 beats/minute, respiratory rate 22 breaths/minute, oxygen saturation 98% on Jackson Rees circuit, and temperature 38°C. Fetal heart rate was 168 beats/minute. A cesarean section was performed under general anesthesia, with estimated blood loss of 400 mL (200 mL of packed red cells transfused intraoperatively), urine output of 250 mL, and 1000 mL of Ringer's lactate administered.

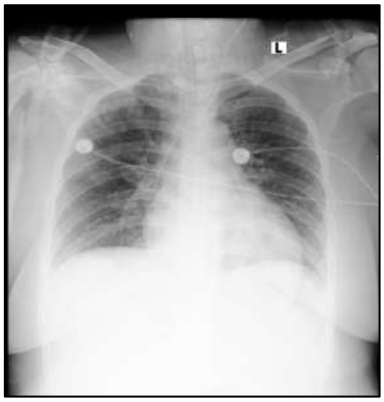

Figure 1. Post-Operative Chest X-ray

Post-operatively, the patient returned to the ICU with the following parameters: blood pressure 109/72 mmHg, pulse 130 beats/minute, respiratory rate 24 breaths/minute, oxygen saturation 96-97% on P-SIMV mode (R 12, PC 12, PS 10, PEEP 10, FiO2 50%), temperature 37.6°C, and blood glucose 370 mg/dL. Sedation was changed to dexmedetomidine at 0.4 mcg/kg/hour, nitroglycerin was discontinued, and insulin was titrated to achieve the target blood glucose range. A follow-up chest radiograph was ordered (Figure 1).

On the third day of treatment, the patient's consciousness and hemodynamic status exhibited improvement, with a reduction in rhonchi. However, blood glucose levels remained unstable, necessitating insulin administration in accordance with target blood glucose levels. Potassium correction was performed due to observed hypokalemia, potentially attributable to insulin administration and diuretic therapy. Gradual ventilator weaning was initiated. Repeat urinalysis and arterial blood gas analysis were conducted to assess ketone levels, pH, and bicarbonate.

On the fourth day of treatment, the patient's condition showed marked improvement, with a Glasgow Coma Scale score indicating full consciousness. Vital signs were as follows: blood pressure 129/69 mmHg, pulse rate 97 beats per minute, respiratory rate 20 breaths per minute, oxygen saturation 97%, and temperature 36.7°C. Ventilator weaning progressed to spontaneous mode, culminating in extubation. Sedation was titrated to cessation, insulin was titrated to achieve target blood glucose levels, and furosemide dosage was adjusted.

On the fifth day of treatment, the patient remained stable post-extubation, demonstrating no increase in respiratory effort and maintaining hemodynamic stability without support. Blood glucose level was recorded at 200 mg/dL with an insulin infusion rate of 2 units per hour. Subsequently, the patient was transferred to a step-down unit.

DISCUSSION

Preeclampsia (PE) is a significant contributor to maternal and neonatal morbidity and mortality worldwide, accounting for approximately 8% of all gestational-related complications. It is responsible for more than 50,000 maternal deaths and over 500,000 fetal deaths annually. PE is characterized by the onset of hypertension and end-organ dysfunction in a previously normotensive woman after 20 weeks of gestation, with or without proteinuria. Recent guidelines from the American College of Obstetrics and Gynecology (ACOG) have expanded the diagnostic criteria for PE, acknowledging that it can also be diagnosed in the absence of proteinuria when accompanied by complications such as pulmonary edema, renal insufficiency, impaired liver function, thrombocytopenia, or new-onset cerebral or visual disturbances. This expansion of the diagnostic criteria has important implications for early detection and intervention in high-risk pregnancies.[4]

Risk factors for developing PE are multifactorial, with a history of PE being the most significant predictor for recurrence in subsequent pregnancies. Other well-established risk factors include diabetes mellitus, multiple gestations, renal disease, autoimmune disorders, chronic hypertension, obesity, advanced maternal age, and nulliparity. Additionally, non-diabetic women who have previously experienced PE are at increased risk of developing type 2 diabetes later in life.[2,3] Although the exact etiology of PE remains unknown, the clinical manifestation of the disease is thought to result from a combination of maternal risk factors and abnormal placental development, leading to endothelial dysfunction, vasospasm, and impaired angiogenesis. This pathophysiology disrupts the balance between pro- and anti-angiogenic factors, increasing oxidative stress and ischemia, and ultimately contributing to widespread organ dysfunction, including edema.[3,4]

One organ that may be affected in PE is the pancreas, though there is limited research examining its role in this context. PE is often associated with abnormal glucose metabolism, including insulin resistance and decreased levels of glucagon-like peptide-1 (GLP-1), a peptide that promotes insulin secretion and helps regulate blood glucose levels.[5] Insulin resistance, a hallmark of PE, disrupts glucose homeostasis and is further compounded by lipid metabolism dysfunction and excessive inflammatory responses. These metabolic abnormalities are thought to contribute to dyslipidemia, hyperuricemia, hyperglycemia, and insulin resistance, all of which exacerbate the clinical condition of the patient.[1]

