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PERIOPERATIVE EUGLYCEMIC KETOACIDOSIS DESPITE DISCONTINUATION OF SGLT2 INHIBITORS: A REVIEW OF REPORTED CASES

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ABSTRACT

Background: Sodium-glucose cotransporter 2 (SGLT2) inhibitors are increasingly used in the treatment of type 2 diabetes mellitus, heart failure, and chronic kidney disease. A rare but potentially life-threatening adverse effect is euglycemic diabetic ketoacidosis (euDKA), particularly in the perioperative setting.
Aim: To analyze reported cases of perioperative euDKA occurring despite preoperative discontinuation of SGLT2 inhibitors.
Material and Methods: A literature review of PubMed publications from February 2020 to January 2026 was conducted. Case reports and case series describing perioperative euDKA after prior discontinuation of SGLT2 inhibitors were included.
Results: Eight cases met the inclusion criteria. The duration of drug withdrawal ranged from 18 hours to 5 days before surgery. euDKA developed intraoperatively or in the early postoperative period and was characterized by high anion gap metabolic acidosis with normal or mildly elevated glucose levels. Six patients recovered, whereas two patients died.
Conclusions: Perioperative euDKA may occur despite adherence to current discontinuation guidelines. Ketone monitoring and heightened clinical vigilance are essential throughout the perioperative period.

KEYWORDS

SGLT2 Inhibitors, Perioperative Period, Surgery, Euglycemic Diabetic Ketoacidosis, euDKA

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1. Introduction

Sodium-glucose cotransporter 2 (SGLT2) inhibitors constitute an important class of medications with a broad range of clinical applications. Initially developed for the treatment of type 2 diabetes mellitus, they are now also used independently in heart failure due to their cardioprotective effects (in both reduced and preserved left ventricular ejection fraction), as well as in chronic kidney disease owing to their nephroprotective properties, including slowing disease progression and reducing albuminuria [1-3].

The primary mechanism of action of SGLT2 inhibitors involves inhibition of glucose and sodium reabsorption in the kidneys. By inhibiting the SGLT2 cotransporter, gliflozins increase urinary glucose excretion (glucosuria), resulting in reduced blood glucose levels in an insulin-independent manner. Additionally, increased excretion of sodium and water contributes to lowering blood pressure and reducing both preload and afterload of the heart [4-6].

One of the rare but serious adverse effects associated with SGLT2 inhibitor therapy is euglycemic diabetic ketoacidosis (euDKA) [5,7]. Euglycemic diabetic ketoacidosis is characterized by metabolic acidosis with elevated ketone body levels and normal or only moderately increased blood glucose concentrations. Unlike classical diabetic ketoacidosis, euDKA lacks marked hyperglycemia, which may delay diagnosis and increase the risk of severe clinical deterioration. The estimated incidence of euDKA is ≤1 per 1,000 patients per year [5,8].

Mechanism of euDKA in surgical patients

The perioperative period represents a high-risk clinical setting that predisposes patients to the development of euDKA due to metabolic stress, fasting, fluid restriction, and preoperative adjustments to insulin therapy. Reported cases of postoperative euDKA were among the primary reasons for implementing recommendations to discontinue SGLT2 inhibitors before surgical procedures. The incidence of euDKA has been estimated at approximately 0.17% in nonemergent procedures and 1.1% in emergent procedures [1,4,9-14].

The pathophysiological mechanism underlying euDKA in surgical patients treated with SGLT2 inhibitors is multifactorial. Inhibition of sodium-glucose cotransporter 2 (SGLT2) increases urinary glucose

excretion (glycosuria), resulting in reduced plasma glucose concentrations and relative euglycemia. Lower glucose levels, together with reduced insulin reserve and perioperative fasting, decrease insulin availability. Simultaneously, surgical stress stimulates the release of counterregulatory hormones, including glucagon, cortisol, and catecholamines. Relative insulin deficiency combined with excess counterregulatory hormones enhances lipolysis and ketogenesis, leading to high anion gap metabolic acidosis despite normal blood glucose levels [4,5,9].

