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Anesthetic Management of A Parturient With Diabetic Keto-Acidosis

Case Report

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Abstract

Diabetic ketoacidosis (DKA) in pregnancy is a life threatening medical emergency. It can compromise both the fetus and the mother profoundly. The incidence of DKA during pregnancy ranges between 2 to 3%, and carries a 10–20% risk of fetal death.1 DKA is characterized by a biochemical triad of ketonemia, hyperglycemia and acidemia. We present a case of a parturient with mismanaged DKA that was brought to the operating room for a STAT cesarean section (C/S) due to fetal distress and discuss the anesthetic implications.

Key Words: Diabetic Keto-Acidosis; Pregnancy; Anesthesia.

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Introduction

DKA in pregnancy is of special concern. It tends to occur at lower plasma glucose levels and more rapidly than in non-pregnant patients and usually occurs in the second and third trimesters. The marked increase in insulin resistance and enhanced lipolysis/ketosis associated with pregnancy account for the greater risk of DKA during gestation. Although DKA is usually seen in patients with type 1 diabetes mellitus (DM), it can also occur in women with type 2 diabetes as well as gestational diabetes. Predisposing factors include infection, vomiting, dehydration, starvation, stress, omission of insulin doses, insulin pump failure and the obstetric use of β-sympathomimetic drugs for tocolysis, and glucocorticoids used for fetal lung maturity [2,3].

Classical presentations include anorexia, nausea, vomiting, polyuria, polydipsia, tachycardia and abdominal pain or muscle cramps. If severe, signs of volume depletion (e.g., hypotension and oliguria), lethargy to coma, normal-to-cold body temperature, Kussmaul hyperventilation and a fruity odor may be noticeable in the patient's breath. Diabetic parturients can develop ketoacidosis with remarkably low blood glucose values (as low as 200 mg/dL) [4,5]. The presence of ketones, maternal arterial pH of less than 7.30, decreased serum bicarbonate level and elevated anion gap confirm the diagnosis.

Case

A 22 yo G3P1 at 29 weeks gestation with a history of Type I DM with no evidence of diabetic microvascular or macrovascular complications, poorly controlled on insulin/non-compliant with medications (HbA1C of 9.9) and prior C/S was admitted via the emergency room, tachycardic to the 150's and tachypneic with a respiratory rate in the 30's. She complained of vomiting with lower abdominal pain for 1 day and being unable to eat.

A work up was begun and she was noted to have a serum blood glucose of 278mg/dL, pH 7.05, HCO_3^- 2mmol/L, 4+ ketones in urine and a WBC count of 16K/uL. A diagnosis of DKA was made and the admissions orders were entered for an ICU bed. It was at this time that a hyperglycemia order set was chosen instead of a DKA admission protocol. The patient was started on a subcutaneous insulin sliding scale and put on maintenance IV fluids.

When an ICU bed was available, she was transferred to the intensive care unit where she was continued on the admitting hyperglycemia protocol. The patient had also begun having regular painful uterine contractions. The patient was also becoming slightly confused. Because fetal heart tones were non-reassuring (Category 3 with FHTs in the 80s) the admitting team and the obstetric team decided she required a STAT C/S. It was only at this time when the mother and the fetus were decompensating that the admitting team realized she was not being treated for DKA, rather for hyperglycemia. It was decided that delaying surgery to correct the DKA would only worsen the outcome for the fetus, so she was taken to the OR for a stat C/S under general anesthesia.

The patient was intubated uneventfully with a rapid sequence intubation and general anesthesia was administered. The surgery proceeded swiftly and uneventfully. The baby was delivered and intubated at birth with APGAR's of 1/2/4 and transferred to the

neonatal intensive care unit. Despite lack of complications during the surgery the maternal ABG pH was 7 and glucose was 388.

Intraoperatively, a DKA treatment protocol was initiated. An insulin bolus followed by a drip was started to correct the hyperglycemia, aggressive fluid resuscitation was used to correct the hypovolemia, acidemia was treated with bicarbonate, and electrolyte repletion for potassium deficit once the patient was making urine.

The patient was transferred back to ICU intubated, with DKA treatment protocol in progress. In the ICU, the patient was started on broad-spectrum antibiotics and as the anion gap closed she was switched to subcutaneous NPH and regular insulin and extubated. She was subsequently discharged home with appropriate endocrine follow-up.

