Severe metabolic acidosis and respiratory distress due to acute starvation in pregnancy: a case report

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Abstract

This case report presents a pregnant woman with acute starvation ketoacidosis. Metabolic acidosis can be a consequence of acute starvation in the third trimester of pregnancy. Maternal acidosis is a medical emergency in which both mother and child are at risk for significant morbidity and mortality. The problem of acute starvation should be identified rapidly and the right treatment with substitution should be given in time. When starvation has occurred, substituting nutrients, intravenous glucose and

prevention of circulatory hypovolemia is recommended. In this case we present warning symptoms, such as respiratory distress, and the severe consequences of a maternal acidosis.

Keywords maternal acidosis, starvation ketoacidosis, respiratory distress.

Introduction

Acute starvation ketoacidosis in pregnancy is a rare affliction with a severe course of disease for both mother and child. Early recognition and immediate management are required to minimize the risk of severe outcome. Ketoacidosis occurs more often in patients with diabetes mellitus type 1 or alcoholic abuse.[1-2] In rare cases, metabolic acidosis can be a consequence of acute starvation in the third trimester of pregnancy in non-diabetic patients.[3-8]Many cases show patients presenting themselves with a short period of fasting, accompanied by vomiting while patients look clinically disproportionally well. This kind of presentation also may cause a delay in recognition and therapy, resulting in a progression of metabolic acidosis with potentially fatal consequences. We describe a case of respiratory distress and starvation induced severe metabolic acidosis during third trimester in a pregnancy complicated by preeclampsia and gestational diabetes.

Case presentation

A 29 year old multipara woman, at 28 weeks of gestation, presented with dyspnoea, maternal tachycardia and tachypnea. Past medical history included an appendectomy and one uncomplicated previous pregnancy. At first presentation a CT-scan was performed, which showed no signs of lung embolism. At a gestation of 30 weeks, pregnancy induced hypertension was diagnosed with a blood pressure of 140/80 mmHg. Laboratory results did not show any abnormalities and the urinalysis was normal. Cardiotocograph was normal. After one week, at a gestation of 31 weeks and 2 days, blood pressure raised to 150/84 mmHg, after which methyldopa 500 mg 3 times a day was started. Fasting glucose was 7.7 mmol/L (normal range: <7.0 mmol/L) and the diagnosis gestational diabetes was concluded, although no anti-diabetic medication was started. At 34 weeks of gestation, the patient presented malaise and false contractions. Urinalysis showed 2+ protein with a protein-creatinine ratio of 92.1 mg/mmol (normal <30 mg/mmol). Dyspnoea and palpitations accompanied the complaints of malaise. Physical examination showed a maternal tachycardia of 124 bpm, a respiratory rate of 24 breaths per minute and a blood pressure of 130/75 mmHg**.** Due to the presence of hypertension and albuminuria, preeclampsia was diagnosed. Another CT-scan was performed, which again showed no signs of pulmonary embolism. Random glucose was 9.6 mmol/L without antidiabetic medication. Laboratory results showed a hypokalaemia of 3.3 mmol/L (normal 3.5-5.0 mmol/L) without other abnormalities. The patient was admitted at the obstetric unit for observation and further analysis, with preeclampsia.\sout The next day (34+1), the symptoms persisted and were accompanied by nausea. A physical examination showed a blood pressure of 135/69 mmHg, a heart rate of 110 bpm and a saturation of 99%. There was no registration of respiratory rate. There were no signs of oedema and the knee reflexes were normal. A systolic souffle was found during auscultation. Cardiologic exam concluded hyperdynamic circulation and anaemia (haemoglobin: 7.7 mmol/L). At a gestation of 34 weeks and 2 days, symptoms were unchanged. Due to high fasting glucose (7.2 mmol/L) treatment with insulin was started. Due to the gestational diabetes, an ultrasound examination was performed: head circumference (HC) p98, abdominal circumference (AC) >p100 and estimated fetal weight (EFW) >p100, from which macrosomia was concluded. Laboratory results still showed a hypokalaemia of 3.4 mmol/L, as well as an elevated uric acid of 0.57 mmol/L (0.12-0.34 mmol/L). 24-hour urine showed 0.66 grams of protein. At a gestation of 34 weeks and 4 days, symptoms worsened with vomiting (three times a day) and increase of malaise. Respiratory rate was 24 breaths per minute, saturation 99% without oxygen, blood pressure 140/90 mmHg and heart rate 120 bpm. During physical exam, no other abnormalities were found. Due to high blood pressure, methyldopa was changed to 1000mg three times day. Urinalysis showed 4+ ketones. Blood test showed an elevated uric acid of 0.77 and a random glucose of 6.8 mmol/L. Rehydration with sodium chloride 0.9% was started. The insulin dosage was increased. At 34 weeks and 5 days of gestation, the patient and her family expressed their concerns. Her blood pressure was 140/80 mmHg, maternal heart rate was 128 bpm and her respiratory rate was 20-25 per minute. Venous blood test showed a severe metabolic acidosis with an incomplete respiratory compensation (pH 7.15, pCO2 2.8 kPa, bicarbonate 7.4 mmol/L, base excess -19.5 mmol/L, Lactate 1.9) with an increased anion gap (22.6 mmol/L), for which the Intensive Care Unit (ICU) was consulted. With the probability diagnosis: starvation induced severe metabolic acidosis. Sodium bicarbonate (8.4%), glucose and thiamine were given intravenously.\sout Due to the severe acidosis, an emergency caesarean section was performed under general anaesthesia, with a total blood loss of 800cc. Peri-operative patient received 10% glucose IV to continue correcting the starvation. A male newborn (3850 grams) was delivered with an APGAR score of 2/5/7 and a pH level of 7.05, pCO2 7.3 kPa, base excess -14.9 mmol/L. Her child was transferred to an academic centre for continuous positive airway pressure (CPAP) treatment due to idiopathic respiratory distress syndrome (IRDS) and hypoglycaemia. On arrival at ICU, patient had a blood pressure of 151/94 mmHg, a pulse of 140/min and patient was still intubated. Admission arterial blood gas showed a persisting severe metabolic acidosis (pH 7.14, pCO2 4.1 kPa, base excess -17.2 mmol/L, bicarbonate 10.5 mmol/L). Further laboratory findings showed an anion gap 21 mmol/L, chloride 111.0 mmol/L, lactate 1.9 mmol/L and a glucose 22.5 mmol/L. The intravenous glucose (10%) fluid, alongside a sodium chloride fluid therapy was continued. Furthermore, intravenous insulin, potassium and thiamine were started, to compensate for the electrolyte shift induced by the therapy. The patient was extubated,

