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SODIUM-GLUCOSE COTRANSPORTER-2 INHBITORS AND THE RISK OF EUGLYCEMIC DIABETIC KETOACIDOSIS: A NARRATIVE REVIEW

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ABSTRACT

Background: Euglycemic diabetic ketoacidosis (EuDKA) is a rare but life-threatening metabolic complication associated with the increasing use of sodium-glucose cotransporter-2 inhibitors (SGLT2i). Due to the absence of significant hyperglycemia, clinical diagnosis is frequently delayed, necessitating a high index of suspicion among clinicians.
Methods: This review synthesizes current clinical data, retrospective studies, and international guidelines regarding the pathophysiology, risk factors, and management of SGLT2i-induced euDKA. Specific focus was placed on perioperative protocols and emerging monitoring technologies.
Results: Clinical data indicate that patients with euDKA often present with milder acidosis but a significantly higher incidence of hypoglycemia compared to classic DKA. Effective management requires early initiation of 5–10% glucose infusions alongside insulin therapy to safely inhibit ketogenesis. Risk factors include rigorous low-carbohydrate diets, infection, and major surgery, with some data suggesting that up to 22% of cases initially diagnosed as type 2 diabetes are subsequently reclassified as LADA or type 1 diabetes. Continuous ketone monitoring emerges as a promising tool for early metabolic decompensation detection.
Conclusion: Therapeutic success in euDKA depends on a multidisciplinary approach and rigorous adherence to preventive measures. Key strategies include the discontinuation of SGLT2i 3 to 4 days prior to elective procedures and comprehensive patient education regarding "sick day" rules and hydration.

KEYWORDS

Sodium-Glucose Cotransporter-2 Inhibitors, Euglycemic Diabetic Ketoacidosis, Metabolic Acidosis, Perioperative Care

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Introduction

Sodium-glucose cotransporter 2 inhibitors (SGLT2i), commonly known as flozins, have transformed the management of cardiometabolic diseases. Initially introduced as second-line therapy for type 2 diabetes, they proved their efficacy in reducing cardiovascular event risk and mortality in high-risk patients through landmark trials such as EMPA-REG OUTCOME and DECLARE-TIMI 58 (Kluger et al., 2018; Zinman et al., 2015). ubsequent milestones, including the DAPA-HF and EMPEROR-Reduced trials, solidified their position as one of the four pillars of heart failure treatment, as reflected in the 2023 European Society of Cardiology (ESC) guidelines (Authors/Task Force Members: et al., 2024; McMurray et al., 2019; Packer et al., 2020). Despite these undeniable clinical benefits, the widespread use of flozins has revealed a serious complication described as euglycemic diabetic ketoacidosis (euDKA).

Ketoacidosis is a relatively well-understood complication of untreated or improperly managed type 1 diabetes (T1DM), affecting up to 5% of patients with this condition. It may also occur in patients with type 2 diabetes (T2DM) treated with SGLT2i in the presence of acute events such as major infection, trauma, or other severe illness. It is characterized by hyperglycemia, anion gap acidosis, and the presence of ketone bodies in the blood. EuDKA is also characterized by a triad of symptoms: metabolic acidosis, the presence of ketones, and, most atypically, relatively low plasma glucose levels, usually below 250 mg/dl. Low glycemia is misleading during diagnostics in hospital emergency departments, and delayed recognition is associated with poorer treatment outcomes. It is currently believed that euDKA occurs quite rarely; however, this may result from underdiagnosis of this condition (Dagdeviren et al., 2024). In the era of mass SGLT2i use, including in patients without diabetes, awareness of this complication becomes a critical element of patient safety. The aim of this paper is to review the literature and present the current state of knowledge regarding euDKA.

Methodology

To prepare this review, a comprehensive literature search was conducted across medical databases, including PubMed, Scopus, and Google Scholar. The search strategy encompassed articles published up to 2026, utilizing a combination of keywords such as: "euglycemic diabetic ketoacidosis," "euDKA," "SGLT2 inhibitors," "flozins," "type 2 diabetes," and "metabolic complications".

The selection process focused primarily on the most recent guidelines from scientific societies, meta-analyses, and cohort studies providing epidemiological and clinical data. The analysis also included case reports that uniquely illustrate atypical triggers of euDKA in patients without diabetes or those undergoing specific medical procedures, such as bariatric surgery or TAVR. The inclusion criteria were based on the scientific credibility of the publications and their direct relevance to the pathophysiology, diagnostics, and modern management protocols of euDKA. In total, several dozen references were analyzed, with a particular emphasis on publications from the last five years, allowing for a reliable presentation of the current state of knowledge and the evolution of therapeutic recommendations.

