


CASE REPORT

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Euglycemic Diabetic Ketoacidosis — A Silent Emergency in Pregnancy: A Case Report

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ABSTRACT

Introduction: Diabetic ketoacidosis (DKA) has been widely recognized as a life-threatening acute complication of diabetes mellitus that may occur in pregnancy, thus endangering the mother and fetus. Euglycemia can obscure DKA, posing a diagnostic challenge, particularly in the emergency unit. **Case:** A 26-year-old woman at 33 weeks of pregnancy presented with irregular uterine contractions. Her initial random blood glucose was 157 mg/dL, with +3 ketonuria, and she had no previous diagnosis of diabetes mellitus. After 24 hours of observation, she experienced premature rupture of membranes followed by tachypnea, Kussmaul breathing, and nausea. Laboratory evaluation showed β-hydroxybutyrate 4.9 mmol/L, pH 7.22, bicarbonate 5.7 mmol/L, and anion gap 27.3 mmol/L, with fasting blood glucose of 123 mg/dL, confirming euglycemic diabetic ketoacidosis (EDKA). She was treated with intravenous fluids, insulin, and dextrose. Emergency cesarean delivery was performed because of progressive maternal deterioration and persistent fetal compromise. A previous random blood glucose of 234 mg/dL and HbA1c of 7.5% suggested previously unrecognized maternal dysglycemia. The mother recovered after intensive care, while the neonate required NICU admission for respiratory distress and hypoglycemia. **Discussion:** Pregnancy-associated metabolic adaptations can predispose susceptible women to EDKA at relatively normal blood glucose levels. The coexistence of unrecognized maternal dysglycemia and acute obstetric stress may accelerate ketoacidosis and compromise both maternal and fetal health. Clinicians should maintain a high index of suspicion for EDKA when ketonemia and high-anion-gap metabolic acidosis are present despite euglycemia. **Conclusion:** EDKA in pregnancy may be overlooked when the initial presentation is predominantly obstetric, and blood glucose is not markedly elevated. The presence of ketonuria and subsequent clinical deterioration prompted evaluation for metabolic derangement despite near-normal glucose levels. Careful interpretation of previous abnormal glucose values and early assessment for ketosis are important for timely recognition and management of EDKA in pregnancy.

Keywords: Case report, diabetes mellitus, euglycemic diabetic ketoacidosis, pregnancy.

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INTRODUCTION

Diabetic ketoacidosis (DKA) is a life-threatening acute complication of diabetes mellitus that can occur during pregnancy. Although DKA is traditionally associated with marked hyperglycemia, pregnancy is a ketosis-prone state in which DKA may develop at relatively low or near-normal blood glucose concentrations. This phenomenon, known as euglycemic diabetic ketoacidosis (EDKA), may be particularly challenging to recognize because the absence of pronounced hyperglycemia can provide false reassurance and delay diagnosis and treatment.[1–3]

Pregnancy-related physiological adaptations contribute to an increased susceptibility to ketosis and DKA. Increased insulin resistance during late pregnancy, increased glucose utilization by the fetoplacental unit, and accelerated starvation may facilitate lipolysis and ketone production. Acute stressors, including reduced caloric intake, infection, vomiting, and obstetric complications, may further precipitate ketoacidosis.[3-4] Therefore, pregnant women with diabetes who develop unexplained nausea, respiratory distress, or metabolic acidosis should be evaluated for DKA regardless of their blood glucose concentration.[2,4]

Early recognition of EDKA is essential because maternal metabolic derangement may adversely affect both maternal and fetal outcomes. Recent evidence indicates that EDKA represents a substantial proportion of DKA episodes occurring during pregnancy, emphasizing that normal or mildly elevated blood glucose should not be used to exclude DKA.[3,5]

We report a case of EDKA in a previously undiagnosed diabetic pregnant woman at 33 weeks of gestation who initially presented with preterm uterine contractions and without marked hyperglycemia. The diagnosis became apparent after the development of ketonemia and severe high-anion-gap metabolic acidosis accompanied by Kussmaul breathing. The case highlights the diagnostic challenge of EDKA in pregnancy

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and the importance of considering metabolic causes of maternal and fetal deterioration even in the absence of severe hyperglycemia.

CASE REPORT

A 26-year-old woman, Gravida 2 Parity 1, at 33 weeks of gestation presented to the emergency department with irregular uterine contractions. She had never been diagnosed with diabetes mellitus or received antidiabetic medication, although a random blood glucose (RBG) level of 234 mg/dL had been documented during a previous pregnancy. On admission, her blood pressure was 127/79 mmHg, heart rate 117 beats/min, respiratory rate 20 breaths/min, and temperature 36.7°C. Fetal heart rate (FHR) was 141 beats/min, and cardiotocography (CTG) was category I. Ultrasonography showed a singleton live fetus in cephalic presentation, with an estimated fetal weight of 2,499 g, amniotic fluid index (AFI) of 11 cm, and cervical length of 1.26 cm. Initial RBG was 157 mg/dL with positive ketonuria (+3). She was managed for preterm uterine contractions and received nifedipine.

