VOLUME - 14, ISSUE - 03, MARCH - 2025 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

General Medicine

"NON-DIABETIC KETOACIDOSIS TRIGGERED BY ACUTE PANCREATITIS: A RARE METABOLIC COMPLICATION"

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ABSTRACT Diabetic ketoacidosis (DKA) is a serious metabolic disorder primarily linked to uncontrolled diabetes mellitus. However, its occurrence in individuals without a prior diabetes diagnosis is uncommon and often secondary to other medical conditions. This report presents a rare case of DKA in a non-diabetic patient, triggered by acute pancreatitis. We explore the pathophysiological mechanisms, diagnostic challenges, and therapeutic considerations. Recognizing DKA as a potential complication in non-diabetic patients with pancreatitis is crucial for timely diagnosis and appropriate management.

KEYWORDS : Non-diabetic ketoacidosis, Diabetic ketoacidosis, Acute pancreatitis, Metabolic acidosis, Hyperglycemia

INTRODUCTION

Diabetic ketoacidosis (DKA) is a severe and potentially life-threatening complication commonly seen in individuals with type 1 diabetes mellitus (T1DM) and, occasionally, in type 2 diabetes mellitus (T2DM). It is characterized by hyperglycemia, ketonemia, and metabolic acidosis. While DKA is typically associated with underlying diabetes, rare cases have been reported in non-diabetic individuals due to factors such as acute pancreatitis, prolonged fasting, excessive alcohol consumption, or medication use.

Acute pancreatitis, an inflammatory condition of the pancreas, is known to cause transient hyperglycemia and insulin resistance. However, its role in precipitating DKA in non-diabetic patients is not well understood. Here, we present a unique case of a non-diabetic patient who developed DKA secondary to acute pancreatitis. This case highlights the need for clinicians to consider ketoacidosis in the differential diagnosis of metabolic acidosis in non-diabetic patients.

CASE PRESENTATION

Clinical History and Examination

A 45-year-old male with no known history of diabetes mellitus presented to the emergency department with severe epigastric pain radiating to the back, nausea, vomiting, and generalized weakness lasting two days. The patient reported recent heavy alcohol intake but denied prior episodes of hyperglycemia or pancreatitis.

Upon examination, the patient appeared dehydrated and tachycardic (heart rate: 110 bpm) with mild hypotension (BP: 95/60 mmHg). Abdominal examination revealed epigastric tenderness with guarding, while no clinical signs of chronic liver disease or neuropathy were observed.

Laboratory Investigations

Initial laboratory findings included:

Serum glucose: 290 mg/dL (normal: 70-140 mg/dL)

Arterial blood gas (ABG): pH 7.25, bicarbonate (HCO_3^-) 12 mmol/L, anion gap 20

Serum ketones: Positive

Serum amylase: 850 U/L (normal: 30-110 U/L)

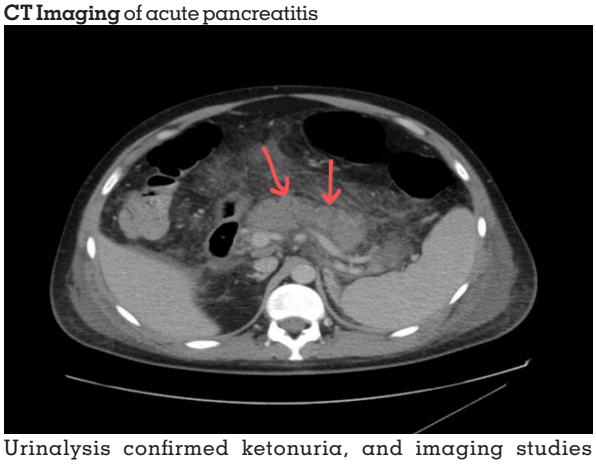
Serum lipase: 1200 U/L (normal: 23-300 U/L)

Serum lactate: 3.0 mmol/L (normal: <2.0 mmol/L)

HbA1c: 5.4% (normal: <5.7%)

C-peptide: 3.1 ng/mL (normal: 0.8-3.85 ng/mL)

CT Imaging of acute pancreatitis



Urinalysis confirmed ketonuria, and imaging studies (abdominal ultrasound and computed tomography) revealed pancreatic inflammation without gallstones or necrosis, consistent with acute pancreatitis.

DIAGNOSIS

Despite no prior diagnosis of diabetes and a normal HbA1c level, the patient met the criteria for DKA based on hyperglycemia, ketonemia, and high anion gap metabolic acidosis. Given the concurrent diagnosis of acute pancreatitis, a final diagnosis of non-diabetic ketoacidosis secondary to acute pancreatitis was made.

DISCUSSION

Pathophysiology of DKA in Non-Diabetic Patients

DKA primarily arises due to absolute or relative insulin deficiency, leading to increased lipolysis, ketone body production, and metabolic acidosis. While this is a hallmark of diabetes, its occurrence in non-diabetic individuals is rare and usually associated with physiological stress.

In acute pancreatitis, inflammatory mediators can impair

pancreatic beta-cell function, reducing insulin secretion. Additionally, systemic inflammation and counter-regulatory hormone activation (glucagon, cortisol, catecholamines) contribute to transient insulin resistance, promoting lipolysis and ketogenesis. Alcohol use can further exacerbate this process by inhibiting gluconeogenesis and increasing fatty acid metabolism.

Diagnostic Challenges

Distinguishing non-diabetic DKA from stress-related hyperglycemia in pancreatitis is challenging. Key distinguishing features include:

Severe metabolic acidosis with a high anion gap

Presence of ketonemia and ketonuria despite no prior diabetes diagnosis

Normal HbA1c and C-peptide levels, indicating no pre-existing chronic hyperglycemia

Clinicians must remain vigilant, as failure to recognize ketoacidosis in non-diabetic patients can lead to delayed or inadequate treatment.

Management And Clinical Course

The standard approach to treating DKA was followed, including:

- Fluid resuscitation with isotonic saline to correct dehydration and electrolyte imbalances
- Insulin therapy to suppress ketogenesis and normalize blood glucose levels
- Electrolyte replacement, particularly potassium, to prevent cardiac complications
- Supportive care for pancreatitis, including pain control and bowel rest

As the patient's ketoacidosis resolved, insulin therapy was tapered off. Blood glucose levels normalized without the need for long-term insulin treatment, confirming transient hyperglycemia rather than undiagnosed diabetes.

CONCLUSION

This case underscores the importance of recognizing non-diabetic ketoacidosis as a rare but serious complication of acute pancreatitis. Inflammatory stress, insulin resistance, and altered metabolism can contribute to ketoacidosis, even in patients without a history of diabetes. Early identification and appropriate management are critical for preventing complications and improving patient outcomes. Clinicians should consider ketoacidosis in non-diabetic patients presenting with metabolic acidosis and pancreatitis, especially those with additional risk factors such as alcohol use.

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