CASE REPORT

Incidental diagnosis of euglycaemic diabetic ketoacidosis: A case report

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Abstract

Euglycaemic diabetic ketoacidosis (eu-DKA) is a rare but serious complication of sodium-glucose co-transporter 2 (SGLT2) inhibitors, characterized by metabolic acidosis, elevated ketones and may be overlooked because of the absence of marked hyperglycaemia. We present the case of a 57-year-old male with well-controlled type 2 diabetes (HbA1c 6.5%), hypertension and squamous cell carcinoma of the tongue, admitted to Aga Khan University Hospital, Karachi, Pakistan for elective surgery. The diagnosis of euglycaemic DKA was incidental as the patient was asymptomatic and metabolic abnormalities were detected during preoperative laboratory evaluation. Investigations revealed high anion gap metabolic acidosis with ketonuria and a normal blood glucose level (73 mg/dL). He had been on SGLT2 inhibitors and had abstained from solid food intake for two weeks due to his cancer diagnosis. Management included intravenous fluids, insulin infusion and electrolyte monitoring. His metabolic parameters normalized within 8 hours and he was placed on an adjusted basal-bolus insulin regimen.

Keywords: Diabetic Ketoacidosis, DKA, Sodium Glucose Transporter 2 Inhibitors, SGLT-2 Inhibitors, Diabetes Mellitus, Type II.

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Introduction

Diabetic ketoacidosis (DKA) is a severe and potentially life-threatening condition that can affect individuals with both type 1 and type 2 diabetes mellitus. It is defined by a triad of hyperglycaemia, anion gap metabolic acidosis and elevated ketone levels in the blood.[1] On the other hand, euglycaemic diabetic ketoacidosis (eu-DKA) is marked by relatively lower blood glucose levels, typically under 200 mg/dl, which can lead to delayed diagnosis and treatment, increasing the risk of adverse metabolic outcomes.[1] Since the widespread adoption of sodium-glucose co-transporter 2 inhibitors (SGLT2i) in the treatment of type 2 diabetes mellitus, the occurrence of euglycaemic diabetic ketoacidosis (eu-DKA) has risen. Additionally, reduced caloric intake has also been linked to the onset of eu-DKA.[1,2] It can be overlooked if only clinical signs are considered, as it may not present with typical features of DKA, such as dehydration. Patients on SGLT2 inhibitors may experience less polyuria and polydipsia due to milder hyperglycaemia, instead presenting with symptoms like malaise, anorexia, tachycardia, or tachypnoea, sometimes without fever.[1] Furthermore, it can be misdiagnosed due to normal blood glucose levels delaying treatment. Prompt recognition of acidosis and serum ketone measurement are crucial for accurate diagnosis and management.[2]

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Case Report

A 57-year-old male with well-controlled type 2 diabetes mellitus (HbA1c=6.5%), hypertension and a recent diagnosis of squamous cell carcinoma of tongue was electively admitted to Aga Khan University Hospital, Karachi, Pakistan in January 2024 for surgical resection of the tumour. The patient reported no symptoms suggestive of metabolic decompensation, including nausea, vomiting, abdominal pain or dyspnoea. His medication regimen included a morning dose of empagliflozin/metformin 12.5/1000 mg and an evening dose of sitagliptin/metformin 50/850 mg.

Upon presentation, the patient was awake, alert, and oriented, with a noticeable mass on his tongue. His vital signs were normal, with a blood pressure of 120/70 mmHg and a pulse rate of 72 beats per minute. The remainder of the systemic examination was unremarkable.

As part of the preoperative workup, laboratory tests were sent and revealed the following: a normal complete blood count (CBC), an electrolyte panel showing a low bicarbonate level of 12.2 mEq/L, and a high anion gap of 22.8 (Na: 132mm/L, Cl:97 mm/L). Arterial blood gas analysis indicated a pH of 7.38 and a pCO_2 of 30, with a normal blood glucose level of 73 mg/dl (Table 1). Although the arterial pH was within the normal range, the markedly reduced bicarbonate level and elevated anion gap were consistent with a primary metabolic acidosis with compensatory respiratory alkalosis. Urinalysis detected ketonuria of 2+.

Upon further investigation, he admitted to abstaining from solid food intake for the past two weeks, though he

Table-1: Metabolic Panel.

Laboratory test	Before treatment	After 8 hours of treatment	Reference Range
Random blood glucose	73	112	80-160 mg/dl
Sodium	132	130	136-145 mmol/L
Potassium	4.3	3.9	3.5-5.1 mmol/L
Chloride	97	102	98-107 mmol/L
Bicarbonate	12.2	20.9	
Venous blood gas			
pH	7.38	7.45	7.35-7.45
$PaCO_2$	30	32	35-48 mmHg
HCO_3	15	22	22-28 mmol/L
Anion gap	22.8	7.1	4-12 mmol/L

pH: Potential of Hydrogen; $PaCO_2$: Partial Pressure of Carbon Dioxide; HCO_3: Bicarbonate

continued to consume liquids and maintained his oral medications. Additionally, the patient denied any history of methanol, ethanol, or paraldehyde ingestion.

Based on these laboratory findings, the endocrinology team was taken on board and the diagnosis of euglycaemic diabetic ketoacidosis (eu-DKA) was made, attributed to the use of the SGLT-2 inhibitors and reduced oral intake.

