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Keep Baby In Mind, Don't Be Unkind: A Case Report On Alcoholic Ketoacidosis In Pregnancy

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ABSTRACT:
Alcoholic ketoacidosis is one of the underdiagnosed ketoacidosis condition in non- diabetic patients with chronic alcoholism. There are only a few studies reported till now on Alcoholic ketoacidosis in pregnancy. Here we report a case of 30 years old pregnant female who presented with complaints of pain abdomen, vomiting with a history of chronic alcohol intake. Patient was treated in-hospital with aggressive fluid therapy, Delivered baby normally with spontaneous labour. In pregnant alcoholics, there is an early production of ketoacids due to metabolic derangements. It is very important to differentiate alcoholic ketoacidosis from starvation and diabetic ketoacidosis as the management varies completely. Early diagnosis and management improve the prognosis of the patient.

Key Words: *Alcoholic ketoacidosis, Pregnancy, Ketoacidosis, Starvation, Diabetic Ketoacidosis.*

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I. Introduction

One of the unrecognized causes of ketoacidosis in non-diabetic patients is chronic alcoholism (1). Ketoacidosis in chronic alcoholic is not only due to excessive accumulation of ketoacids leading to complex abnormalities of Acid-base balance but also related to depletion of extracellular volume and electrolytes (2). Often patients present with history of recent bout of heavy alcohol consumption with complaints of vomiting and abdominal pain with the period of relative starvation (3). Alcoholic ketoacidosis can be fatal if it is associated with electrolyte abnormalities and subsequent development of cardiac arrhythmias. The diagnosis of Alcoholic Ketoacidosis can be delayed or missed, in patients who have ketoacidosis and severe lactic acidosis (4).There are very few studies that have reported alcoholic ketoacidosis in pregnant women, as this is a very rare entity in pregnancy. It should be recognized early and differentiated from other acidemias seen in pregnancy like Diabetic Ketoacidosis, Starvation Ketosis and Lactic Acidosis. They have detrimental effects on the survival of the fetus.

II. Case Report

A 30 Years old female with obstetric score G3P2L2, in her 24th week of gestation, presented to Emergency Department with complaints of pain abdomen radiating to the back since 5 days associated with nausea, multiple episodes of vomiting and anorexia. She denied any diarrhea, fever or any vaginal bleeding or discharge. There was a history of long standing alcohol abuse and she admitted to heavy alcohol binge for one week prior to her admission.

On examination, patient was found to be distressed, tachypneic and severely dehydrated. Pulse rate was 140 bpm, BP 70/40 mm of Hg, Respiratory rate was 50/minute and Kussmaul in character. Her respiratory and cardiovascular system examinations appeared normal. Per abdomen examination revealed ovoid abdomen with generalized tenderness, normal bowel sounds. Fetal heart rate 130bpm. Pelvic examination was done, her cervix was closed and non effaced.

Arterial Blood Gas showed pH 6.925, pCO2 10 mm Hg, HCO3 1.7 mmol/L. Calculated anion gap was 32.3 mmol/L. Serum Glucose was 133 mg/dL. Urine analysis showed ketonuria (3+) but absence of protein and glucose. Ultrasonography of the abdomen showed gravid uterus with intrauterine live gestation corresponding to 23weeks 5days, maternal liver, kidney, pancreas were structurally normal.

The findings of ketoacidosis with normal serum glucose in a patient with history of heavy alcohol consumption made the diagnosis of Alcoholic Ketoacidosis likely.

The patient was intubated and mechanically ventilated in view of severe metabolic acidosis and respiratory distress. She was started on aggressive fluid therapy (Ringer Lactate/5% Dextrose), Inj Thiamine was supplemented. Eventually her blood pressure normalized and heart rate came down to 90 to 100 bpm. Arterial Blood Gas over the next 24 hours showed significant improvement in keeping with an improved clinical state. Patient was subsequently extubated. She went into spontaneous labour and delivered a live baby boy weighing 750gms. Baby was immediately shifted to NICU for further care.

III. Discussion

AKA is usually diagnosed clinically, evaluated and managed in the Emergency Department. Several pathophysiologies have been postulated. The basic pathophysiology is due to poor glycogen stores and elevated nicotinamide adenine dinucleotide and hydrogen which results in elevated beta-hydroxybutyrate levels with metabolic acidosis (5).

During alcoholic ketoacidosis various hormonal changes occur mostly as a result of alcohol-induced hypoglycemia which includes low insulin levels and increased levels of cortisol, growth hormone, glucagon, and epinephrine. This promotes lipolysis, in turn increases the levels of free fatty acids which are available for conversion to ketones.

Additional mechanisms that may contribute to ketosis include ketones from acetate; increased rate of ketosis due to mitochondrial structural changes due to alcohol; and mitochondrial phosphorus depletion, which in turn inhibits the utilization for NADH and elevates ketone body formation. Finally, vomiting and starvation overlaying on chronic malnutrition also contribute to ketoacidosis (6).

The main differential diagnoses for ketosis in our patient included AKA and starvation ketosis but in most starvation ketosis, a mild ketosis are noted to develop after 12–24 h of fasting.

However, the body overcomes this physiologically by utilizing peripheral ketone bodies, stimulating increased insulin release inspite of the presence of low glucose levels, and direct inhibition of further lipolysis by the ketone bodies. Hence, only a mild acidosis is noted in starvation ketosis (7).

There is a similar effects seen with pregnancy and ethanol on carbohydrate, lipid and protein metabolism.

In pregnancy, glycogen is depleted faster due to increased usage of carbohydrates and converting primary metabolic pathway to lipolysis. Glucose being the sole source of energy for the fetus. This causes accelerated starvation and results in lower fasting glucose levels in pregnant women. In reflex, there is increased levels of glucagon, lipolytic hormones, growth hormone and cortisol.

As the conceptus matures, there is increased production of Human chorionic somatotropin. There is also transient increase in somatotropin levels in the fasting or malnourished patient. As a result there is increased stimulation of lypolysis and gluconeogenesis (10).

There is a contradictory influence in fetal acid- base status due to placental permeability to carbon dioxide . Fetal alkalosis occurs in order to 'balance' the buffer equation as a result of maternal compensation in a very low $PaCO_2$ which would further lower fetal $PaCO_2$ (11).

The main principle of emergency management is adequate fluid resuscitation. To reduce factors contributing to the severe acidosis like lactic acid, ketoacids and acetic acid by Increasing volume status and providing increased perfusion to tissues

Significant management is not only to promote rapid recovery from AKA, but also prevent complications associated with alcohol excess, in particular treating malnutrition and electrolyte deficiencies(8).

As mentioned earlier electrolyte disturbances are the main cause of mortality in AKA, it should be corrected immediately to prevent cardiac arrhythmias.

Based on the risk of seizures, Intravenous benzodiazepines can be administered from impending alcohol withdrawal. To manage nausea and vomiting antiemetics such as ondansetron or metoclopramide may also be given (3).

AKA should be suspected in any patient who has a history of chronic alcohol dependency, malnutrition or recent episode of binge drinking.

IV. Conclusion

In general, as early as the condition is identified and treated , there is good prognosis for a patient presenting with AKA. Delay in presentation or diagnosis might result in end-organ damage such as acute renal failure with tubular necrosis (9).

The key is to differentiate alcoholic ketoacidosis from starvation and diabetic ketoacidosis (DKA).

In patients with AKA, Systematic and timely management can lead to enhanced patient outcomes. However, it is equally important to prevent recurrence and long-term irreversible damage from alcohol abuse by referring the patient to alcohol abuse rehabilitation programs after adequate management (3).

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