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How Low Can You Go? Severe Acidemia in a Patient with Type 2 Diabetes and Diabetic Ketoacidosis

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ABSTRACT

Introduction: Type 2 diabetes mellitus (T2DM) is a common disease encountered in the emergency department. Diabetic ketoacidosis (DKA) is a potentially life-threatening metabolic disturbance characterized by hyperglycemia, metabolic acidosis, and ketonemia. Traditionally associated with type 1 diabetes mellitus, DKA is becoming increasingly common in type 2 diabetics.

Case Report: We present an extreme case of a 66-year-old female with known type 2 diabetes mellitus who presented with altered mental status and severe metabolic ketoacidosis with a pH of 6.55 and blood glucose of 963 mg/dL. The patient rapidly decompensated in the emergency department, requiring emergent intubation and central venous access. Fluid resuscitation was applied, and the patient was started on an insulin infusion. Her blood pressure was supported with dual vasopressor therapy, and she was transferred to the medical intensive care unit. She recovered rapidly and was discharged from the hospital 5 days later with no neurologic deficits.

Conclusion: This case is notable for the patient's extreme acidemia, one of the lowest recorded in the literature for a type 2 diabetic who survived and was discharged from the hospital. It highlights the importance of early, aggressive treatment of DKA in the emergency department coupled with continued critical care management in the intensive care unit.

Keywords: Critical care, resuscitation, diabetic ketoacidosis, diabetes mellitus, DKA, metabolic acidosis

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Introduction

Type 2 diabetes mellitus (T2DM) accounts for 90%–95% of the cases of diabetes mellitus in the United States (1). Diabetic ketoacidosis (DKA) is a potentially life-threatening metabolic disturbance characterized by hyperglycemia, metabolic acidosis, and ketonemia (2). DKA arises when there is a deficiency of insulin along with an increased counterregulatory hormone activity (3). Although typically thought of as a complication of type 1 diabetes, DKA occurs in type 2 diabetics as well, especially in those of African or Hispanic descent (2, 4). In 2006, 35% of reported DKA cases in the United States occurred in type 2 diabetics (2). The most common causes of DKA in T2DM are lack of compliance with insulin therapy and increased catabolic stress, such as infections. Despite continued research and new drug therapies, hospital admissions for DKA are on the rise. Over the last two decades, admissions have increased nearly 75% in the United States, from 80,000 in 1988 to 140,000 in 2009 (2, 3, 5). However, the DKA related age-adjusted mortality rate has decreased 64% to 17.3 per 100,000 diabetic patients in 2009. DKA admissions account for an estimated 500,000 hospital days per year with an estimated annual cost of US$2.4 billion (2). Part of the increased incidence of DKA may be related to the increased prevalence of DKA in T2DM (5).

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Defined as pH ≤7.00, bicarbonate level ≤10.00 mEq/L, anion gap >12 mEq/L, positive ketones, and altered mental status, severe DKA is seen more commonly in low- and middle-income countries where there is less access to medical services (3, 6). This has limited the study of severe DKA in T2DM and resulted in a lack of evidence for recommendations on the treatment of this increasingly common condition.