Discussion

Pathophysiology of DKA

The major metabolic changes in carbohydrate, lipid and amino acid metabolism associated with pregnancy have been described as a combination of 'insulin deficiency', 'insulin resistance' and 'starvation'. Insulin sensitivity has been demonstrated to fall by as much as 56% through 36 weeks of gestation, causing a relative insulin deficiency.[6] In addition, the action of counter-regulatory hormones such as glucagon, cortisol, growth hormone, epinephrine, human placental lactogen and prolactin result in insulin resistance, resulting in hyperglycemia and subsequently, glycosuria. Insulin requirements progressively rise during pregnancy because of insulin resistance and 'deficiency'; this explains the higher incidence of diabetic ketoacidosis in the second and third trimesters. To compound the problem, the physiological rise in progesterone with pregnancy decreases gastrointestinal motility, which increases the absorption of carbohydrates and promotes hyperglycemia.

Pregnancy is a relative state of starvation, especially in the second and third trimesters. The fetus and the placenta use large amounts of maternal glucose as a major source of energy, leading to decreased maternal fasting glucose. This, associated with relative insulin deficiency, causes the body to change from metabolism based on carbohydrate, to fat oxidation and muscle catabolism. Serum levels of glycerol and free fatty acids (FFAs) rise because of unrestrained lipolysis. Glycerol and alanine provide substrates for hepatic gluconeogenesis, which is stimulated by the excess of glucagon that accompanies insulin deficiency. Glucagon also stimulates mitochondrial conversion of FFAs into ketones. The major ketoacids produced, acetoacetic acid and β-hydroxybutyric acid, are strong organic acids that create metabolic acidosis. This is further aggravated by lactic acidosis caused by dehydration & poor tissue perfusion [7].

Glycosuria causes osmotic diuresis, leading to water and potassium loss. Accumulation of ketone bodies contributes to abdominal pain and vomiting. Vomiting in combination with increased insensible water losses due to tachypnea will further worsen the state of dehydration. Dehydration can lead to decreased kidney perfusion and acute renal failure. If dehydration continues unhindered, there is a progression to decreased cardiac output, hypotension, shock, and death.

Because of acidosis, the cellular H+/K+ antiporter exchanges intracellular potassium for plasma protons. K+ ions enter the circulation, initially leading to hyperkalemia; this is aggravated by dehydration and renal failure. The initial massive diuresis of DKA leads to potassium wasting. Depending on the duration of DKA, serum K+ at diagnosis may be high, normal or low, but the intracellular K stores are always depleted. Increasing acidosis often leads to acidotic breathing and acetone smell in the breath and eventually causes impaired consciousness and coma [8,9].

In addition to a thorough history and physical exam, investigations used to help confirm the diagnosis and follow therapy of DKA include blood glucose levels, arterial blood gases, plasma osmolality, electrolytes, anion gap, complete blood count (CBC), BUN and creatinine, and urine and blood ketones.

DKA should be treated in a high dependency or intensive care unit under combined medical and obstetric care. All pregnant patients with a live fetus >24 weeks should be monitored continuously for fetal heart rate. Some degree of fetal distress may be present as placental transfer of ketoacids may cause the fetus to become acidotic. However, fetal distress improves with stabilization of maternal hyperglycemia and acidosis, thus immediate fetus delivery is reserved for persistent fetal compromise after maternal resuscitation [10]. Delivery of a compromised fetus should be undertaken ONLY after the mother is metabolically stable.

The goals of therapy include aggressive re-hydration. Correction of acidemia & hyperglycemia via Insulin administration. Restoration of electrolyte homeostasis and elimination of the underlying causes while monitoring for complications of treatment. About 1–1.5 L of isotonic saline (0.9% NaCl) is given during the 1st hour. Subsequent choice for fluid replacement depends on the state of hydration, serum electrolyte levels, and urine output. Potassium supplementation is usually added when urine output is adequate. Continuous infusion of regular insulin therapy is necessary to reduce ketogenesis. Regular monitoring of serum electrolytes, glucose, urea, creatinine, and venous pH should be performed to assess the efficacy of treatment. In general, acidosis is not treated with buffers. As insulin and glucose levels improve, the ketoacidosis is corrected, and the lactic acidosis resulting from poor perfusion responds to intravascular fluid replacement. If the pH approaches 7.00 and the bicarbonate level is less than 10 mEq/L and hypotension fails to respond to intravascular fluid administration, sodium bicarbonate therapy may be required. Phosphate replacement is only indicated in specific situations and thus should be individualized [11,12].