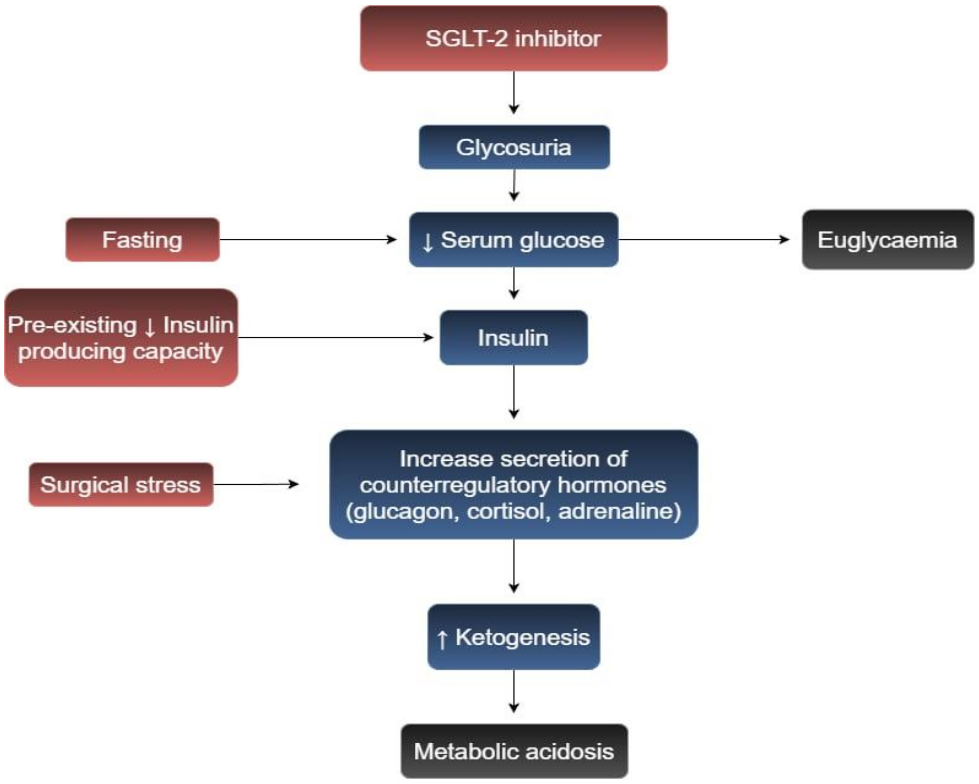

Fig. 1. *Mechanism of euDKA in surgical patients [4]*

There is significant variability in international recommendations regarding the perioperative management of SGLT2 inhibitors. Most commonly, it is recommended to discontinue empagliflozin, dapagliflozin, and canagliflozin for ≥3 days, and ertugliflozin for ≥4 days before elective surgery. However, these recommendations are largely based on pharmacokinetic data (Table 1) and case reports, as high-quality prospective studies are currently lacking [1,4,5].

Table 1. Summary of the available SGLT2 inhibitors and their elimination half-lives [4]

Drug name (generic)	Elimination half-life (t½)
Dapagliflozin	13 h
Canagliflozin	11 h
Empagliflozin	12 h
Ertugliflozin	17 h
Ipragliflozin	15 h
Luseogliflozin	14 h
Tofogliflozin	6 h
Sotogliflozin	20 h
Bexagliflozin	12 h

Therefore, the aim of this study was to analyze reported cases of perioperative euDKA occurring despite preoperative discontinuation of SGLT2 inhibitors and to identify potential clinical risk factors.

2. Material and methods

This review was conducted to identify and analyze reported cases of perioperative euglycemic diabetic ketoacidosis occurring despite prior discontinuation of SGLT2 inhibitors. A comprehensive literature search of the PubMed database was performed for articles published between February 2020 and January 2026. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords, including: "SGLT2 inhibitors", "perioperative period", "surgery", and "euglycemic diabetic ketoacidosis" or "euDKA", using Boolean operators (AND/OR). Eligible publications included case reports and case series published in English. Studies were included if they described perioperative euglycemic diabetic ketoacidosis in patients who had discontinued SGLT2 inhibitors prior to surgery. Eight cases meeting the inclusion criteria were identified and analyzed with regard to patient characteristics, type of surgery, timing of drug discontinuation, clinical presentation, and outcomes. Non-human studies and conference abstracts without full-text availability were excluded.

