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Case Report

Euglycemic Ketoacidosis in a Patient with Prolonged Malnutrition Syndrome

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Abstract

We present the case of a 53-year-old woman admitted to the emergency department for vomiting and abdominal pain, ultimately diagnosed with euglycemic ketoacidosis. The patient had a history of severe malnutrition associated with depressive disorder, with almost exclusive fruit intake for at least 2 months. Evaluation revealed high anion gap metabolic acidosis, significant ketonemia, but normal blood glucose levels. Exogenous intoxication and diabetes mellitus were excluded. Fluid resuscitation and bicarbonate led to clinical and laboratory improvement. This case highlights the importance of considering malnutrition as a cause of ketosis and metabolic acidosis.

Keywords: Diabetic Ketoacidosis, Ketosis, Malnutrition, Acidosis, Metabolic, Malnutrition/complications, Anorexia

INTRODUCTION

Euglycemic ketoacidosis (EKA) is a rare subtype of high anion gap metabolic acidosis, characterized by elevated serum ketones in the absence of significant hyperglycaemia (blood glucose <250 mg/dL) [1]. Although frequently associated with sodium-glucose co-transporter 2 inhibitors (iSGLT2) [2], EKA may also arise in the context of prolonged fasting, malnutrition, pregnancy, alcoholism, or acute illness [3,4].

The pathophysiology of EKA involves reduced insulin activity and increased counter-regulatory hormones, leading to enhanced lipolysis and hepatic ketogenesis [1]. In malnourished individuals, depleted glycogen reserves and impaired gluconeogenesis may exacerbate ketosis and acid-base imbalance [4]. Because hyperglycaemia is absent, EKA may be misdiagnosed or delayed, hindering appropriate treatment.

We describe a case of EKA in a non-diabetic patient with severe dietary restriction due to depressive illness, highlighting the need to consider nutritional status when evaluating metabolic acidosis.

CASE

We present the case of a 53-year-old woman, previously independent in her activities of daily living, with a history of adjustment disorder with depressive features, thyroid nodules (with normal thyroid function), and breast nodules under surveillance. She had no known history of diabetes mellitus. Her regular medications included esomeprazole, atorvastatin, sertraline, trazodone, as-needed ethyl loflazepate, an oral contraceptive, and occasional paracetamol, which she denied having taken recently. The patient reported no regular alcohol consumption, smoking, or illicit drug use. She described a restrictive diet over the past five months, predominantly fruit-based, resulting in significant weight loss (~15 kg - BMI 22.4 kg/m^2 when admitted to the floor), in the context of anorexia linked to a depressive episode triggered by her partner's prolonged hospital admission. She presented in the emergency department with lower abdominal pain, nausea, and recurrent postprandial vomiting, accompanied by complete oral intolerance, beginning the previous day. She also reported a single episode of low-grade fever and altered bowel habits with soft stools, without other associated symptoms.

Table 1. Arterial blood gas and biochemical results indicating severe high–anion gap metabolic acidosis consistent with ketoacidosis.

Test	Reference Range	Patient Value
Arterial Blood Gas (ABG)		
pH	7.35 – 7.45	7.066
pCO_2 (mmHg)	35 – 45	12.6
HCO_3^- (mEq/L)	22 – 26	3.6
Anion Gap (mEq/L)	16 ± 4	27
Lactate (mmol/L)	0.5 – 2.5	0.3
K^+ (mEq/L)	3.5 – 5.3	5.4
Biochemistry		
Glucose (mg/dL)	<140	109
Creatinine (mg/dL)	<1.1	0.99
BUN (mg/dL)	6 – 20	25.43
iPhosphate (mg/dL)	2.5 – 4.5	1.8
AST (U/L)	<40	57
Additional Tests		
Ketonemia (mmol/L) (POCT)	<0.6	>3

ABG, Arterial Blood Gas, pCO_2; Partial pressure of carbon dioxide, HCO_3^-; Bicarbonate, K^+; Potassium, BUN; Blood Urea Nitrogen, iPhosphate; Inorganic Phosphate, AST Aspartate Aminotransferase, POCT; Point-of-Care Testing.

On examination, she was conscious, cooperative, and oriented, with no neurological deficits but clinical signs of dehydration. She was afebrile (36.2 ºC), normotensive (BP 118/82 mmHg), in sinus rhythm (HR 90 bpm), eupnoeic, and maintaining an oxygen saturation of 99% on room air. Abdominal examination revealed generalized tenderness without peritoneal signs.

