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Safeguarding mother and baby: Shifting focus on DKA management in preterm delivery

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Abstract

Diabetic Ketoacidosis (DKA) is a severe complication that can arise in type I diabetic patients, and is especially fatal in those who are pregnant. Complications of DKA impact both the mother and the fetus, resulting from insulin deficiency and improper insulin management.

Timely glucose measurements and insulin compliance are essential in preventing hyperglycemia. This case report will focus on a 28-year-old female with type I diabetes who, at 32 week's gestation, presented with nausea, vomiting, abdominal pain, irregular contractions, and non-reasoning fetal heart tones. Our paper analyzes the diagnosis and step-by-step management of DKA in pregnancy and delivery. Given the difficulty in diagnosing and treating DKA in pregnant patients with signs of preterm delivery, this paper will analyze the current data and various steps in the management and outcomes that may arise. Prioritizing the treatment of DKA before addressing obstetric complications can positively alter maternal and fetal outcomes.

Keywords: Diabetic ketoacidosis; Insulin management; Preterm delivery; Case report.

Introduction

Diabetes mellitus is a group of endocrine disorders that results in hyperglycemia. It is associated with end-organ dysfunction, including the brain, retina, kidney, nerves, heart, and blood vessels [1]. There are multiple variations of diabetes that include Type 1 Diabetes Mellitus (T1DM), Type 2 Diabetes Mellitus (T2DM), gestational diabetes, and prediabetes, amongst others. T1DM is an autoimmune variant that results in a total lack of insulin production. Although it is not fully understood, the pathophysiology involves an autoimmune attack by CD8+ T-cells of the pancreatic beta cells that results in the destruction of insulin, leading to absent uptake of glucose into various tissues [1]. In 2022, approximately 8.75 million people

with diabetes were living with the condition worldwide, with 1.52 million under 20 years of age [2]. For years, research has shown that T1DM has genetic tracings. However, environmental factors, such as a previous viral infection, can also trigger an autoimmune reaction, leading to T1DM. Clinical symptoms include polydipsia, polyuria, polyphagia, weight loss, and thirst, amongst others. The American Diabetes Association (ADA) uses the following criteria for diagnosis: A random plasma glucose level of 200 mg/dL or higher, along with classic symptoms of hyperglycemia, or ≥2 abnormal test results of hyperglycemia [2]. Other tests for hyperglycemia can include fasting blood glucose of 126 mg/dL or higher, a 2-hour oral glucose tolerance test of ≥200 mg/dL, or an HbA1c of 6.5% or higher.

T2DM accounts for over 90% of DM cases [3]. The pathophysiology is related to defective insulin secretion by pancreatic β-cells and the inability of insulin-sensitive tissues to respond to insulin [4]. There is a strong genetic predisposition, as well as environmental causes such as obesity. T2DM is most often diagnosed in ages >40, but may also present earlier in life. While T2DM is more prevalent than T1DM in pregnancy, T1DM may also produce fatal complications. This includes congenital birth defects, such as caudal regression syndrome. Additionally, neonatal hypoglycemia may arise from persistently elevated insulin levels in mothers that can cross the placenta and harm the fetus [5,6]. While these adverse effects from T1DM have been heavily studied, what has often been overlooked is the impact of Diabetic Ketoacidosis (DKA) and the implications it can have on morbidity and mortality of both mother and fetus.

Diabetic ketoacidosis often results from poorly controlled T1DM, whereby lack of insulin intake leads to unregulated and persistent fatty acid oxidation in the liver into acetyl-CoA. This is then used as a substrate to produce ketone bodies, such as beta-hydroxybutyrate and acetoacetate [7,8]. This can have a variety of effects, including flu-like symptoms of fever, nausea, vomiting, dehydration, acetone breath, and altered mental status and coma in severe cases [8]. Labs will show a low pH from increased acid production and decreased bicarbonate production, and patients will compensate by hyperventilating to release CO_2 and return pH to normal. Oftentimes, glucose levels are above 250 mg/dL [8]. Treatment includes prompt IV 0.9% normal saline infusion to flush out acid, followed by the addition of dextrose 5% when blood glucose levels reach around 200 mg/dL. This is then followed by IV regular insulin infusion to restore blood glucose levels, with careful watch of potassium levels to prevent hypokalemia. Subcutaneous insulin can then be given once blood glucose levels are less than 200 mg/dL and the anion gap is cleared [8,9]. Careful care must be taken not to reduce the glucose levels too rapidly, as this can perpetuate cerebral edema [10]. The key metabolic goals of insulin therapy include reducing blood ketones by 0.5 mmol/L per hour, increasing venous bicarbonate by 3 mmol/L per hour, and lowering capillary glucose by 3 mmol/L per hour and regular blood draws are essential to monitor the patient's progress and adjust interventions as necessary [11].

