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CASE REPORT

Case Report: Diabetic Ketoacidosis During Pregnancy Due to Insulin Omission

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Abstract: Diabetic ketoacidosis is an acute and severe complication commonly occurring in individuals with type-1 diabetes mellitus due to absolute insulin deficiency. A 28-year-old Black woman, gravida 2, para 2, secondary school teacher was admitted at 31 weeks of gestation to the obstetric ward on August 12/2022 with a two-day history of nausea and vomiting. She had a history of insulin-dependent diabetes mellitus four years earlier. She missed her insulin dose one day due to traveling to the village for greeting her family. She presented with a two-day history of nausea and vomiting, and a one-day history of shortness of breath, abdominal tenderness, hypotension, elevated heart rate, increased respiratory rate, frequent urination, and fatigue. Ketone testing done using her urine sample showed ketonuria of 3^{+}. Her chest X-ray revealed coarse crackles on auscultation. Her breath odor revealed acetone-smelling breathing. Upon admission, she was treated with insulin infusion for 24 hours and 60 milliequivalents per liter of potassium chloride intravenously was also initiated. On the same day, 0.9% of normal saline 500 mL was initiated intravenously stat and repeats until systolic blood pressure was greater than 90 mmHg. The main objectives of diabetic ketoacidosis management are to restore volume status, normalize hyperglycemia, replace electrolytes lost, and lower ketoacidosis.

Keywords: diabetic ketoacidosis, insulin omission, pregnancy

Introduction

Diabetic ketoacidosis is a life-threatening condition caused by either absolute insulin deficiency, commonly in type-1 diabetes mellitus and complete insulin sensitivity, or occasionally in type-2 diabetes mellitus.[1] Diabetic ketoacidosis in pregnancy is an emergency that significantly causes maternal and fetal morbidity and mortality.[2] Physiological changes that occur during pregnancy elevate the risk of ketosis and subsequent acidosis.[3] Pregnancy is a diabetogenic state with increased insulin resistance, enhanced lipolysis, elevated free fatty acids and increased ketogenesis.[1] There are three pivotal pathophysiology of hyperglycemia in diabetic ketoacidosis: elevation of gluconeogenesis, the elevation of glycogenolysis, and lower peripheral glucose uptake due to a decrease in insulin action in the receptors or a decrease in insulin levels.[4] The goal of diabetic ketoacidosis management in pregnancy comprises restoring circulation, reversing progressive acidosis; reversing proteolysis and lipolysis, normalizing blood glucose concentration, and replacing electrolyte losses.[5,6] For treatment of diabetic ketoacidosis in pregnancy, insulin is frequently given via intravenous route, starting with a bolus of regular insulin at a dose of 0.1 unit/kg body weight, and then, within five minutes followed by a continuous infusion of regular insulin of 0.1 unit/kg/hour.[7–9] Normal saline (0.9% NaCl) is recommended as the initial intravenous fluid replacement in diabetic ketoacidosis occurred during pregnancy.[10] This case report demonstrates diabetic ketoacidosis occurred during pregnancy due to insulin omission, in a pregnant woman.

Case Report

A 28-year-old Black woman, gravida 2, para 2, secondary school teacher was admitted at 31 weeks of gestation to an obstetric ward on August 12/2022 with a two-day history of nausea and vomiting. She had a history of insulin-dependent diabetes mellitus four years earlier and was treated with neutral protamine Hagedorn insulin at 40 and 60 international

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units four years earlier. She had no history of stroke, trauma, pancreatitis, alcohol use, obesity, abused drug use, car accident, underlying infection, or no family history of diabetes mellitus. She missed her insulin dose one day due to traveling to the village for greeting her family.

She presented with a two-day history of nausea and vomiting, and a one-day history of shortness of breath, abdominal tenderness, hypotension, elevated heart rate, increased respiratory rate, frequent urination, and fatigue. Her common vital signs showed: mean arterial blood pressure of 88/59 mmHg, pulse rate of 125 beats/minute, a body temperature of 35.9°C, a height of 1.59 m, a body weight of 63 kg, and respiratory rate of 22 breaths/minute. Her body mass index showed 24.9 kg/m2 (normal value: 18.5–24.9 kg/m2).

Her laboratory investigations revealed: serum glucose level of 437 mg/dL (normal value: 70–100 mg/dL), glycosylated hemoglobin of 5.4% (normal value: 4–6.5%), pH arterial blood of 7.09 (normal value: 7.32–7.43), anion gap level of 17 mEq/L (normal value: 3–10 mEq/L), blood urea nitrogen of 30 mg/dL (normal value: 6–21 mg/dL), serum creatinine of 1.6 mg/dL (normal value: 0.59–1.04 mg/dL), partial pressure of carbon dioxide of 23 mmHg (normal value: 38–42 mmHg), serum bicarbonate level of 8.0 mEq/L (normal value: 22–29 mEq/L), serum phosphate level of 2.6 mg/dL (normal value: 2.8–4.5 mg/dL), white blood cell count of 19,400 cells/mm3 (normal value: 4500–11,000 cells/mm3), serum sodium level of 119 mEq/L (normal value: 135–145 mEq/L), serum chlorine level of 107 (normal value: 96–106 mEq/L), serum potassium level of 3.2 mEq/L (3.5–5.5 mEq/L); and serum ketone level of 3.8 mmol/L (normal value: 0.6–1.5 mmol/L).