Laboratory investigations showed a blood glucose level of 109 mg/dL, ketonemia >3 mmol/L (point-of-care testing taken hours after admission, once the possibility of de differential diagnosis of ketoacidosis, the initial value is supposed to be significantly higher), and urinary ketones of 150 mg/dL. Arterial blood gas analysis revealed severe metabolic acidosis with an increased anion gap: pH 7.066, HCO_3^- 3.6 mmol/L, pCO_2 12.6 mmHg, calculated anion gap of 27. There was no lactic acidosis (lactate 0.3 mmol/L). Renal function was preserved (creatinine 0.99 mg/dL), although blood urea nitrogen (BUN) was elevated at approximately 19.3 mg/dL (urea 54.5 mg/dL). She also had hypophosphatemia (1.8 mg/dL), mildly elevated potassium (5.4 mmol/L), and liver function tests were unremarkable, except for a mild increase in AST (57 U/L). Toxicology screening revealed negative levels for paracetamol and ethanol. Methanol and ethylene glycol were not measured due to a lack of clinical suspicion. Full blood count showed leucocytosis with neutrophilia (13.200/µL [normal range 4.000-11.000/µL]) and a haemoglobin level of 13.7 g/dL (normal range for adult females between 12.1-15.1g/dL). **Table 1** presents all relevant lab results, and **Figure 1** demonstrates the evolution of arterial blood gas parameters throughout our approach.

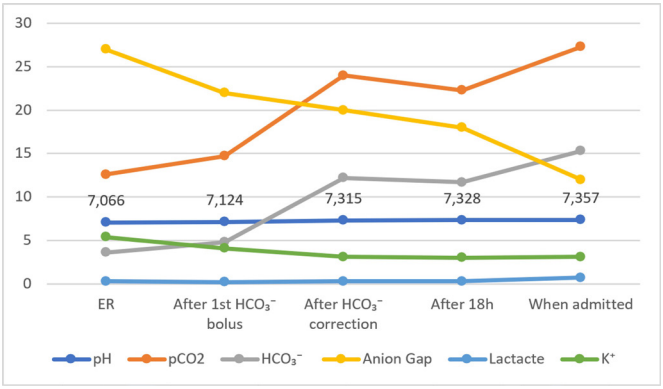

Figure 1. Evolution of arterial blood gas parameters

MANAGEMENT AND OUTCOME

Given the combination of significant ketonemia, severe metabolic acidosis with increased anion gap, and normoglycemia, a diagnosis of euglycemic ketoacidosis in the context of prolonged malnutrition was established. Treatment was initiated with intravenous solution 8.4% sodium bicarbonate (a total of 300 mL) to tamper the initial acidosis detected alongside targeted electrolyte correction (mild hyperkalemia [5.4 mmol/L] with no electrophisiologic repercussions, treated with inhaled salbutamol (2.5 mg nebulized every 4 hours) and intravenous insulin (2 units/hour) in 5% dextrose in normal saline to shift potassium intracellularly while preventing hypoglycemia. Potassium levels were monitored every 6 hours, and treatment was adjusted accordingly. Hypophosphatemia (1.8 mg/dL) detected in a second moment is a hallmark of refeeding syndrome and was .promptly corrected with intravenous potassium phosphate (0.08-0.16 mmol/kg infused over 6 hours). Phosphate levels were monitored every 6 hours during refeeding, with adjustments made to maintain levels above 2.5 mg/dL and prevent complications of severe hypophosphatemia, such as cardiac arrhythmias or respiratory failure, concomitantly with initiation of nutritional support. The patient was monitored by the Nutrition team that adjusted dietary supplements and diet troughout her stay. A psychiatric evaluation was requested to assess her psychopathologic state. Given the patient's history of adjustment disorder with depressive features and restrictive eating habits, the psychiatric evaluation focused on identifying potential underlying mood disorders or eating disorders that contributed to her malnutrition. The evaluation suggested IRSS as well as inclusion in a psychotherapy session for both grief support and eating disorders, recognizing that addressing these psychological factors is crucial for preventing relapse and promoting long-term recovery. Her symptoms improved within the first 18 hours, allowing the introduction of a light oral diet. She was discharged following metabolic stabilization, with referral for multidisciplinary follow-up in Nutrition and Psychiatry outpatient clinics.