When DKA is suspected in a pregnant patient, laboratory investigations are necessary to confirm the diagnosis. Key laboratory findings indicative of DKA include the presence of serum and urine ketones, elevated blood glucose (hyperglycemia), low serum bicarbonate levels, arterial pH <7.30, an anion gap >12, and an elevated base deficit >4. Potassium levels may also be elevated (>5) in some cases.

DKA in pregnancy must be prevented and managed precipitously due to risks to the mother and fetus. De Guisto et al. performed a retrospective cohort study and reported an overall incidence of 6.3 per 100,000 pregnancies in the UK for diabetic ketoacidosis [12]. The majority of the DKA episodes occurred in the third trimester, with common precipitating factors including infection, as well as steroid use for preterm birth, and medication noncompliance [12]. The study reported no maternal deaths, which included 25% of patients being admitted to the ICU without cases of cerebral edema or aspiration pneumonia. Unfortunately, neonatal mortality was observed in 16% of cases, including 11 stillbirths and 1 neonatal death [12]. It is noteworthy to mention that a higher prevalence of DKA was seen in patients with microvascular complications and mental health conditions [12]. Key preventative measures for DKA in pregnancy, most notably tight glycemic control, self-monitoring of glucose 6-10 times per day, maintaining proper hydration, and continuing insulin use during illnesses and hyperemesis. Addressing mental health conditions and socio-economic barriers that increase the risk for DKA was also found to be essential in preventing further complications [12].

In addition, Coetzee et. al performed a prospective cohort study following 54 episodes of DKA amongst 47 women. The average age at delivery was 34 weeks' gestation, with 85% of deliveries being preterm and 55% coinciding with a DKA admission [13]. Mothers with hypertensive disorders had an increased risk of fetal losses, with 31% of cases occurring in women with pregestational hypertension and two additional cases in women with gestational hypertension that progressed to preeclampsia [13]. High rates of concurrent obesity, as well as suboptimal glycemic control, contribute to a higher number of intrauterine deaths [14]. Healthcare providers should undergo structured diabetes education to be able to improve patient outcomes.

Pregnant patients who are noncompliant with routine glucose monitoring may delay the diagnosis of hyperglycemia, raising the risk of developing DKA and intrauterine fetal loss [14] DKA should be identified through a combination of clinical symptoms and laboratory findings [14]. Cases of DKA may be classified as mild, referring to blood glucose levels that may not be as exaggerated as expected, which may lead to a delayed diagnosis and the development of complications [15]. This puts patients at risk for severe complications, including sepsis, infection, altered consciousness, and coma [16,17]. Additionally, careful evaluation must be placed on electrolyte imbalance as arrhythmias and cardiac arrest may arise from both hyperkalemia or hypokalemia [18].

Recovery from DKA in pregnancy is defined by the following criteria: blood ketone levels less than 0.6 mmol/L, a pH greater than 7.3, and a serum bicarbonate level greater than 15 mmol/L. Furthermore, the correction of the anion gap provides additional confirmation that DKA has resolved [11].

Case Report

The patient, a 28-year-old female at 32 weeks' gestation, presented to labor and delivery around 10:00 PM with abdominal pain, weakness, nausea/vomiting, and irregular contractions. She had mentioned that she had type I diabetes since the age of 14, controlled with injectable Lantus 22 units once per day, as well as Novolog 20 units three times per day before meals. However, during her pregnancy, she was using

Neutral Protamine Hagedorn (NPH) insulin twice per day and was compliant. She had been prescribed a Humulin Kwikpen to inject her insulin subcutaneously at 100 units/mL before meals, but had missed her most recent dose earlier in the day. Due to signs of neonatal distress with non-reassuring fetal heart tones, the patient was rushed into delivery and underwent a repeat transverse cesarean section (C-section) with the use of spinal anesthesia with an estimated blood loss of 800 mL. Urine output was adequate and clear.