3. Results

Eight cases of perioperative euDKA occurring despite preoperative discontinuation of SGLT2 inhibitors are presented in Table 2, with corresponding biochemical data summarized in Table 3.

Table 2. Summary and clinical characteristics of patients

Case	Patient Summary	Procedure	SGLT2 inhibitor	Preoperative SGLT2 inhibitor discontinuation	Onset of euDKA	Outcome
1	82-year-old female; colon adenocarcinoma, HFrEF, CAD s/p staged PCI, CKD G3b, HTN, HLD	Elective robotic right colectomy	E	72 h	Intraoperative	Full recovery
2	50-year-old female; T2DM, leiomyosarcoma	Right upper lobectomy	E	75 h	Intraoperative	Resolved
3	60-year-old male; T2DM, CAD, HLD	Elective CABG	E	48 h	Postoperative	Resolved
4	68-year-old female; T2DM, hyperthyroidism	Elective laparoscopic distal pancreatectomy	D	24 h	POD 8	Resolved
5	61-year-old male; T2DM, mild obesity, undiagnosed CAD	Elective hip replacement surgery	E	< 24 h	POD 4 (initial symptoms); POD 5 (cardiac arrest)	Death
6	69-year-old female; T2DM, CAD, HTN, HLD, hypothyroidism, GERD	Elective CABG	E	5 days	POD 1 (initial diagnosis); recurrence on POD 3	Resolved
7	60-year-old male; T2DM, malignant melanoma with brain metastases	Elective craniotomy	E	< 24 h	Preoperative symptoms, intraoperative diagnosis	Resolved
8	Female in her 40s; T2DM, moyamoya disease	Elective cerebral revascularization	E	18 h	Postoperative	Death

Abbreviations: E - empagliflozin, D - dapagliflozin, HFrEF - heart failure with reduced ejection fraction, CAD s/p staged PCI - coronary artery disease status post staged percutaneous coronary intervention, CKD G3b - chronic kidney disease stage 3b, HTN = hypertension, HLD - hyperlipidemia, CABG - coronary artery bypass grafting, IV – intravenous, SC – subcutaneous, POD - postoperative day, T2DM – type 2 diabetes mellitus, CAD - coronary artery disease, GERD - gastroesophageal reflux disease.

Table 3. Biochemical data of patients

Case	Glucose (mg/dL)	β-hydroxybutyrate (mmol/L)	pH	Bicarbonate HCO_3^- (mmol/L)	Anion gap (mmol/L)	HbA1c (%)	Urine ketones
1	125	2.42	7.09	13	15	5.3	Not measured
2	197	-	7.132	13.7	-	-	3+
3	138	6.52	7.275	15	25	9.6	Not measured
4	168	-	7.072	2.3	25.7	7.9	4+
5	261	3.2	6.85	5.9	18.1	-	Not measured
6	146	7.9	7.30	17	22	8.2	Positive
7	147	3.1	7.30	18	20	-	Not measured
8	149	7.7	7.01	-	17	-	4+

"–" indicates data not available

Case descriptions:

CASE 1: An 82-year-old non-diabetic female (HbA1c 5.3%) was scheduled for an elective robotic right colectomy for colon adenocarcinoma. Her medical history included HFrEF (LVEF 25%) managed with empagliflozin, which was discontinued 72 hours prior to surgery. Following induction, intubation and incision, an intraoperative arterial blood gas revealed severe metabolic acidosis (pH 7.09, HCO_3^- 13 mmol/L) with an increased anion gap (15 mmol/L), despite near-normal glycemia (125 mg/dL). Laboratory tests confirmed euglycemic ketoacidosis with an elevated serum β-hydroxybutyrate (2.42 mmol/L). The patient was treated intraoperatively with intravenous insulin, dextrose and sodium bicarbonate infusions. Postoperatively, she was transferred to the ICU, where metabolic derangements resolved within a few hours (β-hydroxybutyrate decreased to 0.15 mmol/L). She was extubated without complications and discharged home on POD 4. Empagliflozin was not restarted to prevent recurrence [15].