Results

Epidemiology and Triggering Factors

The epidemiology of euDKA indicates a rare but clinically significant phenomenon, accounting for approximately 2.6% to 3.2% of all hospitalizations due to DKA (Jenkins et al., 1993). Although the incidence of euDKA in the general population of T2DM patients receiving SGLT2 inhibitors is estimated at 0.16 to 0.76 events per 1,000 patient-years, the overall risk of developing DKA in this therapeutic group increases nearly sevenfold compared to patients not using SGLT2i (Blau et al., 2017). Collectively, the total incidence of DKA following SGLT2 inhibitor administration is estimated at approximately 0.1% (Erondu et al., 2015a). Particularly high risk is observed in T2DM patients using this class of drugs off-label, where the incidence of DKA events ranges between 5–12%, though it should be noted that only a portion of these cases follow the euglycemic variant(Goldenberg et al., 2016). These data emphasize that while euDKA is a rare complication, its epidemiology is strictly correlated with the pharmacotherapy profile and diabetes type, necessitating particular vigilance in high-risk groups.

Current 2024 guidelines from the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD) list key risk factors, which include: inappropriate insulin doses (resulting from increased unforeseen exertion or consumption of an insufficient meal), acute infections, major trauma, alcohol consumption, states of dehydration, and restrictive low-carbohydrate (ketogenic) diets(Umpierrez et al., 2024). Pregnancy is also a significant risk factor, as it involves an increased demand for carbohydrates that is not always adequately met (Nasa et al., 2021a). Particular attention is given to patients after gastrointestinal

surgeries, in whom preoperative fasting, prolonged food restriction, and delayed gastrointestinal absorption due to surgery lead to a state predisposing to ketosis (Milder et al., 2018).

Pathophysiology

In healthy individuals, blood glucose levels are maintained within a relatively narrow physiological range to ensure that every organ has sufficient access to it (Ghezzi et al., 2018). Glucose is a crucial energy source for the cells of every organ. The kidneys play an important role in maintaining glucose at physiological concentrations. Initially, glucose enters the urine; however, reabsorption occurs in the distal tubule involving sodium-glucose cotransporter proteins and the GLUT-2 protein. Typically, all of its molecules are reabsorbed, and the final urine contains no glucose. However, when the blood glucose concentration exceeds the renal threshold, transport proteins are unable to capture all molecules. Then, glucose can be detected in the final urine (Gerich, 2010).

This mechanism is utilized by SGLT2i. They act by blocking transport proteins in the proximal tubule. In this way, they lower the renal threshold, causing constant glucose excretion in the urine regardless of its plasma concentration (Wright, 2021).In addition, SGLT2i also exhibit cardioprotective effects, the mechanism of which has not yet been fully elucidated. Many explanations are proposed related to increased diuresis, natriuresis, blood pressure reduction, or decreased oxidative stress (Lopaschuk & Verma, 2020; Salvatore et al., 2022).

The fundamental problem occurring in the classic form of DKA is insulin deficiency (relative or absolute). Absolute insulin deficiency typically occurs in T1DM, when destroyed pancreatic cells are unable to secrete sufficient amounts of it. Relative insulin deficiency occurs in cases of increased insulin resistance (such as inflammation or other stressful situations for the body). In such cases, the inhibitory effect of insulin on glucagon secretion is lacking. An excess of insulin-antagonistic hormones causes hyperglycemia by increasing glycogenolysis and hepatic gluconeogenesis, and by decreasing intracellular glucose transport. They also act on adipose tissue, increasing gluconeogenesis and ketogenesis from free fatty acids in adipocytes. Ketone bodies become an energy substrate for cells, partially replacing glucose in this function, as it cannot be transported inside the cell due to the lack of insulin (Rosenstock & Ferrannini, 2015). The accumulation of ketone bodies is responsible for metabolic acidosis. Hyperglycemia, by causing glucosuria and osmotic diuresis, induces hypovolemia as a result of water removal along with glucose through the kidneys (Cartwright et al., 2012).

Understanding the mechanism of euglycemia in euDKA is crucial for avoiding diagnostic errors. By lowering the renal threshold for glucose and reducing its plasma concentration, SGLT2i also decreases insulin secretion (Ferrannini et al., 2014). Additionally, it is suggested that they directly increase glucose levels. The disruption of the insulin-glucagon balance predisposes patients to higher ketone body concentrations even prior to the clinical onset of euDKA (Ferrannini et al., 2016). This increases sensitivity to any subsequent bodily imbalance, such as fasting, surgery, pregnancy, or major infection. Reduced or insufficient carbohydrate intake in such situations further lowers insulin levels and shifts the balance toward glucagon (Chow et al., 2023). SGLT2i-induced glucosuria additionally exacerbates dehydration. Furthermore, glucosuria is accompanied by natriuresis, which disrupts the electrochemical gradient in the tubular fluid, enhancing the reabsorption of ketone bodies and thus intensifying acidosis (Ferrannini et al., 2014).