However, the patient had experienced persistent nausea, abdominal pain, and hyperventilation. Blood glucose measurements were taken several hours after delivery and showed a blood glucose of 458 mg/dL. Other notable lab values obtained included a white blood cell count of 25,400/mm^3, up from 9,000/mm^3 prior to delivery, a bicarbonate (HCO3-) level of 10 mmol/L, and evidence of glucosuria (>1,000 mg/dL) and ketonuria (>150 mg/dL) on urinalysis. The patient was then transferred to the Intensive Care Unit (ICU) and started on IV 0.9% normal saline and IV regular insulin drip for close monitoring of Diabetic Ketoacidosis (DKA) management.

Her laboratory investigations showed a serum glucose of 458 mg/dL (normal value 70-100 mg/dL), glycosylated hemoglobin of 7.4% (normal value: 4-6.5%), anion gap level of 14 mEq/L (normal value 3-10 mEq/L), partial pressure of carbon dioxide of 30 mmHg (normal value: 48-32 mEq/L), serum carbon dioxide level of 10.0 mEq/L (normal value: 22-28 mEq/L), white blood cell count of 25,400 cells/mm3 (normal value: 4,500-11,000 cells/mm3), serum sodium level of 128 mEq/L (normal value: 136-145 mEq/L), serum chlorine level of 108 mEq/L (normal value 95-105 mEq/L), serum potassium level of 2.8 mEq/L (normal value 3.5-5.0 mEq/L), and serum urine ketone level of >150 mg/dL (normal value 20-30 mg/dL).

One major cause of complications in this patient's management was the delay in obtaining blood glucose levels immediately when brought to the labor and delivery floor. At the time, the focus of care was primarily placed on the evidence of fetal distress and evidence of preterm delivery complications, which further led to elevated blood glucose levels over time.

Discussion

Delayed diagnosis of DKA in obstetrical paper

Diabetic Ketoacidosis (DKA) is a severe and potentially life-threatening complication during pregnancy, requiring prompt identification and management to ensure the safety of both the mother and the fetus. Several factors may precipitate DKA in pregnant women, including hyperemesis gravidarum, gastroparesis, infections, insulin noncompliance, and insulin pump failure [19]. Effective management of DKA is critical to prevent adverse outcomes. Given the complexity of the condition, it is essential that the treatment involves an interdisciplinary team, which typically includes an obstetrician, endocrinologist, critical care physicians, and nurses or midwives.

Management of DKA in pregnancy is guided by six critical parameters: Intravenous (IV) fluid management, IV insulin therapy, electrolyte management, evaluation of the need for bicarbonate administration,

identification and treatment of precipitating factors, and monitoring maternal and fetal responses to treatment [11].

Close fetal monitoring is essential during maternal treatment to ensure fetal well-being as fetal mortality rates rise significantly following a DKA episode [20]. In an acute setting, DKA during pregnancy can lead to a metabolic imbalance that can disrupt fetal oxygenation and nutrient supply [21]. The use of Non-Stress Tests (NST) or Biophysical Profiles (BPP) is recommended as part of this monitoring. NSTs assess fetal heart rate patterns and can identify signs of fetal distress, while BPPs provide a more comprehensive evaluation by including measurements of fetal movement, fetal tone, amniotic fluid volume, and fetal breathing movements. These tests offer valuable insight into the fetus's condition and can guide clinicians in making timely decisions to optimize both maternal and fetal outcomes. Frequent monitoring can help detect early signs of fetal distress, ensuring that interventions, such as early delivery or additional support measures, can be implemented if necessary. Early delivery is not required if no significant fetal distress is present and the mother's condition has stabilized [21].