CASE 2: A diagnosis of euglycemic diabetic ketoacidosis was established in a 50-year-old female during thoracic surgery, 75 hours after discontinuation of empagliflozin. She had been diagnosed with type 2 diabetes mellitus at the age of 36. Prior to surgery, she was treated with empagliflozin 10 mg/day, intensive insulin therapy with a total daily dose of 36 IU, as well as vildagliptin, metformin, and miglitol. The aim of the surgery was management of a ruptured pulmonary tumor causing chest pain and dyspnea. Intravenous glycemic control was used perioperatively, and all oral antidiabetic medications were discontinued. In the preoperative period, blood glucose levels remained <200 mg/dL, and ketone levels were not routinely assessed before surgery. During the procedure, arterial blood gas analysis revealed metabolic acidosis with decreased bicarbonate concentration, while blood glucose levels remained within the normal range. Only subsequent measurement of urinary ketones allowed the diagnosis of euglycemic diabetic ketoacidosis to be established. Continuous insulin infusion was initiated, leading to gradual normalization of acid–base balance parameters [16].

CASE 3: A 60-year-old male with T2DM (HbA1c 9.6%), CAD, and HLD was admitted for elective coronary artery bypass graft (CABG) surgery. Home medications included empagliflozin, which was discontinued 48 hours prior to surgery, with the patient transitioned to a subcutaneous insulin regimen. Admission laboratory results revealed normal bicarbonate and anion gap levels, although glucosuria and mild ketonuria were present. A few hours postoperatively, the patient developed an elevated anion gap metabolic acidosis (pH 7.275, anion gap 25 mmol/L) despite near-normal glycemia (138 mg/dL). Laboratory testing confirmed euDKA with a significantly elevated β-hydroxybutyrate level (6.52 mmol/L). Treatment was initiated with IV insulin and 5% dextrose infusions, along with transient norepinephrine for hypotension. The

ketoacidosis resolved within 48 hours, and the patient was successfully transitioned back to subcutaneous insulin. The remainder of the hospital course was uneventful, and the patient was discharged [17].

CASE 4: A 68-year-old female with T2DM (HbA1c 7.9%) and hyperthyroidism was admitted for an elective laparoscopic distal pancreatectomy due to a pancreatic body adenocarcinoma. Her home medication included dapagliflozin (10 mg/day) (which was discontinued 24 hours prior to surgery), metformin and glimepiride. The early postoperative course was uneventful, and dapagliflozin was restarted on POD 5. However, on POD 8, the patient developed acute tachypnea, tachycardia, dyspnea, and palpitations. Laboratory tests revealed near-normal glycemia (168 mg/dL) but severe high anion gap metabolic acidosis (pH 7.072, HCO_3^- 2.3 mmol/L, anion gap 25.7) and ketonuria (4+). The patient was diagnosed with euDKA and transferred to the ICU. Treatment followed the DKA protocol, including IV insulin infusion, IV fluids, and potassium supplementation. Her condition improved within 12 hours. After two days in the ICU, she was transitioned to subcutaneous insulin and later discharged home on her previous oral antidiabetic regimen. At 3-month follow-up, her glycemic control remained stable with no recurrence of euDKA [18].

CASE 5: A 61-year-old male with T2DM (treated with empagliflozin, metformin, and recently added long-acting insulin) and mild obesity underwent elective hip replacement surgery. Preoperative assessment was unremarkable. Empagliflozin and metformin were discontinued approximately 24 hours prior to surgery (on the evening before the procedure). The surgery was uneventful. On POD 2, his chronic doses of empagliflozin and metformin were resumed. On POD 4 and POD 5, the patient reported malaise and vomiting; however, blood gas analysis was not performed, as glycemia remained below 10 mmol/L (180 mg/dL). On the evening of POD 5, the patient suffered a sudden cardiac arrest (ventricular fibrillation) due to previously undiagnosed coronary artery disease, triggered by severe metabolic derangement. After 35 minutes of CPR and ROSC, the patient was admitted to the ICU. Laboratory tests revealed severe mixed metabolic acidosis (pH 6.85, base excess -29 mmol/L) consisting of lactic acidosis, hyperchloremic acidosis, and euDKA (β-hydroxybutyrate 3.2 mmol/L). The acidosis was attributed to several precipitating factors: major surgery, postoperative fasting, and impaired insulin secretion. Despite intensive care, the patient died on POD 8 due to severe post-hypoxic brain injury [19].