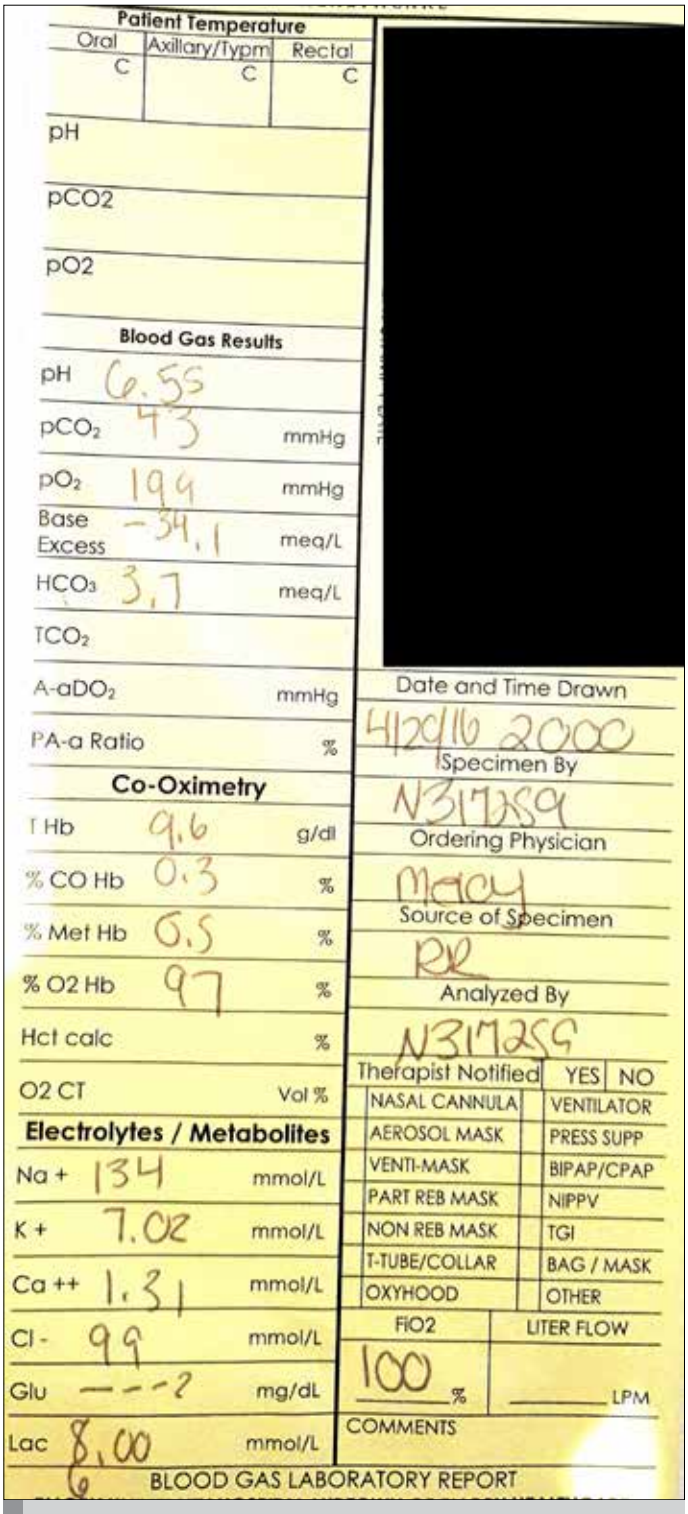

FIGURE 1. Original arterial blood gas report

We present a case of a 66-year-old female with known T2DM who presented to the emergency department with altered mental status and severe metabolic ketoacidosis with a pH below 6.6. She was successfully resuscitated and ultimately survived with no residual deficits.

Case Report

A 66-year-old female with a history of T2DM was brought to the emergency department by emergency medical services with a documented chief complaint of dehydration and altered mental status. Upon arrival, the patient was hypertensive (blood pressure 177/68 mmHg) and tachycardic (heart rate 111 beats per minute). She was oriented only to self and not following commands. The patient had pulled out her only peripheral IV en route to the hospital. Her initial point of care blood glucose was greater than 500 mg/dL. Given the patient's mental status and difficulty obtaining further peripheral access, a central subclavian line was placed. Volume resuscitation with a bolus of two liters normal saline, and an insulin infusion at 0.1 units/kg/hour were started for management of presumed DKA. After obtaining a computed tomography (CT) head scan that did not show any acute abnormalities, the patient's mental status continued to decline, and she was intubated.

Initial lab findings were significant for a profound metabolic acidosis with an arterial pH of 6.55 (Figure 1), hyperglycemia (963 mg/dL), hydroxybuterate level greater than 8mmol/L, hyperkalemia (7.6mmol/L), bicarbonate of 3.7 mEq/L, anion gap of 27 mEq/L, creatinine of 5.5 mg/dL, and a lactate of 5.25mmol/L. Serum ketones are not measured in our facility, although urine ketones were 4+. A 12-lead electrocardiogram was obtained, which indicated sinus tachycardia without obvious evidence of hyperkalemia.

