How to manage a severe form of euglycemic diabetic ketoacidosis caused by SGLT2 inhibitors: a case report and literature review

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Abstract

The use of sodium-glucose cotransporter-2 inhibitors is becoming increasingly widespread not only for the treatment of diabetes mellitus, but also for heart failure and kidney disease. However, these medications can lead to very severe complications, sometimes even fatal, such as Euglycemic Ketoacidosis (EDKA). In this report, we present the case of a 79-year-old woman, affected by type 2 diabetes mellitus on insulin therapy and empagliflozin, who was admitted to our emergency department for a severe EDKA. Treatment with bicarbonates did not improve her condition worsening the hypokalaemia and complicating the administration of insulin. For this reason, we decided to administer octreotide subcutaneously with a rapid resolution of EDKA and an improvement in clinical conditions, demonstrating that octreotide can be an excellent therapeutic option for EDKA. Starting from our experience, we conducted a review of the relevant literature on this issue.

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Highlights

- *Sodium-Glucose Cotransporter-2 (SGLT2) inhibitors can cause a rare but fatal side effect known as Euglycemic Diabetic Ketoacidosis (EDKA);*
- *EDKA is an endocrine emergency characterized by elevated ketone bodies and metabolic acidosis despite normal or near-normal blood glucose levels that typically manifests with nausea, vomiting, malaise, or fatigue;*
- *Management is based on immediate discontinuation of the glifozine, hydration, insulin therapy and bicarbonates;*
- *The therapy is often difficult due to the presence of hypokalemia and a vicious circle between glucagon and insulin;*
- *Octreotide can be used to reduce insulin secretion and stabilize blood glucose levels without further insulin administration.*

Introduction

The use of Sodium-Glucose Cotransporter-2 inhibitors (SGLT2-i) is becoming increasingly common due to their role in managing diabetes and their pleiotropic effects on glomerular hyperfiltration and heart failure.[1,2] Unfortunately, despite their positive effects on reducing cardiometabolic risk, these medications can also cause severe urinary tract infections and in certain conditions such as sepsis, surgery, or fasting, they may lead to a serious condition known as Euglycemic Ketoacidosis (EDKA),[3] defined as the presence of the following: diabetic patients with normal or not particularly high glucose levels; development of high anion gap metabolic acidosis; ketonemia (>3.0 mmol/L) or significant ketonuria (2+ or more on standard).

EDKA usually develops in association with precipitating factors, including fever, infection, concomitant diseases, post-operative phase, reduced caloric and/or fluid intake, alcohol abuse, pregnancy, and a low-carbohydrate diet.[4] Beyond the prevention of precipitating factors, the real challenge for the emergency doctor is therapy: EDKA is complicated by the presence of hypokalemia and acidotic state. In those forms of DKA a sort of vicious circle amplified the effects of glucagon. Somatostatin therapy, in addition to standard therapy, could be useful for a rapid resolution of ketoacidosis and an improvement in the clinical condition.

Case Report

We report the case of a 79-year-old woman with permanent atrial fibrillation treated with rivaroxaban, and type 2 diabetes mellitus on insulin therapy and empagliflozin, who recently underwent right femur osteosynthesis surgery. The patient presented to our emergency room in a state of confusion, tending to be drowsy but responsive to verbal stimuli (GCS 12/15), experiencing diarrhoea and severe polypnea. On admission, her physical parameters were as follows: blood pressure of 90/50 mmHg, heart rate of 140 bpm, oxygen saturation of 97% in the room ambient, respiratory rate of 25 breaths/min, body temperature of 38° C, and diuresis of 800 mL of clear urine.

Blood Gas Analysis (BGA) showed a severe metabolic acidosis with pH 7.0, sodium 155 mmol/l (normal value 136-145), K 2.3 mmol/l (normal value3.5-5.1), HCO3 5 mmol/l (normal value 22-26), glucose 200 mg|dl (normal value 70-110), Cl 102 (normal value 98-106), calcium 8.4 (normal value 8.6-10.0) and an increased anion gap of 22. Presence of ketones in urine over 24 hours (200 mg). Laboratory tests showed high inflammation indices such as C-reactive protein (15.23 mg/dL, normal value 0.0-0.5) and neutrophil leukocytosis (10 000/mm^3, N 85 %), with normal renal and hepatic function.

