Dawit Kebede, Munib Khalid, Joseph Huang, Charles B. Sherman. *Ethiop Med J, 2020, Vol. 58, No. 3*

CASE REPORT

AN EXTREME CASE OF LIFE THREATENING METABOLIC ACIDOSIS AND DIABETIC KETOACIDOSIS

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ABSTRACT

Metabolic acidosis is one of the common manifestations of diabetic ketoacidosis (DKA). Concurrent hypokalemia often complicates management and mandates correction before administration of insulin. We report a unique case of a young woman with extreme life-threatening metabolic acidosis (pH 6.57) and hypokalemia due to DKA who survived without any sequelae.

Key words: *Severe metabolic acidosis, diabetic ketoacidosis, hypokalemia*

INTRODUCTION

Diabetic ketoacidosis (DKA) is a potentially life threatening complication of diabetes (DM) and is commonly associated with significant morbidity and mortality. Infections and drug discontinuation/under dosing are the most common precipitating factors. A triad of uncontrolled hyperglycemia, metabolic acidosis, and ketosis is the hallmark of DKA (1). Treatment relies on fluid and insulin administration, and correction of resulting metabolic acidosis and electrolyte abnormalities.

We report a case of a young woman with severe metabolic acidosis due to DKA who survived with no neurological sequelae. To our knowledge, such life threatening metabolic acidosis associated with DKA and

CASE SUMMARY

A 16-year-old female patient with known Type 1 DM on NPH insulin (60 IU AM and 50IU PM) for 8 years presented with complaints of worsening of fatigability, vomiting, watery diarrhea and abdominal pain of 02 days duration. Her physical examination was evident for a semi-comatose patient with Glasgow Coma Score (GCS) of 6/15, dehydration, and deep sighing respirations. Her vital signs included a respiratory rate of 34 breaths/min, pulse rate of 110 beats/min, axillary temperature of 36.7^{o}C and blood pressure of 130/70mmHg.

Initial laboratory values are shown in Table 1. These confirmed severe DKA with severe meta-

Table 1: Results initial Laboratory Investigations

Random Blood Sugar	>600mg/dl
Sodium	157 mmol/L (135-145
Potassium	2.6 mmol/L (3.5-5.1)
Chloride	135 mmol/L (97-111)
Calcium (Ionized)	4.6 mg/dl (4.2-4.9)
Urea	25 mg/dl (10 - 50)
Creatinine	1.5 mg/dl (0.5-1.3)
Phosphorus	5 mg/dl (2.5-5.0)
Urinalysis	Glucose 3+
	Protein 1+
	Ketone 3+
White blood cell count	7800/ul (4,000-11,000)
% Polys	64%
Hempglobin	12 gm/dl (12.0-15.5)
Platelets	158,000/ul (150,000-450,000)

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Concomitantly, her condition was complicated with multiple electrolyte abnormalities (hypokalemia and hyperchloremia) and pre-renal azotemia. The patient was initially treated in the Emergency Department (ED) with 5 liters of normal saline over 16 hours. Insulin was not started as the serum potassium level was below 3.3mmol/L, the cut-off level where insulin administration is contraindicated until potassium correction has occurred (1, 2, 3). She was transferred to the medical intensive care unit and treatment continued. However, there was again a delay in starting insulin due to the ongoing degree of hypokalemia and the unavailability of potassium chloride (KCL) for replacement.

Unfortunately, her mental status further deteriorated (GCS 3/15) and she was intubated and mechanically ventilated. The team then decided to start intravenous regular insulin infusion despite the hypokalemia; eventually potassium replacement was started once KCL was obtained. Ringer's lactate and later a D5W infusion were also given. Hypernatremia developed during the course of her treatment and was appropriately managed. Her metabolic derangements normalized within 36 h (Table 2), after which the patient was extubated and transferred to the general ward. She was discharged three days later with no sequelae, and normalization of all her chemistries.

