CASE REPORT

Differential Diagnosis of Ketoacidosis in Hyperglycemic Alcoholic Diabetic Patient: Role of Insulin

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Abstract

It is important to differentiate between diabetic ketoacidosis (DKA) and alcoholic ketoacidosis (AKA) in an alcoholic diabetic patient since it has significant management implications. Ketoacidosis in an alcoholic diabetic patient is a diagnostic challenge as both these clinical entities have metabolic acidosis with high anion gap. Most patients with DKA have hyperglycemia. The majority of AKA patients present with normal or low glucose levels; however, AKA may also present with high glucose levels, more so in diabetics. The situation becomes quite perplexing when an alcoholic diabetic patient presents with hyperglycemia since it can be attributed to DKA or AKA.

Keywords: Alcoholic ketoacidosis, Binge drinking, Diabetic ketoacidosis, High anion gap, Ketonemia.

Indian Journal of Critical Care Medicine (2021): 10.5005/jp-journals-10071-23988

Introduction

Diabetic ketoacidosis (DKA) is an acute life-threatening complication mainly seen in type 1, but not uncommon with type 2 diabetes. It is a state of absolute or relative insulin deficiency and requires emergency treatment with insulin and intravenous fluids.[1]

Alcoholic ketoacidosis (AKA) occurs most commonly in chronic alcoholics with a recent history of binge alcohol intake along with little or no food intake and persistent vomiting.[2] Dextrose and fluid replacement reverses the pathophysiologic derangements that lead to AKA by increasing endogenous serum insulin levels and suppressing the release of glucagon and other counter-regulatory hormones.

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How to cite this article: Garg SK, Garg P. Differential Diagnosis of Ketoacidosis in Hyperglycemic Alcoholic Diabetic Patient: Role of Insulin. Indian J Crit Care Med 2021; 25(10):1203–1204.

Source of support: Nil

Conflict of interest: None

Case Description

A 47-year-old male patient presented to the emergency department with a history of palpitation, dizziness, and vomiting for 10 to 12 hours. His vitals included heart rate 151 per minute regular, blood pressure 180/106 mm Hg, respiratory rate 24 per minute, and normal temperature.

His known medical conditions included diabetes mellitus type 2 and hypertension on medication which he stopped on his own for the last few days. He has been drinking alcohol of approximately 150 to 200 mL daily for the last 10 years.

Laboratory investigations revealed metabolic acidosis with pH 7.23, anion gap 23, positive urinary ketone, blood sugar 14.6 mmol, serum bicarbonate 13.5 mmol, serum lactate 10.1 mmol/L, and serum β-hydroxybutyrate 5.5 mmol. His HbA1C was 10.8 and serum cholesterol 10.43 mmol/L. Laboratory values were significant for hypomagnesemia and hypophosphatemia. Amylase and lipase values were normal.

At this point, we were not confirmed about the cause of ketoacidosis since he was an alcoholic diabetic patient and presented with hyperglycemia which could be attributed to DKA or AKA. Before starting the treatment, we wanted to find out the cause of ketoacidosis in this patient since if it was AKA, starting insulin alone could lead to rapid hypoglycemia. To rule out AKA, we specifically asked about the alcohol intake and it was then revealed that he had binge alcohol intake during the last few days and had excessive vomiting which started around 10 to 12 hours back. This was suggestive of AKA.

So the management was done very aptly by starting insulin and dextrose saline simultaneously in view of AKA in a patient who was diabetic. Ketoacidosis was resolved completely within next 24 hours. On the third day, he developed symptoms of alcohol withdrawal for which he was maintained on an increasing dose of benzodiazepine.

Discussion

Diagnosis of alcoholic or diabetic ketoacidosis in a typical patient is rather straightforward. The difficulty arises when a patient who is both diabetic and alcoholic presents with hyperglycemia and ketoacidosis.

Alcoholic ketoacidosis is a frequently underdiagnosed cause of ketoacidosis. Most patients with AKA present with normal or low glucose, but this condition can also present with high glucose levels.[3,4]

Possibility of developing hyperglycemia in AKA is even more if an alcoholic patient is diabetic. Ketoacidosis in an alcoholic diabetic patient can be misdiagnosed as DKA if the patient is hyperglycemic, which is partly contributed by less awareness among clinicians about AKA, and therefore, such patients are inappropriately started

with insulin without dextrose and have a high risk of developing rapid hypoglycemia.[4]

The distinction between DKA and AKA based on sugar level in an individual patient is difficult especially when a patient is an alcoholic diabetic. Though unusual, patients of AKA may have high sugars and 10% of the DKA population presents with so-called euglycemic DKA where glucose level is ≤250 mg/dL.[5] Patients with DKA and AKA differ in their metabolic parameters more than in their hormonal profile. The initial hormonal profile in both ketoacidosis states is characterized by reduced insulin levels and elevated levels of counter-regulatory hormones.[6] The metabolic profile of DKA is characterized by a higher plasma glucose concentration and lower β-hydroxybutyrate to acetoacetate and lactate to pyruvate ratio compared with AKA. Absolute values of ratio or lactate or β-hydroxybutyrate to differentiate acidosis in AKA or DKA have not been defined. Lactate may be elevated in both DKA and AKA.[2] Lactic acidosis is common in DKA, with 68% of patients having some degree of elevation and 40% of patients with levels greater than 4 mmol/L.[7]

Though physical examination dictates that patients of AKA are more lucid and alert in comparison with DKA, AKA cannot be ruled out if a patient is not alert since other variables may lead to altered sensorium in AKA.[2,3]

Since there is no confirmatory investigation available for the diagnosis of AKA in an alcoholic diabetic patient who presents with hyperglycemia, history of binge alcohol intake followed by poor oral intake favors AKA and patient should be managed with insulin and dextrose. In case no such history is available due to any reason, diagnosis can be made based on response to insulin as an alcoholic diabetic patient of AKA and DKA responds differently.

Administration of insulin alone in AKA leads to rapid fall in blood sugar and confirms the diagnosis of AKA, while DKA requires a substantial amount of insulin without dextrose initially. One should be given insulin alone to look for the initial response under strict supervision.

Conclusion

It is important to establish the cause of ketoacidosis in an alcoholic diabetic patient given significant management implications. If clear and adequate history is not available, insulin administration should be closely monitored at least initially to avoid hypoglycemia.

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