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Case Report

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Ketoalkalosis: Masked Presentation of Diabetic Ketoacidosis With Literature Review

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Abstract

Most dreaded complication in type 1 diabetes mellitus remains diabetic ketoacidosis (DKA): plasma blood glucose > 250 mg/dL, serum bicarbonate < 18 mEq/L, anion gap metabolic acidosis and ketosis. Insulin deficiency with high levels of glucagon and stress hormones causing ketogenesis in liver, elevated lipolysis in peripheral tissues, and increased free fatty acids contribute the formation of ketones leading to metabolic acidosis. And hence DKA is also termed as ketoacidosis. Unusually, patients with diuretic use, alkali ingestion, intractable vomiting, or hypercortisolism may present with alkalemia in DKA. Contraction (Metabolic) Alkalosis masquerades metabolic acidosis with anion gap and low to normal bicarbonate that uncovers on provision of intravenous fluids. We present a case of a 25-year-old female with DKA presenting with intractable vomiting, alkalotic pH and high anion gap.

Keywords: Ketoalkalosis; Type 1 diabetes; Diabetic ketoacidosis; Vomiting; Metabolic alkalosis; High anion gap

Introduction

Diabetic ketoacidosis (DKA) remains the most dreaded complications of type 1 diabetes mellitus (T1DM). Non-adherence to insulin, concomitant infections, or new diagnosis of DKA ensues in the uninhibited production of ketones and chaotic metabolic state with hyperglycemia, ketoacidosis and ketonuria. DKA is defined as blood glucose greater than 250 mg/dL, blood pH less than 7.3, serum bicarbonate less than 18 mEq/L and elevated anion gap metabolic acidosis. Atypically use of diuretic, intractable vomiting, or hypercortisolism may present with alkalemia in DKA. On presentation, these patients' acidosis would be masqueraded by alkalosis. We present a unique case of 25 years old female with DKA and alkalemia.

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Case Report

A 25-year-old female with type 1 diabetes mellitus presented with complaints of intractable vomiting, generalized weakness and abdominal pain. Six hours prior to arrival to emergency department (ED), she started to experience diffuse abdominal pain around periumbilical area and subsequently started intractable non-bilious vomiting. At home, she was prescribed to insulin detemir 25 units once a day along with insulin aspart 6 units along with three meals. She stated of adherent to insulin dosages, but missed the morning dose. On admission, she was lethargic, dry mucous and oral mucosa, Kussmaul respiration and intense periumbilical pain. Patient was afebrile, blood pressure 107/58 mm Hg, respiratory rate 26 breaths/min, pulse 99 beats/min, and oxygen saturation 99% on ambient air. Electrocardiogram interpreted normal sinus rhythm with rate of 86 beats/min with QT corrected prolongation of 507 ms.

Initial laboratory investigations included blood glucose 534 mg/dL, serum sodium 133 mEq/dL, chloride 93 mEq/dL, potassium 5.1 mEq/dL, creatinine 0.94 mg/dL, bicarbonate 20 mEq/dL, albumin 3.5 g/dL, white blood cells 7,100/mm^3, hemoglobin 10.1 g/dL, platelets 273,000/mm^3, magnesium 1.6 mg/dL, lactic acid 1.5 mmol/L and serum ketones large positive. Blood gas analysis showed pH 7.43, pCO_2 28 mm Hg, and bicarbonate 19 mmol/L. Urinalysis showed high ketones and urine glucose > 500 mg/dL.

Treatment with intravenous (IV) saline and replenishment of magnesium was started immediately, and insulin was given 8 units subcutaneously once. After receiving two liters of IV saline, patient was awake and alert and she was ordered to be given 10 units of long-acting insulin. On repeating metabolic panel in 5 h, serum sodium level showed 130 mEq/dL, potassium 3.3 mEq/dL, chloride 102 mEq/dL, bicarbonate 11 mEq/dL and blood glucose 263 mg/dL. Initial corrected anion gap for albumin was 21.25 mEq/L and subsequent anion gap improved to 18.75 mEq/L. Blood gas analysis showed pH 7.27, pCO_2 19 mm Hg, and bicarbonate 9 mmol/L (Table 1). Patient was upgraded to intensive care unit (ICU), initiated potassium IV supplements, and started on dextrose 5% water-normal saline 150 mL/h. After receiving potassium, she was started on insulin 2 units hourly via IV. Following 8 - 10 h, patient's ab-