CASE 6: A 69-year-old female with T2DM (HbA1c 8.2%) and multivessel coronary artery disease was admitted for elective coronary artery bypass grafting (CABG). Her home medications included empagliflozin (25 mg/day) and metformin. Both drugs were discontinued 5 days prior to surgery, and the patient was transitioned to a subcutaneous basal-bolus insulin regimen. The surgery was uneventful. On POD 1, despite near-normal glycemia (146 mg/dL), the patient developed metabolic acidosis (pH 7.30, HCO_3^- 17 mmol/L, anion gap 22 mmol/L). Laboratory testing confirmed euDKA with a significantly elevated β-hydroxybutyrate level (7.9 mmol/L). Treatment was initiated with IV insulin and 10% dextrose infusions. Although the acidosis initially resolved, euDKA recurred on POD 3 following the transition back to subcutaneous insulin, despite the patient tolerating oral intake. Persistent ketonuria and glucosuria were observed for 5 days postoperatively. The patient's condition finally stabilized, and she was discharged on POD 5 on a regimen of metformin and glargine; empagliflozin was permanently discontinued [20].

CASE 7: A 60-year-old male with type 2 diabetes mellitus (T2DM) and metastatic melanoma was admitted for an elective craniotomy. His home medication included empagliflozin, which was continued until the day before admission. On the night preceding surgery, the patient presented with altered mental status and expressive aphasia. Preoperative laboratory results revealed severe metabolic acidosis (bicarbonate 11 mmol/L, anion gap 29 mmol/L) with normal blood glucose levels, which were initially overlooked. Intraoperatively, persistent acidosis unresponsive to fluid resuscitation prompted suspicion of euglycemic diabetic ketoacidosis due to SGLT2 inhibitor use. Diagnosis was confirmed by an elevated β-hydroxybutyrate level (3.1 mmol/L). Treatment was initiated with intravenous insulin and 5% dextrose in normal saline. Following three days of postoperative management in the neuro-intensive care unit, the metabolic acidosis resolved, and the anion gap closed [21].

CASE 8: A woman in her 40s with T2DM and moyamoya disease was admitted for scheduled cerebral revascularization. Her home medications included empagliflozin, metformin, pioglitazone, atorvastatin, and levothyroxine. Empagliflozin was discontinued 18 hours prior to surgery, and blood glucose was managed with insulin lispro. The surgery was uneventful; however, a few hours postoperatively, the patient suffered an acute left anterior cerebral infarct. Within 24 hours, she developed progressive metabolic acidosis (pH 7.01, anion gap 27 mmol/L) despite near-normal glycemia. Laboratory testing confirmed euDKA with significant ketonuria and elevated β-hydroxybutyrate (7.7 mmol/L). Treatment with intravenous insulin and dextrose

infusions successfully closed the anion gap. Despite metabolic improvement, the patient's clinical condition worsened due to the severity of brain injury, resulting in death [22].

The age of patients ranged from the fifth to the ninth decade of life. Type 2 diabetes mellitus was present in seven patients, while one patient had no previously diagnosed diabetes. Most procedures were elective and predominantly involved cardiac, neurosurgical, and orthopedic interventions.

The duration of SGLT2 inhibitor discontinuation prior to surgery ranged from 18 hours to 5 days. In most cases, euDKA developed intraoperatively or within the early postoperative period. Biochemical findings were consistent with high-anion gap metabolic acidosis. Arterial pH values ranged from 6.85 to 7.30. When measured, β-hydroxybutyrate levels were elevated. Despite the severity of metabolic acidosis, blood glucose concentrations were normal or only mildly elevated in the majority of patients.

Six patients recovered, whereas two patients died.