Ketone testing done using her urine sample showed ketonuria of 3+. Her chest X-ray revealed coarse crackles on auscultation. Her breath odor revealed acetone-smelling breathing. She had also lowered skin turgor and dry tongue, drowsy, Kussmaul breathing, lowered jugular venous pressure, abdominal tenderness, sunken eyes, decreased conscious level, and pale peripheries on examination.

Upon admission, she was treated with insulin infusion for 24 hours and 60 milliequivalents per liter of potassium chloride intravenously was also initiated. Her blood glucose level was checked five times a day. On the same day, 0.9% of normal saline 500 mL was initiated intravenously stat and repeats until systolic blood pressure was greater than 90 mmHg. On the next day, systolic blood pressure was 96 mmHg, and the attending obstetrician initiated 0.9% of normal saline 1000 mL for four successive hours.

The patient showed marked improvement (fasting blood sugar level lowered to 165 mg/dL) and then, the attending obstetrician switched intravenous insulin to subcutaneous insulin (neutral protamine Hagedorn insulin at 40 and 60 international unit) and discharged her back to her home. She was recommended to carry on follow-up monthly at the ambulatory clinic.

Discussion

Diabetic ketoacidosis has commonly occurred as a complication of type 1 diabetes mellitus and it is a life-threatening disorder that frequently presented with hyperglycemia, metabolic acidosis, and elevated serum ketone level of bodies.[11] Diabetic ketoacidosis is also defined as the biochemical triad of ketonemia, hyperglycemia, and academia.[12] Diabetic ketoacidosis frequently occurs in pregnant women; who had a previous history of type-1 diabetes mellitus, and rarely occurred in type-2 diabetes mellitus.[13]

Pathophysiology of diabetic ketoacidosis comprises two main mechanisms: The first mechanism is altered glucose metabolism: which is converted to hyperglycemia, water deficit, and hemoconcentration consecutively.[14,15]

The successive steps of ketoacidosis formation in this mechanism include absolute insulin deficiency __ altered glucose metabolism __ increased glucose production and decreased glucose utilization __ hyperglycemia __ glucosuria __ osmotic diuresis __ loss of water, sodium, and potassium __ dehydration __ hemoconcentration __ shock __ ketoacidosis.[16]

The second mechanism is altered fat metabolism. Absolute insulin deficiency in diabetic ketoacidosis causes the breakdown of fats in adipose tissue. After the breakdown of fats, glycerol and free fatty acids are produced. Glycerol and free fatty acids are transported into the liver. After they are transported to the liver, there is the oxidation of free fatty acids results in the secretion of two ketoacids (beta-hydroxybutyric acid and acetoacetic acid), but only acetoacetic acid forms real ketoacidosis. Then the accumulation of ketoacids in the body produced ketosis. Finally ketosis converted to ketoacidosis.[17–19]

The successive steps of ketoacidosis formation in this mechanism include absolute insulin deficiency __ altered fat metabolism __ lipolysis __ glycerol __ increased free fatty acid __ increased free fatty acid oxidation __ ketosis __ and ketoacidosis.[20]

Besides the above two pathophysiology of diabetic ketoacidosis, pathophysiology of diabetic ketoacidosis in pregnancy is also characterized by insulin resistance, accelerated starvation, and respiratory alkalosis, thus creating ketosis-prone state. During pregnancy there is production of cortisol, a hormone that promotes hyperglycemia, and placenta produces hormones that antagonize insulin's actions. In pregnant women, there is the glucose pass freely from the maternal circulation to the fetal circulation; hyperglycemia in the mother will stimulate secretion of fetal insulin. Treatment of diabetic ketoacidosis in pregnancy is intended to normalize hyperglycemia and acidosis and correct electrolyte imbalances.[21] Upon admission, she was treated with insulin infusion for 24 hours, and 60 milliequivalents per liter of potassium chloride intravenously.[22] On the same day, 0.9% of normal saline 500 mL was initiated intravenously stat and repeats until systolic blood pressure was greater than 90 mmHg.[23] Since dehydration and sodium loss are common during pregnancy in diabetic ketoacidosis intravenous normal saline was primarily initiated.[24] On the next day, systolic blood pressure was 96 mmHg, and the attending obstetrician initiated 0.9% of normal saline 1000 mL for four successive hours.[25] If plasma glucose is less than 250 mg/dL, the infusion rate should be lowered to 0.05 to 0.2 units/kg/hr, and a dextrose solution of 5% in 1/2-normal saline should be infused at a rate of 150 to 200 mL/hr.[26]

Conclusion

Diabetic ketoacidosis is a severe manifestation of absolute insulin deficiency and is characterized by hyperglycemia, secretion of ketoacids, hemoconcentration, acidosis, and eventually coma. Diabetic ketoacidosis is a rare but major risk during pregnancy among women with diabetes and endangers the life of the mother and fetus. She presented with a two-day history of nausea and vomiting, and a one-day history of shortness of breath, abdominal tenderness, hypotension, elevated heart rate, increased respiratory rate, frequent urination, and fatigue. Upon admission, she was treated with insulin infusion for 24 hours, and initiated 60 milliequivalents per liter of potassium chloride intravenously was also initiated.

Ethical

Institutional approval was not required to publish this case report.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report.

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