Oral hypoglycaemic medications were discontinued and electrolyte levels were closely monitored. The patient was treated with an intravenous infusion of Dextrose 5% (D5W) at 150 cc/hour and insulin infusion was initiated at a rate of 0.05 unit/kg/hour.[3,4] Blood glucose values remained between 70–120 mg/dL during insulin infusion, necessitating careful titration to avoid hypoglycaemia. After 8 hours, his electrolyte levels improved (Na: 130 mmol/L, Cl: 102 mmol/L), with a bicarbonate level of 20.9, normalized blood gas with a pH of 7.45 and closure of the anion gap (Table 1). Insulin infusion was stopped after 4 hours and changed to a subcutaneous basal-bolus regime which included 8 units of insulin Glargine.

The patient experienced no complications. Due to the risk of recurrence, the patient's surgery was postponed for one week. Upon discharge, he was prescribed insulin glargine 6 units once a day and Insulin Lispro 2 units AC, while the SGLT2 inhibitors were discontinued indefinitely. One week later, during a follow-up clinic visit, his electrolyte levels were within normal range, and his anion gap had normalized.

Discussion

This case underscores the importance of recognizing eu-DKA as a potential incidental finding, particularly in asymptomatic patients undergoing preoperative evaluation.

SGLT2 inhibitors lower glucose by blocking its reabsorption in the kidneys, reducing insulin levels, increasing glucagon secretion and promoting lipolysis, leading to increased ketogenesis.[5] Hypoglycaemia is a well-known complication of DKA treatment that poses acute neurological risks, including coma and seizures and long-term consequences like cognitive decline and increased stroke rates with repeated episodes. Jordan Sell et al. observed significantly higher rates of hypoglycaemia (blood glucose <70 mg/dL) in patients with eu-DKA compared to hyperglycaemic DKA, with a trend toward more severe hypoglycaemia (blood glucose <54 mg/dL).[6] This finding highlights the need for careful glucose monitoring and potential adjustments to standard DKA treatment protocols in patients with eu-DKA. Hypoglycaemia also heightens the risk of life-threatening arrhythmias due to abnormalities in cardiac depolarization and repolarization, further emphasizing the necessity of avoiding this adverse event.[6] This was particularly relevant in our case where the patient already presented with a blood glucose level of 73 mg/dL, necessitating extremely cautious administration of insulin to prevent further hypoglycaemia during treatment.

Several case reports have documented instances of euglycaemic diabetic ketoacidosis (eu-DKA) triggered by carbohydrate-restricted diets in patients taking SGLT-2 inhibitors.[5] A dietary change, particularly reduced carbohydrate intake, shifts metabolism towards fat utilization for energy, which increases ketone production. This shift can contribute to the development of diabetic ketoacidosis (DKA), especially under stressful conditions.[7] In such clinical contexts, starvation ketosis represents an important differential diagnosis, especially in patients with prolonged fasting or malignancy-related cachexia. However, starvation ketosis is typically associated with mild ketonaemia and minimal metabolic acidosis with bicarbonate levels mostly higher than 18 mEq/l, whereas eu-DKA is characterized by marked anion gap metabolic acidosis that necessitates insulin therapy.[8] In our patient, the presence of a significantly elevated anion gap and substantially reduced bicarbonate level, despite normal glucose concentrations, favoured a diagnosis of eu-DKA rather than starvation ketosis. The continued use of SGLT-2 inhibitors in the setting of two weeks of markedly reduced oral intake, likely amplified ketogenesis and contributed to the development of eu-DKA.

A retrospective study by Stamatiades et al. found no significant differences in the precipitating factors or clinical presentations between patients with eu-DKA and those with hyperglycaemic DKA (h-DKA). Most patients exhibited symptoms such as nausea, vomiting, and abdominal pain, with common triggers including inadequate oral intake and infections.[9] In a meta-summary of eu-DKA cases induced by SGLT2 inhibitors, Juneja et al. noted that gastrointestinal symptoms (nausea, vomiting, abdominal

pain) and respiratory symptoms (breathlessness) were most frequently reported. The study also highlighted infection as the most common precipitating factor, with most patients presenting with severe metabolic acidosis (median serum pH 7.14, bicarbonate 8.6 mmol/L) and relatively low blood glucose levels (median 184.5 mg/dL).[10] These findings contextualize our patient's asymptomatic but metabolically significant presentation.

Perioperative risk associated with SGLT2 inhibitors is increasingly recognized. Current recommendations by the American Diabetes Association Standards of Care, advise discontinuation most SGLT2 inhibitors (e.g., canagliflozin, dapagliflozin, empagliflozin) at least three days before elective surgery and ertugliflozin up to four days prior. Given that the half-life of SGLT2 inhibitors ranges from 11 to 17 hours, pharmacologic effects such as glucosuria and ketonaemia may persist for several days even after stopping the medication.[11,12] Institutional DKA protocols may require adaptation for eu-DKA, prioritizing ketone clearance over glucose reduction.

Conclusion

This case reinforces the need for heightened awareness of eu-DKA in patients receiving SGLT2 inhibitors, especially in the perioperative setting and in those with reduced caloric intake. Early recognition, protocol adjustment and multidisciplinary management are essential to prevent delays in diagnosis and treatment. Further evidence-based perioperative strategies are needed to optimize the safe use of SGLT2 inhibitors and prevent eu-DKA in surgical patients.

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Author Contribution:

AA: Concept, design, drafting, revision, final approval and agreement to be accountable for all aspects of the work.
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