After 24 hours of observation, the patient experienced premature rupture of membranes, followed by tachypnea, Kussmaul breathing, and nausea without vomiting. Her heart rate increased to 130 beats/min and respiratory rate to 32 breaths/min, while FHR increased to 170 beats/min. Speculum examination demonstrated pooling of amniotic fluid and a positive nitrazine test, with AFI 3.6 cm and category II CTG.

Laboratory evaluation showed fasting blood glucose of 123 mg/dL, β-hydroxybutyrate 4.9 mmol/L, HbA1c 7.5%, sodium 134 mmol/L, potassium 5.5 mmol/L, and chloride 101 mmol/L. Arterial blood gas analysis showed pH 7.22, pCO_2 13.8 mmHg, HCO_3^- 5.7 mmol/L, and base excess −18.7 mmol/L, with a calculated anion gap of 27.3 mmol/L. These findings were consistent with EDKA.

The patient received intravenous NaCl 0.9%, continuous insulin infusion at 10 IU/hour, and 5% dextrose at 125 mL/hour, with serial maternal blood glucose monitoring. Despite treatment, her heart rate increased to 143 beats/min and respiratory rate to 44 breaths/min. FHR reached 177 beats/min with recurrent non-reassuring CTG. Emergency cesarean delivery was performed because of progressive maternal deterioration and persistent fetal compromise in the setting of premature rupture of membranes and suspected intra-amniotic infection.

A male infant weighing 3,205 g was delivered with Apgar scores of 2/5/7 and a Ballard score corresponding to approximately 34 weeks of gestation. The neonate developed respiratory distress and hypoglycemia (RBG 35 mg/dL), requiring neonatal intensive care and ventilatory support. The mother was subsequently managed in the ICU. Her metabolic acidosis improved, with pH increasing from 7.01 to 7.36, and echocardiography showed preserved left ventricular systolic function with an ejection fraction of 59.8%. She was subsequently transferred to the ward and discharged without further complications.

DISCUSSION

The major diagnostic challenge in this case was the absence of marked hyperglycemia at initial presentation. The patient presented primarily with irregular uterine contractions and had no previous diagnosis of diabetes mellitus or history of antidiabetic treatment. Her initial random blood glucose was 157 mg/dL, although ketonuria (+3) was already present. Therefore, DKA was not initially suspected because the characteristic marked hyperglycemia commonly associated with DKA was absent. After 24 hours of observation, the patient experienced premature rupture of membranes, followed by tachypnea, Kussmaul breathing, and nausea, prompting further metabolic evaluation. At that time, fasting blood glucose remained relatively low at 123 mg/dL. At the same time, β-hydroxybutyrate was elevated at 4.9 mmol/L, with severe metabolic acidosis (pH 7.22, HCO_3 5.7 mmol/L), an anion gap of 27.3 mmol/L, and a BE of −18.7 mmol/L. These findings established the diagnosis of EDKA. Recent studies have reported that euglycemic presentations account for a substantial proportion of DKA episodes in pregnancy, including 17.6% in a recent clinical series and 29.7% in a systematic review.[3,6] The absence of marked hyperglycemia should therefore not be used to exclude DKA in pregnancy.

Pregnancy itself increases susceptibility to ketosis through increased insulin resistance, enhanced lipolysis, and greater glucose utilization by the fetus, particularly during the second and third trimesters.[1,2,4] These physiological adaptations can result in accelerated ketogenesis, allowing ketoacidosis to develop at relatively low blood glucose concentrations. Acute stressors, including infection, reduced caloric intake, vomiting, and other pregnancy-related stressors, may further accelerate ketone production.[1,2,4] In this case, the presence of ketonuria at admission suggests that ketogenesis had already begun before overt clinical deterioration.

The available findings also suggest previously unrecognized maternal dysglycemia. The patient had a previous random blood glucose level of 234 mg/dL and an HbA1c of 7.5%. According to the 2026 American Diabetes Association Standards of Care, an HbA1c level of ≥ 6.5% is within the diagnostic range for diabetes.[7] Together, these findings suggest significant maternal dysglycemia that had not previously been recognized, although the exact type and duration of diabetes could not be established. Importantly, the diagnosis of EDKA in this case was based on significant ketonemia and high-anion-gap metabolic acidosis despite relatively low blood glucose, rather than on hyperglycemia alone.[1,2]

Premature rupture of membranes was an important event in the clinical course. After 24 hours of observation, membrane rupture was followed by worsening tachypnea, Kussmaul breathing, maternal tachycardia, and fetal heart rate abnormalities. A direct causal relationship between membrane rupture and EDKA cannot be established; however, acute obstetric stress associated with membrane rupture may have accelerated ketosis. Conversely, underlying maternal dysglycemia may itself be associated with an increased risk of premature rupture of membranes. A 2024 systematic review and meta-analysis identified gestational diabetes as a significant risk factor for premature rupture of membranes, while a 2025 study similarly reported an association between gestational diabetes and membrane rupture.[8,9] The clinical course in this case may therefore reflect an interaction between previously unrecognized dysglycemia, the increased




ketogenic tendency of late pregnancy, and an acute obstetric stressor.