Obstetric Implications of Diabetes Mellitus

Maternal

There is an increased risk for infections such as pyelonephritis and vaginitis in pregnancies complicated by DM. Obesity and chronic hypertension are common co-morbidities and both are risk factors for development of preeclampsia. Cesarean delivery rates are increased in women with DM due to associated comorbidities.

Fetal

Babies born to women with DM are at risk for stillbirths, macrosomia and congenital malformations. Vaginal delivery of a macrosomic baby carries the risk of shoulder dystocia when the fetal shoulder gets impacted on the maternal pelvis, causing obstructed labor. It is suggested that maternal hyperglycemia causes fetal hyperglycemia and hyperinsulinemia which in turn leads to macrosomia. A difficult vaginal birth of a macrosomic neonate

puts the mother at risk of having perineal lacerations.

Poor glycemic control during embryogenesis is associated with fetal congenital anomalies. The incidence of major anomalies is 5 times higher in women with pregestational diabetes as compared to nondiabetics. Common malformations seen are neural tube defects, renal anomalies, caudal regression, cardiac septal and abdominal ventral wall defects. Neonatal hypoglycemia and hyperbilirubinemia are other morbidities seen in these babies.

DKA during pregnancy results in reduced oxygenation of the feto-placental unit due to reduced uterine blood flow and a left shift in the hemoglobin dissociation curve (increased affinity of maternal hemoglobin for oxygen). During DKA, fetal distress is frequently observed, but intervention for fetal compromise should be delayed until the mother is properly resuscitated [13,14].

Anesthesia considerations

Labor epidural analgesia for vaginal delivery effectively controls pain and stress response which predisposes to hyperglycemia. In long standing DM, presence of autonomic neuropathy and potential for exaggerated hypotension following sympathectomy should be kept in mind. Frequent blood pressure monitoring and vigorous intravenous hydration may be indicated in these patients during neuraxial anesthesia. A non-dextrose containing balanced salt solution should be used for volume expansion to prevent peripartum maternal hyperglycemia.

Both spinal and epidural anesthesia are suitable for cesarean section and any resulting hypotension should be aggressively treated to prevent neonatal acidosis. In patients with long standing type I diabetes, glycosylation of tissue proteins leads to stiff joint syndrome. When this affects the atlanto-occipital joint, limited mobility can lead to difficult laryngoscopy and intubation [15].

Conclusion

With increasing practice of antepartum diabetes screening and the availability of early and frequent prenatal care/surveillance, the incidence and outcomes of diabetic ketoacidosis in pregnancy have vastly improved. However, it still remains a major clinical problem in pregnancy since it tends to occur at lower blood glucose levels and more rapidly than in non-pregnant patients, often causing delay in diagnosis.

Pregnancy profoundly affects the management of diabetes. Placental hormones, growth factors, and cytokines cause a progressive increase in insulin resistance, necessitating intensive medical nutrition therapy and frequently adjusted insulin administration to prevent hyperglycemia dangerous to the fetus. Insulin resistance enhances the risk of ketoacidosis in response to the stress of concurrent illnesses or drugs used in the management of obstetrical complications.

- All women with preexisting diabetes who are planning pregnancy or already pregnant should be educated about DKA.
- Providers should have a high index of suspicion for DKA in diabetic pregnant women with nausea, vomiting, abdominal pain, fever, and poor oral intake.
- Protocols for management of DKA during pregnancy include correction of volume depletion, insulin infusion, monitoring and correcting electrolyte imbalances, identifying and treating precipitating factors, and continuous fetal monitoring.
- Initial DKA care is best given in intensive or special care units with experience in monitoring of high-risk pregnancies.
- Immediate delivery may not be necessary for ominous patterns, since correction of DKA often reverts the patterns to normal.

In this case, an error in the admission order set began a series of events that allowed the patient's physiological state to decompensate as the DKA worsened after admission. The severe fetal distress suggested a need to proceed to emergency C/S, but this was a deviation from the course of action suggested in literature. Increased familiarity with the pathophysiology of DKA in pregnancy should help us understand that fetal distress due to ketoacidosis can be improved by treating DKA. It is important for medical, obstetric, and anesthesia providers to have an understanding of how to treat both DKA and the fetal distress associated with it.

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