DISCUSSION

Euglycemic ketoacidosis (EKA) is a rare and frequently underdiagnosed clinical entity, characterized by metabolic acidosis with increased anion gap, ketonemia or ketonuria, and normoglycemia (glucose <250 mg/dL) (1). While it is typically associated with sodium-glucose co-transporter 2 inhibitors (iSGLT2), it can also occur in prolonged fasting, malnutrition, alcoholism, pregnancy, or other catabolic states (2-5). Table 2 shows the causes for metabolic acidosis with elevated anion gap and Table 3 proposes a mnemonic for those causes.

In this case, the absence of diabetes or iSGLT2 suggests that EKA was not related to DM. The pathophysiology is similar to starvation ketoacidosis, where glucose restriction leads to increased lipolysis and hepatic ketogenesis, along with depletion of endogenous insulin (4). Normal glycaemia may delay recognition of the underlying acid-base disorder. In chronically malnourished individuals, reduced hepatic glycogen stores and hormonal imbalances can exacerbate the risk of ketosis (4).

The differential diagnosis of high anion gap metabolic acidosis includes potentially life-threatening causes such as toxic ingestions (methanol, ethylene glycol, salicylates, paracetamol), lactic acidosis, and renal failure (5). In this case, the absence of exposure history, preserved renal function, and negative toxicology results rendered these less likely. Normal lactate levels and haemodynamic stability ruled out type A lactic acidosis (6).

Current literature supports a structured approach to high anion gap acidosis using integrated diagnostic algorithms. Key elements include clinical history, calculation of the anion (and osmolar) gap, and selective toxicology screening (7). Management focuses on halting ketogenesis and correcting fluid and electrolyte disturbances. Administration of glucose and bicarbonate in more severe cases is effective in resolving EKA, as observed in this patient (8). Refeeding syndrome is a serious metabolic complication that can occur in these cases. It is characterized by rapid shifts in fluids and electrolytes, particularly phosphate, potassium, and magnesium, due to increased insulin secretion triggered by carbohydrate intake. These changes can lead to cardiac, respiratory, and neurologic complications (9,10).

Prevention involves identifying at-risk individuals, initiating refeeding with a low caloric intake, and closely monitoring and correcting electrolyte imbalances, especially phosphate. Thiamine should be administered before starting nutritional support (11,12). Additionally, a brief psychiatric evaluation during hospitalization is advisable in malnourished patients, particularly those with eating disorders or psychosocial stressors, to support comprehensive and multidisciplinary care (13).

This case underlines the importance of considering EKA in patients with non-specific symptoms, normoglycemia, and metabolic acidosis-especially in the context of malnutrition, prolonged fasting, or catabolic states. Ongoing nutritional and psychiatric follow-up are essential to prevent recurrence and support recovery.

Table 2. Causes for Metabolic acidosis with elevated Anion Gap

Mechanism	Elevated Anion Gap
Increase production	Lactic acidosis (and D-Lactic acidosis – produced by intestinal flora)
	Ketoacidosis
	Diabetes mellitus
	Malnutrion
	Alcohol (acute poisoning)
	Ingestion
	Methanol
	Ethylene glycol and diethylene glycol (antifreeze)
	Salicylates
	Toluene (only when impaired renal function)
	Propyleneglycol (perfumes)
	Pyroglutamic acid (5-oxoproline) (acetaminophen metabolite)
Diminished renal excretion	Severe renal insufficiency (eGFR <15 to 20 mL/min/1.73 m^2)

Table 3. Causes for Metabolic acidosis with elevated Anion Gap

	Toxin/Clinical disorder	Accumulated acids
G	Glycols (Ethylene glycol, diethylene glycol, and propylene glycol)	Multiple organic and inorganic acids
O	Oxyproline (associated with chronic acetaminophen use)	5-oxyproline (or Pyroglutamic acid)
L	Lactic acid	Lactic acid
D	D-Lactic acid	D-Lactic acid
M	Methanol	Formic acid
A	Aspirin	Multiple organic acids
R	Renal Failure (uraemia)	Multiple organic and inorganic acids
K	Ketoacidosis	β-hydroxybutyric acid and acetoacetic acid

DECLARATIONS

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AI: Not Used

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