Table 2: Changes in Laboratory Investigations from Admission to Time of Extubation

	Admission	**Time of Extubation**
RBS	>600mg/dl	278mg/dl
Na+	157mmol/L	162mmol/L (135 – 145)
K+	2.6mmol/L	2.9mmol/L (3.5 - 5.1)
Cl-	135mmol/L	130mmol/L (97 – 111)
HCO3-	1.9 meq/L	15.5 meq/L (21.0 – 28.0)
Ca+ (Ionized)	4.6 mg/dl	4.8 mg/dl (4.2-4.9)
pH	6.57	7.30
PCO2	15.8 mmHg	29.8 mm Hg
PO2	140 mmHg on 3 -5L/ min supplemental oxygen	89.5 mmHg on FiO2 0.4
Lactate	0.66 mmole/L	0.92 mmol/L (0.56 – 1.39)

DISCUSSION

To date, severe metabolic acidosis to this degree associated with a favourable outcome has not been reported. In fact it is quite unusual that our patient survived as it is known that severe acidemia at presentation (arterial blood pH <7.0) and development of coma are important predictors of mortality (4). There has been only one other similar case in the literature, a patient with an initial pH of 6.74 who survived without complications. This patient was reported to have type 1 diabetes with severe hyperglycemia, sepsis, vasoplegia, acute kidney injury, coma, and metabolic and respiratory acidosis from DKA (5). On the other hand there were a few case reports of severe metabolic acidosis, not as severe as the case reported here who recovered, from other causes who had favourable neurologic recovery (6, 7).In general, DKA results from a decrease in the net effective action of circulating insulin with an increase in glucagon, epinephrine, cortisol, and stress hormone.

This results in increased production of nonesterified fatty acids (NEFA) and glycerol from the breakdown of triglycerides. Glycerol is a substrate for gluconeogenesis and the greater amounts of NEFA results in the production of ketone bodies. Clearance of ketone bodies is impaired by low insulin concentrations, increased glucocorticoids, and decreased peripheral glucose utilization. Metabolic acidosis then develops due to the limited buffer capacity of bicarbonate (1, 4, 8).

In addition, there is often a concurrent respiratory acidosis as was seen in our patient. There are several possible explanations for this finding. The first is simultaneous failure of tissue perfusion resulting in CO_2 retention. Another explanation is the development of circulatory overload and hydrostatic pulmonary edema due to the acute shift of fluid from the intracellular into the extracellular compartment.

The third plausible mechanism is nonhydrostatic pulmonary edema resulting directly from DKA. And finally, diabetes mellitus can alter the structure and function of the lungs. Reduced lung volumes, reduced pulmonary elastic recoil, and reduced capillary lung capacity are well known functional changes that can occur. These underlying abnormalities may cause clinical lung dysfunction under stressful conditions such as DKA (4, 5, 9, 10).

Our patient had severe hypokalemia at presentation, which persisted beyond correction of her acidemia. Liberal and early insulin administration was hindered by her hypokalemia and contributed to the severity of her acidosis and clinical deterioration. The possible reasons for her ongoing hypokalemia included gastrointestinal loss, and fluid and insulin administration (5). Despite the general teaching to hold insulin if the potassium of <3.3mmol/l, we decided to administer insulin early on as her DKA was so severe (coma, pH < 7mmol/l, bicarbonate < 10mmol/l, glucose >250mg/dl, AG >12, urine ketone positive) (6).

Conclusion

To our knowledge, this is the first case report of a patient presenting with such severe metabolic acidosis (pH 6.57) due to DKA who ultimately survived without any long-term sequelae. We believe that the initial lack of resources in diagnostics and treatment (i.e., lack of ABG's in the ED and limited availability of KCL) worsened her condition, but ultimately did not affect her outcome.

Furthermore, management protocols adopted from resource-rich countries need to be modified to address the clinical reality of resource-poor nations. There should be a less stringent and an individualized treatment approach in DKA patients as restrictive criterion like delaying insulin administration in the presence of a potassium level below 3.3mmol/L could result in more extreme complications in those settings.

Competing Interests: The authors whose names are listed above certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript. This case was previously presented at the American Thoracic Society International Conference in San Diego, May 2018 (American Journal of Respiratory and Critical Care Medicine 2018;197:A3375).

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ACKNOWLEDGMENT

We would like to pass our gratitude to East African Training Initiative (Addis Ababa University), Vital Strategies and the Swiss Lung Foundation.

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