Based on her medical history, we supposed that the surgical stress due to the recent orthopedic surgery, in the absence of suspension of glyphosate with prolonged fasting, were responsible for the onset of ketoacidosis.

The patient was admitted to our emergency medicine department and 50 mEq (5 vials 10 mEq/L) of sodium bicarbonate were infused followed by infusion of potassium chloride and loop magnesium sulphate in 250 mL of saline solution sing a pump set to 60 mL/h. Continuous hydration with saline solution 0.9% was provided at a rate of 100 mL/h. BGA was repeated every hour. Despite the potassium infusion, hypokalemia (K 1.9 mEq/L) and severe acidosis persisted (pH 7.20, HCO3 10 mEq/L). The coexistence of acidemia and hypokalemia is always a challenge for the physician, and insulin therapy makes management even more complicated.

We preferred Ringer's lactate saline, which, when infused, produces 40 mEq of bicarbonate, derived from lactate, which could further reduce serum potassium.

After a total infusion of approximately 3 Lt of saline solution 0.9% NaCl, BGA showed a very slight increase in serum bicarbonates (HCO3 11 mEq/L), pH 7.22 and K of 2.5 mmol/l. Therefore, two more vials of potassium chloride (60 mEq) and one vial of magnesium sulfate (10 gr) were infused. Despite all these treatment, no changes have been observed in the pH and potassium levels at serial BGAs. The clinical situation did not improve.

For all these reasons and starting from the experience by Torre and Coll,[5] we decided to administer SC octreotide, a peptide whose structure is similar to that of somatostatin, a hormone that is produced by the hypothalamus, the pancreas, and the gastrointestinal tract. It was synthesized in 1979 by the chemist Wilfried Bauer and was then marketed under the name Sandostatin®.[6] As reported in literature, somatostatin has been successfully used concomitantly with insulin as adjuvant in the treatment of diabetic ketoacidosis: the infusion of 100-500 mcg/hour of somatostatin, accompanied by the simultaneous IV administration of insulin (bolus of 10 IU + infusion of 1-4.8 IU/hour) has been shown to be capable of normalizing glycemic levels within 4 hours and resolving acidosis in 3 hours.

No other experience with octreotide described in literature except a case of cerebral oedema in a 10 years old boy with DKA published in 2010.[7]

We were the first to try in euglycemic ketoacidosis caused by glifozine it and the results were surprising: although the infusion of bicarbonates was suspended due to the persistence of hypokalemia, approximately 12 hours after starting the subcutaneous injection of octreotide at a dose of 0.1 mg/mL (100 micrograms) three times a day, pH improved to 7.24, and bicarbonate levels registered at 13.4 mmol/l, accompanied by the resolution of polypnea. After continuing somatostatin for another 24 hours, the pH rose to 7.32 and the HCO3 18.1 mmol/l. By the third day, the patient had fully regained cognitive functions and resumed oral nutrition. After 10 days of hospitalization, the patient was-discharged with a pH of 7.35 and bicarbonate levels of 25 mmol/l, with potassium levels returning to within normal limits (Table 1).

Discussion

SGLT2 is a high-capacity, low-affinity transporter encoded by the SGLT2 gene. It is primarily located in the S1 segment of the proximal tubule, near Bowman's capsule. This transporter reabsorbs nearly 90% of filtered glucose, operating on a c o-transport ratio of 1:1 for glucose and sodium. SGLT2 inhibitor drugs (SGLT2-i) induce an increase in glucose excretion and a reduction in blood glucose and glycated hemoglobin levels. They induce a glycosuria-dependent osmotic diuresis and natriuresis. This glycosuria leads to increased blood glucose values and a reduced sodium concentration in the renal tubules, which causes activation of the juxtaglomerular apparatus and the Renin-Angiotensin-Aldosterone System (RAAS).