For the physician, the most important conclusion is the fact that glycemia in a patient taking flozins ceases to be a reliable tool for assessing the risk of acidosis. While in classic DKA hyperglycemia serves as a marker of the severity of the disorders, in euDKA a patient can be in a state of acidosis with glycemia <200 mg/dl (Chow et al., 2023).

Case Analysis

The medical literature contains numerous case reports on the occurrence of euDKA. These often involve patients chronically treated for T1DM or T2DM with insulin deficiency, low BMI, and low hepatic glycogen stores (Dagdeviren et al., 2024). Clinical symptoms are usually similar to the classic form of DKA. Most commonly, patients report nausea, vomiting, fatigue, and abdominal pain. However, symptoms may also include dyspnea, tachycardia, and low blood pressure (Menghoum et al., 2021). Nevertheless, the dynamics of symptom progression are typically slower in euDKA. The absence of hyperglycemia in the blood usually results in a lower intensity of symptoms such as polyuria, polydipsia, or altered mental status. The lack of characteristic symptoms complicates the diagnosis (Dagdeviren et al., 2024).

Patients who self-monitor their blood glycemia levels often similarly fail to notice abnormalities. Instead of increasing the insulin dose, they kept the doses unchanged or even reduced them, which exacerbated the

problem. Upon hospital admission, physicians did not recognize DKA due to the absence of hyperglycemia. This resulted in unnecessary examinations and inappropriate treatment (Peters et al., 2015).

In most patients, the occurrence of euDKA is associated with a sudden disruption of the body's metabolic balance. A common cause is the restriction of food intake. This may be related to the perioperative period and the necessary fasting during this time. In individuals treated with insulin, inappropriate dosing may be the cause. Serious infections and exacerbations of chronic diseases can increase insulin resistance and also cause euDKA (Danne et al., 2019).

In the medical literature, descriptions of atypical cases of euDKA can also be discovered in patients without diabetes who take SGLT2i most commonly for heart failure. Umapathysivam et al., 2024 presented descriptions of two such cases. The first involved a young patient with Duchenne muscular dystrophy (DMD) and heart failure with reduced ejection fraction (HFrEF), in whom dapagliflozin (10 mg daily) and chronic prednisolone were used. The development of euDKA occurred in him following gastritis induced by non-steroidal anti-inflammatory drugs (NSAIDs). This condition led to a significant restriction of food and fluid intake, which, combined with the action of the SGLT2 inhibitor, triggered severe metabolic imbalances. The patient presented to the hospital due to vomiting. He took the last dose of the SGLT2 inhibitor 36 hours before hospitalization. Laboratory tests revealed severe metabolic acidosis (pH 7.14) with a high anion gap (25 mmol/l) and decreased bicarbonate concentration (9 mmol/l). Importantly, glycemia was only 70.2 mg/dL, which, combined with concomitant glucosuria, confirmed the diagnosis of euglycemic diabetic ketoacidosis. Normal glycated hemoglobin values (HbA1c 5.2%), documented both during the episode and before initiating the SGLT2i, ruled out previously undiagnosed diabetes. The use of intensive crystalloid fluid therapy supplemented with dextrose allowed for rapid resolution of the acidosis, with minimal requirement for exogenous insulin therapy. Notably, glycosuria persisted even 36 hours after the last dose of dapagliflozin.

Seki et al., 2022 presented a case of DKA in an elderly patient, in whom the causative factors were likely perioperative fasting and a surgical procedure. The patient, with numerous comorbidities including stage II chronic kidney disease and heart failure (EF 30%), was on chronic dapagliflozin therapy. The hospitalization concerned a scheduled transcatheter aortic valve implantation (TAVI) procedure. Despite a preoperative episode of diarrhea, biochemical parameters just before the procedure remained within normal limits, and the HbA1c value (5.4%) ruled out a diabetic background.

