Case Report

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A Starvation Ketoacidosis with Severe Hypoglycemia in Non-Diabetic Pregnant Woman

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Abstract

Pregnancy-related physiological changes attribute in increase the risk of ketoacidosis in pregnant women even with a relatively short period of starvation. Starvation ketoacidosis is a medical emergency associated with adverse maternal and fetal consequences. Early recognition and proper treatment can minimize adverse outcomes. However, delay in the diagnosis is common as patients look clinically well despite the presence of significant metabolic acidosis. Understanding the physiological changes in maternal metabolism form the foundation of management of starvation ketoacidosis. Our case represents the consequences of dual factors; persistent hyperemesis till 34 weeks, and acceleration of ketosis in the third trimester of pregnancy which attributes to poor maternal gain weight, fetal growth restriction, and severe metabolic acidosis.

Keywords: Starvation ketoacidosis; Metabolic acidosis; Fetal distress

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Introduction

Pregnancy is a ketogenic state due to relative insulin resistance [1]. This is due to placental hormones including human placental lactogen, cortisol, and glucagon leads to an insulin-resistance, augments lipolysis, and increases the production of free fatty acid. On the other hand, it is associated with chronic respiratory alkalosis. This is compensated by renal excretion of bicarbonate which results in the reduction of the plasma bicarbonate level and hence insufficient buffering of hydrogen ions. These physiological changes in gestation make pregnant women more prone to develop ketoacidosis even with a short period of starvation in comparison to non-pregnant peers [1,2]. Ketoacidosis in general is a known complication for patients with type 1 Diabetes mellitus. However, other causes including alcohol consumption, starvation can also lead to ketoacidosis in pregnancy [3]. Starvation ketoacidosis is a rare critical condition that adversely affects the mother and the fetus. In many instances, the diagnosis is prolonged due to seemingly well patient despite significant metabolic derangement [4,5]. We present a case of 25 years old woman, who had severe metabolic acidosis due to prolonged vomiting. Started from the 9th week till the 35th week of pregnancy which leads to severe metabolic acidosis, intractable hypoglycemia admitted to the Intensive Care Unit (ICU), and had ICU psychosis. She was managed by a multidisciplinary team and emergency Caesarean section. She delivered a male baby who was admitted to the Neonatal Intensive Care Unit (NICU) for metabolic acidosis and Intraventricular Hemorrhage (IVH). Her condition improved gradually after the termination of pregnancy.

Case Presentation

A 25 years old Yemeni female, Gravida 2, Para 0, Abortion 1 at 34 weeks of gestational age presented to the emergency room with nausea and vomiting associated with epigastric pain and poor maternal gain weight. Admitted due to dehydration and urinary tract infection, her urine routine sample showed ketonuria (4+) and a significant leukocytosis in urine, other laboratory investigations include complete blood count, liver function test, renal function test, and electrolytes all within normal range. Patient treated with metoclopramide, vitamin b complex, omeprazole, and antibiotic for the leukocytosis in the urine routine sample. Her vomiting improved after 2 days and her urine midstream culture and sensitivity were sterile. Discharged on antiemetic medication along with cefuroxime for 5 days. Her symptoms started from, 9th week of pregnancy continued throughout her gestation diagnosed as hyperemesis gravidarum. She had recurrent admission due to persistent vomiting and dehydration which was treated with metoclopramide, vitamin b complex injection and omeprazole leads to relief in her symptoms temporarily. Her previous history

Table 1: The arterial blood gas changes: 0 hours (at admission), after 12 hours, 16 hours, 18 hours, 24 hours, 28 hours, 40 hours.

	0 hours	12 hours	16 hours	18 hours	24 hours	28 hours	40 hours
pH	7.29	7.126	7.121	7.131	7.315	7.293	7.486
pCO_2 mmhge	12.2	7	9.4	9.9	15.6	13	18.7
pO_2 mmhge	130	149	143	139	86.6	130	93
HCO_3 mmol/l	10.6	6.4	6.8	7.1	11.7	10.5	18.4
Ag mmol/l	-	20.2	22.3	20.6	20.3	14.3	9
Be mmol/l	-20.1	-26.5	-25.5	-25	-17.7	19.8	-8.9
Lactate mmol/l	1.1	2.1	2.3	2.4	2	1.5	1.1
Glucose mmol/l	8.8	2.3	3.8	8.9	4.5	9.5	8.4