Large randomized controlled trials have demonstrated the important and consistent benefits of the use of SGLT2-i in patients with type 2 diabetes mellitus, particularly in reducing mortality and Major Adverse Cardiac Events (MACE) in those with Atherosclerotic Cardiovascular Disease (ASCVD) or high/very high cardiovascular risk.[1] Alongside their cardioprotective effects, SGLT2-i drugs have also been shown to improve renal outcomes and prevent the progression of renal disease.[2] Furthermore, treatment with an SGLT2-i is associated with significant reductions in weight, systolic and diastolic blood pressure and uric acid levels.[3] In recent years, the efficacy of SGLT2-i drugs in reducing all-cause and cardiovascular mortality and hospital admissions for heart failure in patients with heart failure with reduced and preserved ejection fraction (HFrEF/HFpEF), regardless of the presence of type 2 diabetes, has been highlighted.[1]

Three SGLT2-i are authorized in the European Union: canagliflozin, dapagliflozin and empagliflozin, which can be used alone or in combination with metformin. Unlike insulin, the inhibitory effect of SGLT2-i disappears approximately two days after discontinuation of the medication, leading to increased renal reabsorption of glucose and reduced glycosuria. Patients may still experience glycosuria levels above 500 mg/dl for up to nine days after stopping treatment. All patients with DM2 should be educated about the importance of maintaining proper hydration and an adequate carbohydrate intake while using SGLT2-i. Clinicians should avoid the use of SGLT2-i to patients who cannot tolerate oral food intake, or in those with excessive weight loss or on a very low carbohydrate diet. Additionally, gliflozine should be suspended several days before any surgery procedure due to the risks of infection, operational stress, and potential fasting issues.

Despite their important benefits, SGLT2-inhibitors present some side effects. The most common adverse events include mild to moderate genital infections, especially in women and patients

with a history of such infections or predisposing factors, such as benign prostatic hypertrophy. These infections typically respond well to standard treatment. Furthermore, due to their diuretic effect, the use of SGLT2-i may cause volume depletion, mainly in elderly patients.

In addition, following the introduction of SGLT2-i use, some rare but serious cases of EDKA-have been described.[3-12]

EDKA was first described by Munro *et al.* in 1973,[15] EDKA is often misdiagnosed due to a serum glucose <250 mg/dL. It is associated with an anion gap metabolic acidosis and ketosis, and it most commonly occurs in patients with a history of low glucose states such as starvation, chronic liver disease, pregnancy, infection, and alcohol use.[12] Insulin deficiency, the increase in insulin counter-regulatory hormones (cortisol, glucagon, catecholamines) and insulin resistance cause hyperglycemia, dehydration, ketosis and electrolyte imbalance, all phenomena underlying the pathophysiology of DKA. For this reason the Food and Drug Administration (FDA) published a safety announcement of 73 cases of ketoacidosis in patients with type 1 diabetes or type 2 diabetes who were treated with SGLT2-i from March 2013 (the date the first drug in this class was released) to May 2015.[9] In many cases, EDKA was diagnosed later, as the increase in glucose levels was only mild and lower than typically seen in classic DKA. In February 2016, the European Medicines Agency (EMA) reaffirmed recommendations aimed at minimizing the risk of diabetic ketoacidosis in patients receiving SGLT2-I.

A fair amount of scientific evidence shows a correlation between EDKA and SGLT2-i In 2020, Menghoum *et al.*[10] described seven clinical cases of EDKA in patients treated with SGLT2-i, highlighting the challenges of management. In 2018, Karakaya *et al.*[12] emphasized the difficulty for emergency physicians to detect ketoacidosis in the presence of euglycemia, which may lead to the seriousness of the condition being overlooked. In 2019, Yii Ess *et al.*[11] illustrated a case of EDKA in a COVID19 patient. In 2020, Sampani *et al* [18] further stressed that the atypical presentation of this condition requires a high level of awareness among physicians, as early recognition can quickly and safely restore acid-base balance. In 2022, Branco *et al.*[28] evaluated 17 articles that described EDKA in patients undergoing perioperative cardiac surgery. The authors recommended preventive measures and management options, with a special emphasis on increasing clinical awareness among care teams regarding this complication.