The infant weighed 3,205 g and had a Ballard assessment corresponding to approximately 34 weeks of gestation. This birth weight is markedly high for a preterm infant when interpreted using gestational-age- and sex-specific standards. This is consistent with large-for-gestational-age status.[10] Maternal glycemic exposure is associated with fetal overgrowth, and higher maternal HbA1c levels are associated with an increased risk of large-for-gestational-age infants.[11] Pregestational diabetes is also associated with adverse perinatal outcomes, including fetal overgrowth.[12] In this case, the combination of a previous random blood glucose of 234 mg/dL, HbA1c of 7.5%, and a markedly high birth weight provides additional clinical evidence suggesting previously unrecognized maternal dysglycemia. However, birth weight alone cannot establish a diagnosis of diabetes.

Management of EDKA during pregnancy requires correction of volume depletion, insulin deficiency, electrolyte abnormalities, and the underlying precipitating factor while avoiding hypoglycemia.[1,2] In this case, treatment consisted of intravenous 0.9% sodium chloride, insulin at 10 IU/hour, 5% dextrose at 125 mL/hour, and serial maternal glucose monitoring. Dextrose administration allowed insulin therapy to continue despite the absence of marked hyperglycemia. Despite treatment, maternal tachycardia and tachypnea progressed, while the fetal heart rate increased to 177 beats/min with recurrent non-reassuring CTG findings. Emergency cesarean delivery was subsequently performed because of progressive maternal deterioration and persistent fetal compromise in the setting of membrane rupture. Thus, the delivery decision was based on the overall maternal and fetal clinical condition rather than EDKA alone.[2,13]

The neonate had a random blood glucose level of 35 mg/dL and required NICU admission and ventilatory support. Serial maternal glucose monitoring was performed, and no maternal hypoglycemia was documented; therefore, neonatal hypoglycemia cannot be attributed solely to maternal insulin administration. Rather, it was likely associated with underlying maternal dysglycemia, potentially compounded by prematurity and perinatal metabolic stress.[12] The neonate was discharged on day 6 following NICU care. Meanwhile, the mother's metabolic status improved, with pH increasing from 7.01 to 7.36 and preserved left ventricular systolic function (ejection fraction 59.8%). She was discharged after three days without further complications. Nevertheless, the potential long-term consequences of maternal ketonemia warrant attention. Chronic maternal ketonemia has been associated with impaired fetal neurodevelopment, and a systematic review and meta-analysis found that higher maternal ketone concentrations were significantly associated with lower mental development index scores, particularly at two years of age.[14,15] These findings highlight the importance of early recognition and prompt multidisciplinary management of maternal EDKA to optimize maternal and neonatal outcomes.

One important limitation of this case was the absence of comprehensive antenatal glycemic records, which limited earlier recognition of maternal dysglycemia. This highlights the importance of routinely measuring, documenting, and appropriately interpreting maternal blood glucose during antenatal care, particularly when previous abnormal values are identified. Clinicians also need greater awareness of EDKA, and further studies may help improve its recognition and management. In pregnant women with ketonuria, unexplained metabolic deterioration, or compatible symptoms, EDKA should be considered even in the absence of marked hyperglycemia.

CONCLUSION

Euglycemic diabetic ketoacidosis is a rare but potentially life-threatening complication of pregnancy that may occur without marked hyperglycemia. This case highlights the diagnostic challenge of EDKA when the initial presentation is predominantly obstetric. Careful measurement, documentation, and interpretation of maternal blood glucose during antenatal care are essential, particularly when previous abnormal values are identified. Early recognition and prompt management of EDKA are crucial to optimize maternal and fetal outcomes.

DECLARATIONS

Consent for Publication

The patient provided written informed consent for publication of this case report and its accompanying clinical details. All personal information has been anonymized to protect the patient's privacy.

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Author Contributions

R.H. contributed to the conception and design of the case report, data collection, literature review, and manuscript drafting. W.W. and R.I. contributed to the clinical interpretation, critical revision, and supervision of the manuscript. All authors reviewed and approved the final version of the manuscript.

Conflict of Interest

The authors declare no financial or non-financial conflicts of interest related to this manuscript.

Declaration on the Use of Artificial Intelligence (AI)

The authors used ChatGPT (OpenAI) solely to assist with language editing and improving the clarity and readability of the manuscript. The authors reviewed, verified, and approved all content and take full responsibility for the final manuscript.

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