Table 1. Laboratory Investigations at the Time of Admission and After IV Fluids Resuscitation

	A: at the time of admission	B: after IV fluids resuscitation
Arterial pH (7.37 - 7.45) on blood gas	7.43	7.27
$PaCO_2$ (mm Hg) on blood gas	28	19
Bicarbonate (mmol/L)	19	9
Sodium (mEq/L)	133	130
Potassium (mEq/L)	5.1	3.3
Chloride (mEq/L)	93	102
Bicarbonate (mEq/L)	20	11
Blood urea nitrogen (mg/dL)	23	23
Creatinine (mg/dL)	0.94	0.83
Glucose (mg/dL)	534	263
Corrected anion gap	21.25	18.75
Albumin (g/dL)	3.5	3.3

Showing point A: hyperglycemia with blood sugar of 534, corrected AG of 21, alkalemic pH and bicarbonate of 20. Point B shows improvement in anion gap and blood sugar after administration of IV fluids; however, we can clearly see pH becomes more acidotic and patient develops more severe metabolic acidosis meeting all four criteria for the diagnosis of DKA.

dominal pain was resolving, no episodes of emesis, attentive and urged to eat. Her blood glucose was 198 mg/dL, received 12 units of long acting insulin and IV insulin was discontinued after 1 h. Order for short-acting insulin 10 units with meal was placed as she tolerated diet. Subsequently, she was transferred to medical floor once deemed stable. After course of 36 h, she was discharged with long acting insulin 16 units twice daily along with 8 units with meals.

Discussion

DKA provoked by the lack of insulin and uninhibited levels of glucagon causes ketogenesis in the liver. This further leads to lipolysis in the peripheral tissues leading to production of free fatty acids and formation of ketone bodies, such as 3-hydroxybutyrate and acetoacetic acid, conjuring acidic pH [1].

Previously there have been publications reporting prevalence of combined acid-base disorders in DKA as high as 47.5%, and of which, most common was metabolic alkalosis [2].

In patients presenting with DKA, they commonly present with profuse vomiting. Intractable vomiting in these patients causes development of fluid volume contraction alkalosis. Vomiting causes chloride depletion that indirectly stimulates bicarbonate resorption and decreasing secretion in the collecting ducts of the kidneys. Additionally, it causes excretion of potassium in the kidneys eliciting the activation of the renin-aldosterone axis resulting in bicarbonate reabsorption [1, 2]. With volume depletion, resulting hyperaldosteronism induces exchange of sodium ions for potassium ions or hydrogen ions. This chaotic metabolic interplay results in state of alkalemia. In case series by Svart et al, they observed six patients with diabetic ketoalkalosis; all six patients had commonly associated symptoms of severe nausea and vomiting [3]. There is also evidence that patients having autonomic neuropathy such as gastroparesis due to poorly controlled diabetes, usually present with recurrent vomiting [4].

High anion gap, usually seen in DKA, is associated with an increase by the addition of new anions, such as lactic acid. In our case, patient had a high anion gap with normal lactate and marked alkalosis.

Treatment of diabetic ketoalkalosis does not differ from the conventional management of DKA. These patients should be treated with aggressive IV fluids, electrolytes supplementation (especially potassium), and hourly IV insulin. Administration of IV fluids remains of utmost priority with timely replenishment of electrolytes. In case reported by Jerrard et al, adding potassium to an IV saline solution aid in quickly reversing the alkalotic state [5].

This case illustrates patients in DKA may present with severe dehydration from vomiting leading coexistence of multiple metabolic derangement, systematic blood gas analysis, and normal or high pH. We hope our case helps to improve index of suspicion with prompt diagnosis and medical treatment in DKA.

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Conflict of Interest

There is no conflict of interest to be disclosed for any authors of this paper.

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