Despite the initiation of volume resuscitation, the patient became hypotensive, which necessitated circulatory support with a norepinephrine infusion, started at 0.1 mCg/kg/min and titrated up to 1.0 mCg/kg/min. Hyperkalemia was managed with calcium chloride for cardiac membrane stabilization along with insulin infusion and a total of 50 mEq of sodium bicarbonate boluses initially. Acidosis was temporized with two further bolus injections of sodium bicarbonate, each 50 mEq, and another 2 liters of normal saline fluid boluses. A nephrologist was consulted for potential emergency dialysis, although this was ultimately declined by the family due to previous discussions with the patient.

Once intubated, started on insulin and norepinephrine infusions, and volume resuscitated, the patient was admitted to the medical intensive care unit (ICU). After 5 hours, she was transferred to the ICU, without any change in her acidosis despite aggressive care. A urine culture came back positive for Klebsiella, which was treated with levofloxacin for a total of 7 days. In addition to norepinephrine, vasopressin was temporarily added at 0.4 units/minute. Both vasopressors were weaned off on hospital Day 2, and the insulin drip was converted to basal and prandial insulin with an additional sliding scale. Acidosis was cleared on Day 2 before starting basal insulin.

The patient was successfully extubated on hospital Day 3. During her stay in the ICU, a psychiatrist was consulted, and the patient was started on fluoxetine for depression. A bed shortage prevented the patient from transferring to the floor until hospital Day 5 after the emergency department admission. She was ultimately discharged from the hospital medicine service on Day 9. Unfortunately, the same patient had three subsequent admissions to the ICU for repeat episodes of DKA in the 6 months following this episode.

Of note, patient information is confidential, and no identifying information can be found in this text.

Discussion

Severe diabetic ketoacidosis in patients with known T2DM is a recognized but relatively rare condition, being much less common for type 2 diabetics than hyperosmolar hyperglycemic nonketotic states. Our patient presented with severe DKA, as evidenced by her critically low pH (6.55) and severe metabolic acidosis (anion gap 27, bicarbonate of 3.7 meq/L) and ketosis. She had a markedly high glucose level, initially measured at 960 mg/dL and peaking at 1,152 mg/dL. Previous literature has shown that the level of hyperglycemia is often independent of the severity of the ketoacidosis (7). This case is notable for the patient's extreme acidemia, one of the lowest recorded in the literature for a type 2 diabetic who survived to hospital discharge. Her resuscitation adhered to established treatment pathways consisting of intravenous fluid, insulin infusion, eventual potassium repletion, and norepinephrine for hypovolemic shock due to dehydration. Calcium chloride was added for the initial severe hyperkalemia. She also received three doses of sodium bicarbonate for her severe academia, although the efficacy of supplemental bicarbonate in DKA has been questioned by recent literature (8).

The likely precipitating factor in our patient's acute decline was a combination of failing to take her insulin as prescribed and urinary tract infection. She likely developed a hyperglycemic hyperosmolar state, which led to an osmotic diuresis, and subsequent concomitant ketosis and lactic acidosis. Acidemia is an infrequent but real cause of altered mental status (8). To date, there have been no studies looking at the long-term morbidity or mortality in patients with T2DM who present with DKA. However, research has shown a higher morbidity and mortality in T2DM with DKA compared to type 1 diabetes mellitus, likely secondary to the older average age and comorbidities (9).

Conclusion

Despite having one of the lowest reported pH levels in a T2DM patient with DKA, aggressive early resuscitation in the emergency department, coupled with the established DKA treatment regimen, led to our patient's remarkable recovery.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of the Department of Emergency Medicine, Emory University School of Medicine.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

Peer-review: Externally peer-reviewed.

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Conflict of Interest: No conflict of interest was declared by the authors.

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