On the day of the procedure, after an overnight fast, the patient took her routine medications, including a dose of the SGLT2 inhibitor. On the first day following the TAVI procedure, worsening metabolic acidosis was observed, accompanied by hypoglycemia (52 mg/dL), a decrease in bicarbonate concentration (15.9 mmol/L), and the presence of ketone bodies and glucose in the urine. The therapeutic intervention was limited to the discontinuation of dapagliflozin, intravenous administration of 10 g of glucose, and a rapid return to oral nutrition. Significantly, metabolic stabilization was achieved without the administration of exogenous insulin, suggesting that the stimulation of endogenous insulin secretion through carbohydrate intake alone was sufficient to inhibit ketogenesis.

Differential Diagnosis and the Role of the Anion Gap

EuDKA is a life-threatening condition. However, treatment is often delayed by the absence of hyperglycemia (Barski et al., 2019). The diagnosis of euDKA is a diagnosis of exclusion; it requires the recognition of high anion gap metabolic acidosis (Modi et al., 2017).

Testing for the presence of ketone bodies in the urine is a widely available, low-cost, and non-invasive diagnostic method that requires neither advanced equipment nor specialized technical skills. Nevertheless, this solution is associated with a range of limitations, from patients' subjective reluctance to collect urine samples to significant diagnostic constraints.

Dipstick tests, based on the nitroprusside reaction, offer only a semi-quantitative measurement of acetoacetate (and sometimes acetone) concentration, which reflects the average ketone content accumulated in the bladder since the last voiding (Walker et al., 1990). Such measurement characteristics may lead to a dangerous delay in diagnosing developing acidosis or an erroneous assessment of the rate of its resolution.

A key diagnostic problem is the fact that during treatment, circulating beta-hydroxybutyrate is oxidized to acetoacetate. This causes a paradoxical increase in urine ketone body concentration with a simultaneous decrease in their blood levels, which may create a false impression of therapeutic failure. Furthermore, since acetoacetate constitutes only approximately 20% of the total circulating ketone body pool, the sensitivity of urinalysis is significantly limited. Interpretation of the results can be further complicated by false-positive findings caused by intense urine coloration or the presence of interfering substances such as levodopa or sulfhydryl compounds (Csako & Elin, 1993).

Finally, the effectiveness of this method depends on the patient's subjective risk assessment and their ability to perform the test. In states of severe dehydration or acute kidney injury accompanied by oliguria, sample collection may be impossible. An additional barrier is the lack of comfortable conditions for material collection in out-of-home situations, which, combined with the aforementioned factors, limits the clinical utility of urinalysis in monitoring euDKA.

Differential diagnosis should include lactic acidosis (particularly in patients with cardiogenic shock or those treated with metformin), alcoholic ketoacidosis, and starvation ketoacidosis (Nasa et al., 2021b).

Alcoholic ketoacidosis is typically associated with chronic alcoholism. The symptoms of this entity are similar to those of euDKA. Such patients are also in a state of chronic carbohydrate deficit, with alcohol being one of its few sources for them (Long, Lentz, & Gottlieb, 2021). Key differences include a higher frequency of hypoglycemia and the patient's medical history. The absence of a prior diagnosis of diabetes and a history of alcohol abuse favor this diagnosis (Suzuki et al., 2013). The dominant ketone body in this case is β-hydroxybutyrate, which may not be routinely detected in urine dipstick tests (McGuire et al., 2006). Therefore, when such a diagnosis is suspected, serum ketone concentrations should be measured.

Differentiating starvation ketosis from euDKA associated with reduced carbohydrate intake is a difficult task. Here as well, the medical history is key. The absence of a prior diagnosis of diabetes, comorbidities, and the non-occurrence of acidosis favors the diagnosis of ketosis caused by fasting (Larroumet et al., 2020).

Sepsis is one of the conditions that can mimic euDKA. The presence of high lactate concentrations and the absence of ketone bodies help to recognize it (Lucero & Chapela, 2018).

Any unexplained condition presenting with a high anion gap and metabolic acidosis in a patient with diabetes or other risk factors should raise suspicion of euDKA. Such risk factors include, in particular: pregnancy, fasting, surgery, infection, and SGLT2i use. Ordered laboratory tests should include: plasma and urinary ketone bodies, electrolytes, glycemia, creatinine, blood gas analysis, lactic acid, procalcitonin, complete blood count, and blood alcohol levels. It is also worthwhile to perform an ECG. Laboratory results are usually sufficient to differentiate euDKA from alcoholic ketoacidosis or serious infection (Nasa et al., 2021a).

Treatment

The treatment regimen for euDKA is similar to the protocols used in the classic form of DKA, although it requires specific modifications due to relatively low glycemia levels. Key is the monitoring of glucose concentrations at hourly intervals, as well as acid-base balance parameters and electrolytes every 2 – 4 hours. In the case of diagnosing a reversible cause of DKA, its treatment is also necessary (Long, Lentz, Koyfman, et al., 2021).