SGLT2 inhibitor-induced EDKA is a medical emergency that can be difficult to identify in the postoperative surgical patient due to the overlap of signs and symptoms with other scenarios common in these patients. An SGLT2 inhibitor-associated reduction in EDKA may be mitigated by appropriate perioperative drug discontinuation.[28]

Chow *et al.*[19] published a landmark paper in 2023 classifying EDKA severity into mild, moderate, and severe based on serum pH and bicarbonate level.

A treatment algorithm is proposed to guide clinicians in the management of EDKA.[24] This treatment algorithm includes monitoring the anion gap and ketones to guide insulin and fluid management, and a slower transition to subcutaneous insulin to prevent relapse.

In 1979, Harano *et al.*[14] conducted a study to evaluate the effectiveness of somatostatin in treating DKA. Somatostatin is a polypeptide hormone produced by the hypothalamus, pancreas, gastrointestinal tract and cells of the APUD (Amine Precursor Uptake and Decarboxylation) system. It is synthetized in various locations in the body, particularly by the antral δ cells of the stomach, where it inhibits the gastrin-producing G cells to help regulate their function; in the hypothalamic-pituitary axis where it inhibits the secretion of Growth Hormone (GH), Thyroid-Stimulating Hormone (TSH), Adrenocorticotropic Hormone (ACTH) and prolactin; and in the δ cells of the pancreas, where it inhibits the release of insulin and glucagon as well as hydrochloric acid pro-

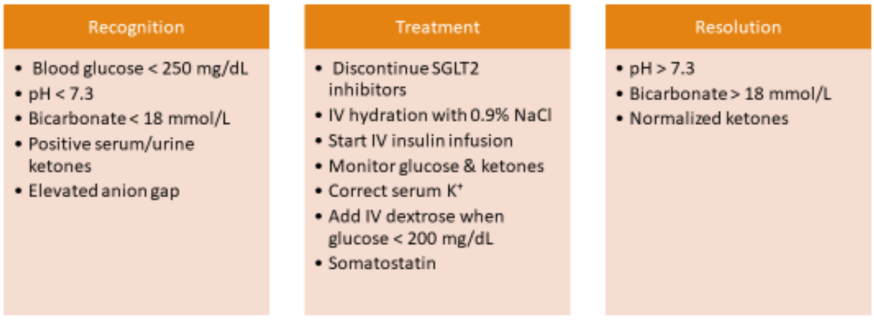

Figure 1. The management of Euglycemic Diabetic Ketoacidosis (EDKA).

Table 1. The table illustrates how, following the administration of octreotide, blood gas values — particularly bicarbonate and potassium levels, changed significantly at 12, 24, and 72 hours.

Blood gas analysis	Initial	3 h	12 h	12 h post Ocreotide	48 h post Ocreotide	72 h post Ocreotide
pH	7.0	7.21	7.24	7.32	7.34	7.4
HCO^3	5 mEq|l	10 mEq/l	11 mEq/l	13.4 mEq/l	14 mEq/l	18 mEq/l
pCO^2	23 mmHg	26mmHg	30mmHg	31 mmHg	38 mmHg	40 mmHg
Na	155mEq/l	152mEq/l	150mEq/l	148 mEq/l	146 mEq/l	134 mEq/l
K	2.3 mEq/l	1.9 mEq/l	2.5mEq/l	3.0 mEq/l	3.2 mEq/l	3.7 mEq/l
Ketones	X	X	x	x	0	0

duction in the stomach. It also suppresses the exocrine functions of the pancreas. Furthermore, somatostatin acts as a neurotransmitter and has a stimulating action on cholinergic and β-adrenergic receptors.

The most marked benefits seemed to occur in those forms of DKA where a sort of vicious circle amplified the effects of glucagon. In 2023, Torre *et al.*[5] described the clinical case of a 47-year-old patient with a severe form of EDKA. The patient was treated with continuous infusion of somatostatin, in addition to standard therapy, resulting in a rapid resolution of ketoacidosis and an improvement in the clinical condition.