In the setting of insulin resistance, pregnant women with PE are at risk for developing diabetic ketoacidosis (DKA). The pathophysiology of DKA involves accelerated starvation and respiratory alkalosis, which induces compensatory renal bicarbonate excretion. This predisposes the patient to the development of metabolic acidosis.[6] Increased insulin resistance or inadequate insulin levels lead to the release of counter-regulatory hormones (cortisol, catecholamines, glucagon, and growth hormone), which in turn stimulate lipolysis, proteolysis, and gluconeogenesis. These processes, together with glycogenolysis, result in uncontrolled hyperglycemia. Additionally, these hormones activate hormone-sensitive lipase in adipose tissue, leading to the synthesis of non-esterified fatty acids, which serve as substrates for ketogenesis in the liver. Elevated glucagon levels and low insulin further enhance ketogenesis, contributing to high anion gap metabolic acidosis and increased respiratory rate and depth, known as Kussmaul respiration. This cascade of events can lead to osmotic diuresis, electrolyte imbalances, hypovolemia, and hypertonicity, all of which require prompt intervention to prevent maternal and fetal complications.[7]

In managing DKA in pregnancy, the primary goal is timely diagnosis, which allows for the correction of hypovolemia and improvement of renal and uteroplacental perfusion. Insulin therapy is essential to reduce serum glucose levels, while acidosis and electrolyte imbalances must be corrected. Additionally, it is crucial to identify and address any underlying causes of DKA, ensuring intensive monitoring of maternal and fetal responses to treatment.

This case highlights the complexity of managing preeclampsia, especially in patients with a history of the condition. The patient in this case has had two prior episodes of preeclampsia, significantly increasing the likelihood of recurrence and complicating her current pregnancy. Additionally, the risk of insulin resistance and metabolic disturbances, including hyperglycemia, further complicates her clinical course.

Pulmonary edema, a life-threatening complication of preeclampsia, can be caused by a variety of mechanisms, including decreased osmotic pressure, increased capillary permeability, and elevated hydrostatic pressure within the vasculature. These processes lead to the extravasation of fluid into the pulmonary interstitium and alveoli, resulting in pulmonary edema.[8,9] In the context of preeclampsia, this complication may occur antepartum or postpartum, and is often associated with endothelial dysfunction, corticosteroid or tocolytic therapy, and iatrogenic volume overload. The patient in this case developed pulmonary edema, likely exacerbated by the altered intravascular colloid-hydrostatic forces and increased vascular permeability associated with preeclampsia.[10,11]

Furthermore, pulmonary edema complicating diabetic ketoacidosis (DKA) is thought to result from increased pulmonary capillary permeability and disturbed colloid-hydrostatic forces. The combination of these pathophysiologic mechanisms necessitates careful management to prevent further deterioration in maternal and fetal health.

CONCLUSION

Preeclampsia with acute pulmonary edema accompanied by diabetic ketoacidosis in pregnancy constitutes a medical and obstetric emergency that necessitates aggressive, coordinated, and early intervention in a specialized unit. The complex management requires a multidisciplinary team to achieve favorable outcomes for both mother and fetus. The primary treatment for preeclampsia is pregnancy termination; however, the patient's condition must be evaluated for surgical suitability to ensure maternal and fetal safety during the perioperative period.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to publication in Journal of Society Medicine.

FUNDING
None

COMPETING INTERESTS
The authors declare that there is no conflict of interest in this review.

AUTHORS' CONTRIBUTIONS
All authors contributed to the work, including data analysis, drafting, and reviewing the article. They approved the final version and are accountable for all aspects.

ACKNOWLEDGMENTS
None

REFERENCE
1. Weissgerber TL, Mudd LM. Preeclampsia and Diabetes. Curr Diabetes Rep. 2015;15(3):579.
2. Wai JKM, Gin T. Preeclampsia and Eclampsia. In: Oh's Intensive Care Manual. 8th ed. 2019;64:787-792.
3. Coetzee A. Pregnancy and diabetic ketoacidosis: fetal jeopardy and windows of opportunity. Front Clin Diabetes Healthcare. 2023;4:1266017.
4. Ram M. Acute pulmonary edema due to severe preeclampsia in advanced maternal age women. Pregnancy Hypertens. 2021;25:150-155.
5. Wardana MP, Dachlan EG, Dekker G. Pulmonary edema in preeclampsia: an Indonesian case–control study. J Matern Fetal Neonatal Med. 2017;30(12):1413-1417.
6. Devi DS. A case of severe preeclampsia presenting as acute pulmonary oedema. Int J Reprod Contracept Obstet Gynecol. 2016 Mar;5(3):899-902.
7. Souabni SA, et al. Preeclampsia complicated with pulmonary edema: a case report. PAMJ Clin Med. 2020;4(103).
8. Mohan M, Baagar KAM, Lindow S. Management of diabetic ketoacidosis in pregnancy. Obstet Gynaecol. 2017;19:55–62.
9. Gómez-Ríos MA, Gómez-Ríos D, Paech MJ, Diéguez-Fernández M. Managing diabetic ketoacidosis in pregnancy. Saudi J Anaesth. 2016;10:238-239.
10. Committee on Clinical Consensus-Obstetrics. Urinary tract infections in pregnant individuals. Am Coll Obstet Gynecol. 2023;142(2):435-445.
11. Yefet E, Bejerano A, Iskander R, Zilberman Kimhi T, Nachum Z. The Association between Gestational Diabetes Mellitus and Infections in Pregnancy—Systematic Review and Meta-Analysis. Microorganisms. 2023;11(8):1956.