Delays in assessment of blood glucose levels has been an expanding issue in complications of patients with DKA [20]. Studies have shown that patients presenting with constitutional and gastrointestinal symptoms, including fatigue, nausea, and vomiting, were more likely to get a full DKA workup than those presenting with obstetric symptoms [21]. This is similar to our case report, as the focus of the patient's workup was more so focused on the obstetric complications of irregular contractions and non-reassuring fetal heart tones rather than on the symptoms, she presented with related to DKA. Protocols should be implemented for any patient presenting with hyperglycemic symptoms, as such patients could be at risk for further complications before, during, and after delivery [22]. This includes volume repletion and balancing abnormal electrolytes such as sodium, potassium, and bicarbonate, and ensuring insulin repletion as needed.

Failure to promptly recognize and treat Diabetic Ketoacidosis (DKA) can significantly exacerbate both maternal and fetal complications. If aggressive treatment successfully achieves target blood glucose levels in the mother without causing evidence of fetal distress, delivery can often be delayed to allow for further stabilization [23]. In our case, the patient underwent a cesarean delivery prior to stabilization of her glucose levels. Proceeding with surgery before achieving glucose control may increase the risk of maternal adverse outcomes, including poor anesthesia management, delayed recovery, higher rates of infection, and impaired wound healing [21]. Furthermore, emergent cesarean delivery during active DKA has been shown to be detrimental to the fetus as well, presenting a higher risk of complications compared to baseline risks associated with women who are hyperglycemic. Despite this, ensuring the safety of the fetus was prioritized, and delivery was performed due to signs of fetal distress. In cases where delivery can be safely delayed, it is recommended to first address maternal blood glucose levels, hydration status, and electrolyte imbalances. Continuous fetal monitoring is essential to detect signs of fetal hypoxia, as improvements in maternal acid-base balance are often mirrored by an improvement in fetal condition [24].

While it is imperative to closely monitor the fetal distress that may present upon preterm delivery in

pregnant patients, it is essential to immediately obtain blood glucose levels in said patient population with type I diabetes. While the patient had stated that she had been carefully monitoring her insulin regimen and blood glucose levels, missing even a single dose can have drastic effects on the body, impacting both the mother and the baby.

Preventive strategies to reduce the risks of DKA during pregnancy are critical for both maternal and fetal health. Education plays a foundational role in preventing DKA episodes. Patients should receive thorough counseling on the importance of blood glucose management throughout pregnancy. This includes not only regular self-monitoring of blood glucose levels but also understanding the signs and symptoms of hyperglycemia and ketosis, which can precede DKA [25].

Further, adherence to prescribed insulin regimens is paramount, as maintaining optimal blood glucose control can prevent the development of DKA. Given the hormonal changes that occur during pregnancy, the insulin requirements for pregnant women with diabetes may change, and adjustments to insulin doses may be necessary. Close collaboration with a diabetes care team, including endocrinologists and obstetricians, is essential for tailoring insulin therapy to each patient's individual needs.

Additionally, frequent prenatal visits should be scheduled to monitor the progression of the pregnancy, detect any early signs of complications, and ensure that the mother's blood glucose levels remain within target range. Regular ultrasounds can be performed to monitor fetal growth, while close attention to the maternal history, such as previous episodes of DKA or poor glucose control, can inform more proactive management strategies [23].

By incorporating these preventive measures, the risk of DKA can be reduced, promoting improved outcomes for both the mother and the baby. A proactive approach that combines patient education, rigorous monitoring, and close management of maternal diabetes can significantly decrease the likelihood of complications and improve both maternal and neonatal health.

Conclusion

Diabetic ketoacidosis is a severe complication in type I diabetics that is especially fatal in pregnant patients and their fetuses that can result in hyperglycemia, nausea/vomiting, altered mental status, coma, and possibly maternal and fetal death. Ensuring immediate management of hyperglycemia and acidosis and restoring electrolyte and volume balance in such patients further impacts the function of other vital organs. This case report shows how delays in such care endanger patients' livelihood and how clinicians' initial steps in management should focus first on obtaining proper glucose measurements and correcting electrolyte imbalances even in the face of irregular contractions, non-reassuring fetal heart tones, and other symptoms related to preterm labor.

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