quickly and successfully. The acid base balance returned to normal within 24 hours of the ICU admission. The ketonuria was completely gone within 48 hours of treatment. The patient was then discharged to the department of obstetrics.

Discussion

Our case had many similarities to cases reported in the literature. Our patient presented with a recent acute starvation with nausea and vomiting and was tachypnoeic and tachycardic with a significant acid-base disturbance. The signals of the respiratory distress were present at a gestation of 34 weeks with complaints of dyspnoea and a respiratory rate of 24 breaths per minute. The laboratory results revealed a severe metabolic acidosis with a high anion gap (22.6 mmol/L). Anion gap is used to distinct the aetiology of the metabolic acidosis. Causes of a high anion gap are the presence of high lactate, ketones or exogenous acids. Our patient did not have an elevated lactate (1.9 mmol/L) or signs of been exposed to any exogenous acids during pregnancy. In our patient, there were elevated amounts of ketones present in her urine sample. Causes of ketoacidosis within pregnancy are alcohol induced, uncontrolled diabetes or starvation induced.[5] Alcoholic-induced acidosis typically occurs in the setting of excessive alcohol consumption followed by prolonged vomiting. In our patient, there were no signs of alcohol abuse. Diabetic ketoacidosis is characterized by hyperglycaemia (glucose >13.9 mmol/L)[9], therefore this diagnosis was excluded. The acute starvation that our patient endured, prior to the worsening of her symptoms made the diagnosis of starvation ketoacidosis more likely. Furthermore, the aspect that in our case the metabolic disturbance rapidly could be corrected with glucose intravenous fluids, helped to confirm our diagnosis. This diagnosis became more likely because the electrolytes disturbance could be rapidly corrected with a glucose containing intravenous fluids.

In healthy non-pregnant women, it takes up to 14 days to reach the maximum of severity of starvation ketoacidosis, which would manifest with mildly elevated ketoacid levels and minimal acid-base disturbances.[5] Early research has shown that starvation ketoacidosis can be present in such an accelerate state, that it might manifest, even within 12-14 hours.[5] This accelerated development is due to many factors including insulin resistance, increased lipolysis and ketogenesis that occur during pregnancy.[5-6] In the third trimester, in normal pregnancy, plasma bicarbonate concentration decreases, which reduces buffering capacity.[8] Hepatic glycogen stores are depleted after prolonged fasting, leading to fatty acid metabolism and ketone body formation and acidosis. Women with comorbidities such as preeclampsia and gestational diabetes may be more at risk for starvation. Preeclampsia can cause severe vomiting, which may worsen the process of starvation due to less nutritional intake. In our case, the diagnosis of gestational diabetes lead to fasting.

Maternal metabolic acidosis may have serious consequences for both mother and the fetus. Maternal metabolic acidosis causes an increased maternal respiratory rate to decrease levels of pCO2 to return the pH level to a normal rate.[10-12] The elevated respiratory rate is in turn associated with an increased risk for cardio-pulmonary arrests and ICU admission.[13-15] Furthermore, the reduced blood flow caused by vasoconstriction due to decreased levels of pCO2 in the maternal circulation can possibly cause fetal acidosis. This may result in impaired fetal neuronal function.[4]Furthermore, the acidity within pregnancy is associated with intrauterine death. It is thought to be caused by the transfer of ketones through the placenta, the maternal electrolyte imbalance and maternal volume depletion.[6]

Conclusions

Maternal acidosis is a medical emergency, in which both mother and child are at risk for significant morbidity and mortality. Acute starvation in the third trimester of pregnancy may give maternal metabolic ketoacidosis. Patients with comorbidity such as gestational diabetes and preeclampsia may be more at risk to develop starvation ketoacidosis. Due to the higher tendency of ketogenesis in pregnancy, clinicians should be aware of the risks of starvation. Urinalysis should be performed when a patients has symptoms of dyspnoea, nausea

and vomiting. A respiratory rate above 16 breaths per minute is one of the warning symptoms for respiratory distress and is associated with an increased risk for ICU admission. When ketones are found in urinalysis and the patient is in respiratory distress, arterial blood gas is advised. When starvation has occurred, substituting nutrients, intravenous glucose and prevention of circulatory hypovolemia are recommended. Potassium should be added to prevent hypokalaemia when acidosis subsides. Daily monitoring of respiratory rate, urine analysis for ketones and electrolytes are recommended to monitor a safe transition to a normal metabolic balance. To prevent maternal and fetal morbidity, the problem of acute starvation should be identified rapidly and the right treatment with substitution should be given on time.

Disclosure of interest

None declared.

Contribution of authorship

KJ and CS researched the case and wrote the manuscript. KJ and GH provided obstetric care. CS and HP provided care at the intensive care unit. GH and HP edited the manuscript. All authors have seen and agree with this case report.

Details of ethical approval

A written consent was obtained from the patient.

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