4. Discussion

The analysis of eight reported cases demonstrates that perioperative euDKA may develop despite preoperative discontinuation of SGLT2 inhibitors in accordance with current guidelines. These findings suggest that current discontinuation protocols may not fully eliminate the risk, particularly in patients undergoing major surgical procedures or experiencing acute neurological complications. In Cases 2 and 6, euDKA developed 75 hours and 5 days after the last dose, respectively, indicating that the metabolic effects of SGLT2 inhibitors may persist beyond the drug's plasma half-life [16,20]. Furthermore, Case 8 highlights the potential danger of a short withdrawal period (18 hours), during which the drug's peak effect coincided with a postoperative stroke, leading to rapid metabolic deterioration [22].

The pathophysiology of perioperative euDKA is related to an intensified metabolic response to surgical stress, fasting, and perioperative treatment modifications. Acute postoperative events, such as ischemic stroke, trigger a surge in counter-regulatory hormones that accelerate ketogenesis. Importantly, this risk is not limited to diabetic patients; individuals receiving SGLT2 inhibitors for heart failure or chronic kidney disease may also be vulnerable to metabolic decompensation. In these patients, the absence of marked hyperglycemia may reduce clinical vigilance and delay diagnosis and treatment.

A significant diagnostic pitfall remains the reliance solely on blood glucose measurements. Normal or mildly elevated glycemia may mask severe metabolic acidosis, leading to delayed recognition of early symptoms. In the most severe cases, sudden metabolic deterioration likely contributed to worse outcomes following primary events such as cardiac arrest or ischemic stroke [19,22]. Additional risk may be associated with premature postoperative reintroduction of SGLT2 inhibitor therapy. Restarting the drug before full oral intake is established may precipitate recurrence of euglycemic acidosis [18,19]. Therefore, SGLT2 inhibitors should be resumed only after achieving hemodynamic stability and adequate nutritional intake.

The standard perioperative discontinuation strategy may be insufficient in selected high-risk patients. For complex procedures or individuals at high metabolic risk, it may be justified to consider extending the withdrawal period. Safe perioperative management should include monitoring of acid–base status and serum β-hydroxybutyrate levels, as glycemic monitoring alone cannot reliably exclude acidosis [8]. The present analysis is limited by the small number of cases and the retrospective nature of available reports; however, it underscores the need for heightened clinical vigilance throughout the perioperative period in all patients receiving SGLT2 inhibitors, regardless of indication.

5. Conclusions

Euglycemic ketoacidosis is a severe, potentially life-threatening perioperative complication associated with SGLT2 inhibitors. Notably, it can occur even when current preoperative discontinuation guidelines are followed, suggesting that standard discontinuation protocols may not fully prevent this complication.

The perioperative period predisposes patients to metabolic derangements due to fasting, surgical stress, dehydration, and restricted carbohydrate intake. These factors, combined with the prolonged metabolic effects of SGLT2 inhibitors, may promote excessive ketogenesis and the development of metabolic acidosis despite normal or only mildly elevated blood glucose levels.

A critical finding is that euDKA can manifest not only in diabetic patients but also in non-diabetic individuals receiving SGLT2 inhibitors for cardiac or renal indications. The absence of overt hyperglycemia, coupled with non-specific clinical symptoms, remains a major barrier to timely diagnosis.

The presented data underscore the necessity for high clinical suspicion and an individualized approach to patients receiving SGLT2 inhibitor therapy during the perioperative period. Further research is required to redefine optimal discontinuation timelines and develop effective prevention strategies for euglycemic ketoacidosis.

Author Contribution:

Conceptualization was carried out by Beata Flis and Martyna Iwanowska. Methodology was developed by Małgorzata Styczyńska and Adam Zysk. Software development was performed by Jakub Zalewski. Validation involved Adrian Goss, Bartosz Fronczak and Maciej Wojewódzki. Formal analysis was conducted by Małgorzata Styczyńska and Alicja Cyrzan. Investigation was performed by Maciej Wojewódzki and Mateusz Ząbek. Resources were provided by Adam Zysk, Bartosz Fronczak and Adrian Goss. Data curation was handled by Adam Zysk and Mateusz Ząbek. The original draft was written by Beata Flis, while review and editing were undertaken by Martyna Iwanowska. Visualization was prepared by Maciej Wojewódzki and Adam Zysk. Supervision was provided by Beata Flis and Martyna Iwanowska. Project administration was managed by Jakub Zalewski and Alicja Cyrzan.

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