The first and most important phase in the initial management of the patient with EDKA consists in the administration of 1/1.5 L of isotonic solution (0.9% saline or Ringer's Lactate) in the first hour. Subsequent administration of fluids must be commensurate with the patient's status (hydration, serum electrolytes, serum glucose, diuresis). IV insulin should be administered only after serum potassium levels have been accurately determined. In particular, insulin should not be administered when potassium levels are between >3 and 3.5 mEq/L. The use of bicarbonates does not improve the clinical picture, favouring hypokalemia; therefore, it is recommended only in cases of pH lower than 7. The prolonged presence of ketonuria above 80 mg/dl highlights a prolonged effect of glucagon with consequent advantage of the use of somatostatin in these euglycemic forms of DKA. In the case reported, the use of this drug speeded up the recovery time, reducing the ketogenic effects.

In 2024, Huang emphasized the measurement of acetone, which is produced through the spontaneous decarboxylation of acetoacetate and can be detected in expired air; however, there is currently no FDA-approved device for this measurement. Recently, a new technology has been introduced for measuring beta-hydroxybutyrate in interstitial fluid[20] . Monitoring ketone levels may be beneficial for assessing adherence to low-carbohydrate diets.

In patients with EDKA, potassium deficiency typically ranges from 2 to 5 mmol/l, even though serum potassium levels may appear elevated. This occurs because intracellular potassium shifts into the extracellular space due to a lack of insulin and high plasma osmolarity. Once insulin infusion begins, potassium moves back into the cells, resulting in a decline in serum potassium levels decline.

Potassium is osmotically active, so it should be infused with caution in patients with high serum osmolarity, for example 40 mEq of potassium in one liter of an isotonic solution. According to the current recommendations,[21] no potassium infusion should be given if the serum potassium level is above 5.5 mmol/L due to the risk of pre-renal acute kidney injury associated with severe dehydration. Potassium administration should be continued until the serum potassium level is less than 5.5 mmol/L and urine output is adequate. If the serum potassium level drops below 3.5 mEq/L, the potassium regimen should be reconsidered. Instead, ensure that potassium levels are above 3.3 mEq/L before administering insulin.

Some articles[14,20] have reported that the use of bicarbonate in patients with EDKA does not significantly affect the resolution of acidosis or the time to hospital discharge, and its use is generally discouraged. Bicarbonate administration is recommended only when the pH level is below 6.9, as a temporary measure to correct acidosis. When bicarbonate is infused it is advisable to administer intravenous calcium to prevent hypocalcemia. EDKA resolution is defined as pH >7.3, bicarbonate >15.0 mmol/L, and blood ketone level <0.6 mmol/L.

SGLT2-i can cause ketoacidosis through a different mechanism of action.[21] They reduce blood glucose levels by increasing the excretion of glucose in the urine, which in turn lowers insulin secretion from the pancreatic beta cells. As insulin levels drop, there is a decrease in insulin's anti-lipolytic activity. This reduction stimulates the production of free fatty acids, which are then converted into ketone bodies through β-oxidation in the liver. Additionally, SGLT2-inhibitors enhance the reabsorption of ketones in the renal tubules, leading to higher concentrations of ketone bodies in the body. It remains unclear whether SGLT2 directly influences glucagon secretion. The increase in glucagon is likely an indirect effect mediated by the reduction in insulin secretion, as there is no evidence to suggest a direct stimulation of the pancreatic alpha cells by SGLT2 inhibitors. This explains how somatostatin and its synthetic analogue can be very useful in unblocking this vicious cycle created between insulin and counter-regulatory hormones and allowing for better management of the risk of hypokalemia in an already highly acidotic patient. Figure 1 proposes a new EDKA therapy diagram.

Conclusions

In cases of severe EDKA, particularly when hypokalemia complicates management, somatostatin can be an effective treatment option. In in this specific case, we administered octreotide via subcutaneous injection obtaining surprising results within just 24 hours. To date, there are no similar experiences in the literature. Therefore, further studies are necessary to demonstrate the reliability of this therapy.

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