was for helicobacter pylori infection, treated before pregnancy. After 3 days from discharge, presented again with severe nausea and vomiting, inability to tolerate orally, and associated with metabolic acidosis and electrolytes imbalance. She had severe ketonuria (4+) and the arterial blood gas was as follows: pH: 7.299, pCO_2: 12.2, pO_2: 130, SpO_2: 97.6%, K:4.1, Glucose: 8.8, Na: 135, CA: 1.27, CL: 119, $BHCO_3$: 10.1. The patient was admitted to the labor room. A medical team was counseled regarding the metabolic acidosis advised correcting the underlying cause of the metabolic acidosis. The patient was treated initially with three liters of sodium chloride 0.9% and sodium lactate of Intravenous Fluid (IVF), metoclopramide, and omeprazole. One Day 2 of admission patient's condition deteriorated became restless, agitated, and disoriented. Her random blood sugar dropped to 1.2 gm/dL and the arterial blood gas worsens with significant ketone bodies in her blood. A multidisciplinary team was involved to rule out the cause of the hypoglycemia. She treated with 100 ml of 50% dextrose solution followed by 5% dextrose solution, hydrocortisone, and bicarbonate. Also, the patient was having hypokalemia, hypocalcemia, and hypomagnesemia which needed replacements. Her cardiotocography was a non-reassuring trace. She underwent an emergency cesarean section. A male baby weighing 1.620 kilograms with APGAR score 8 and 9 at 1 minute and 5 minutes, respectively. Blood gases venous pH: 7.07, lactate: 7.8 mmol/l, had metabolic acidosis and intraventricular hemorrhage grade 1 which progress to grade 3 after 18 days from the delivery, developed neonatal jaundice due to prematurity on day 3 life, required phototherapy, and discharged from NICU after 23 days. A postoperative patient complained of cough. On examination her blood pressure was 91/61 mmHg, heart rate 130 b/min, oxygen saturation 98% and chest auscultation revealed bilateral crepitations. Chest X-ray showed a congested lung due to aggressive fluid replacement. Lasix 20 mg was given and shifted to the intensive care unit. She was investigated for other causes that contributed to her vomiting, metabolic acidosis and hypoglycemic attack include thyroid function test, serum amylase, serum lipase, random cortisol level, insulin, c-peptide, septic workup, and Synacthen test. On day 4 of admission, while the patient still in the ICU, she became confused and had aggressive behavior. The neurology and psychiatry team was involved to rule out any neurological insult or postpartum psychosis. Brain Computerized Tomography Scan (CT) done which was normal. The diagnosis was ICU syndrome which resolved spontaneously after shifting to the postnatal ward. Her working diagnosis was inconclusive, but it was considered that the hypoglycemia and the metabolic acidosis were probably due to persistent vomiting and poor oral intake. Her condition was improving gradually after the termination of pregnancy and discharge home with Magnetic Resonance Imaging (MRI), neurology and psychiatry appointment. Her postnatal follow-up was uneventful.

Discussion

Nausea and vomiting are a common presentation in pregnancy 3. Intractable loss of gastrointestinal contents induces acid-base derangement and electrolytes imbalance [6,7]. Given the likelihood of the aforementioned to happened and with long periods of malnutrition, hepatic glycogen stores can be worn out quickly. This trickles down to the reduction of the amount of pyruvate that enters the citric acid cycle. Consequently, energy production transitions to lipid mobilization and generation of acetyl-CoA from beta-oxidation of free fatty acids. If the body requirement of energy exceeds the capacity of the citric acid cycle, the accumulation of β-hydroxybutyrate, acetoacetate, and acetone, ends with metabolic acidosis10. In pregnancy, a short period of fasting can cause the formation of ketone bodies and hence metabolic acidosis. This is due to the diabetogenic nature of pregnancy. The placental hormones including human placental lactogen, cortisol, and glucagon lead to an insulin-resistance, augments lipolysis, and increases the production of free fatty acid. On the other hand, it is associated with chronic respiratory alkalosis. This is compensated by renal excretion of bicarbonate which results in the reduction of the plasma bicarbonate level and hence insufficient buffering of hydrogen ions. These physiological changes in gestation increase with advanced gestational age and therefore, make pregnant women more prone to develop ketoacidosis even with a short period of starvation (12 hours-14 hours) in comparison to non-pregnant peers (14 days) [8-10]. Metabolic acidosis associated with an increased Anion gap is often linked to lactic acidosis, ketoacidosis (diabetic, chronic alcoholism, or starvation), or toxic ingestions (aspirin, methanol, and ethylene glycol). Ketoacidosis is a state of metabolic acidosis with a high anion gap, normal range (12 mol/L-16 mol/L) 11. The anion gap is useful to determine the underlying cause. We established a diagnosis of starvation ketoacidosis in this woman based on the exclusion of the other reasons, history of persistent

vomiting, and poor oral intake. Along with the presence of ketonuria, ketone bodies in blood and metabolic acidosis, which improved by administration of 50% dextrose solution but not with normal saline or ringer lactate intravenous solutions. Our patient represents the consequences of persistent hyperemesis gravidarum in pregnancy which usually continues to the early second trimester. However, our case persisted till 34 weeks of pregnancy which attributed to poor maternal gain weight, fetal growth restriction. Though the patient was able to compensate for the persistent vomiting till her third trimester when she presented to our hospital. Due to the combined impact of the physiological changes that occur during pregnancy along with her intractable vomiting her condition deteriorated. Furthermore, the keystone of management of this patient is early recognition and the ideal therapeutic approach for starvation ketoacidosis is glucose administration [1,2,4]. In our case, we did not detect the pathogenesis at first, and in the beginning, we started her on ringer lactate and 0.9% normal saline which did not improve her metabolic disturbance. Maternal metabolic acidosis may have severe consequences not only on the mother but the fetus as well. As it may lead to fetal acidosis and hypoxemia. In our case, the fatal outcome was a premature alive baby male with low birth weight complicated by metabolic acidosis intraventricular hemorrhage, and jaundice. Multiple factors contributed to the fatal outcome which includes persistent hyperemesis gravidarum, poor maternal gain, metabolic acidosis, and prematurity.

Conclusion

Metabolic acidosis is a life-threatening condition that adversely affects the mother and the fetus. Understanding the physiological changes during pregnancy and their effect on maternal metabolism forms the basis of management of starvation ketoacidosis. The cornerstone to abide the adverse outcome are early recognition of the ketoacidosis, rapidly address the underlying cause, and appropriate management accordingly.

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