The first therapeutic action taken should be the discontinuation of the SGLT2 inhibitor and intensive fluid resuscitation with crystalloids (1-2 L within the first 2 hours). Standardly, 0.9% NaCl solution is used at a rate of 500–1000 mL/h, after which a decision may be made to reduce the infusion rate to 200–500 mL/h and potentially switch to a hypotonic solution (0.45% NaCl) depending on the dynamics of serum sodium concentration and the patient's condition (Dagdeviren et al., 2024).

In parallel, correction of hypokalemia and initiation of a continuous intravenous insulin infusion (usually at a dose of 0.05–0.1 U/kg/h) are typically necessary. Due to the increased risk of hypoglycemia in patients with euDKA, simultaneous intravenous administration of a 5–10% glucose solution is often required (Sell et al., 2023). This allows for the continuation of insulin administration at a dose sufficient to inhibit ketogenesis without exposing the patient to dangerous drops in glycemia. Intravenous insulin therapy is maintained until the complete resolution of acidosis (pH > 7.30, bicarbonate > 18 mEq/L, normalization of the anion gap) and the patient's return to oral nutrition (Dagdeviren et al., 2024).

Retrospective data presented by Sell et al., 2023 indicate that patients with euDKA are typically characterized by a milder course of acidosis upon admission and shorter hospital stays and intravenous insulin infusions compared to classic DKA. However, due to the significantly higher incidence of hypoglycemia in this group, it is necessary to use modified protocols involving a lower insulin infusion rate and higher glucose concentrations in infusion fluids. Bicarbonate supplementation is restricted solely to cases of life-threatening acidosis (pH < 6.9) (Dagdeviren et al., 2024).

Mortality during the course of classic DKA in developed countries remains at a level between 1–5%, however, it increases drastically in cases of recurrent episodes and in regions with limited access to medical care (Fayfman et al., 2017). Data regarding SGLT2i-induced euDKA are still limited, yet meta-summaries suggest a relatively low mortality rate of approximately 2.4% (Juneja et al., 2023). The prognosis is typically

favorable with early recognition and the implementation of adequate fluid therapy and carbohydrate supplementation.

The foundation of the treatment for SGLT2i-induced euglycemic diabetic ketoacidosis is the discontinuation of the drug, intensive hydration, and insulin therapy. The specificity of euDKA necessitates the frequent use of 5–10% glucose infusions in parallel with insulin, which distinguishes this protocol from standard management in hyperglycemic ketoacidosis. The ultimate goal of treatment is the complete normalization of metabolic parameters and the resolution of the anion gap.

Discussion

Prevention and the role of technology

Before initiating SGLT2 inhibitor therapy, it is essential to conduct a detailed clinical history for the early identification of patients with increased susceptibility to the occurrence of DKA (Patel & Nair, 2022). The high-risk group primarily includes individuals with reduced endogenous insulin secretion reserve, patients with latent autoimmune diabetes in adults (LADA), or a history of chronic pancreatitis, with some authors suggesting routine screening for LADA before initiating SGLT2i (Erondu et al., 2015b). An important aspect is also patient education regarding the maintenance of proper hydration and carbohydrate intake. In cases where acidosis occurs in individuals with diagnosed T2DM, the accuracy of the original diagnosis should be verified, as data indicate that up to 22% of such cases are ultimately reclassified as T1DM or LADA (Hamblin et al., 2019).

SGLT2 inhibitors increase the risk of developing ketoacidosis during periods of infection, severe stress, and insufficient carbohydrate intake. Patients should be advised against alcohol consumption and the use of ketogenic diets, which may disrupt the body's fragile metabolic balance (Somagutta et al., 2022). In the case of patients treated with insulin, the introduction of an SGLT2 inhibitor often requires dosage modification; however, drastic reductions or complete discontinuation of insulin therapy should be avoided, especially during other acute illnesses (Patel & Nair, 2022). This is particularly important in hospitalization settings, where frequent changes in insulin dosing regimens by medical staff can induce a state of euDKA (Selwyn & Pichardo-Lowden, 2023). Cohort studies confirm that in patients using SGLT2 inhibitors, the risk of developing acidosis during a hospital stay is 38% with a median glycemia of 13.5 mmol/L, while in the group treated with other methods, this rate is only 2% at a significantly higher glucose concentration (>25.4 mmol/L)(Hamblin et al., 2019).

Perioperative care requires consideration of the drugs' half-life, which ranges from 11 to 13 hours, and their prolonged pharmacodynamic effect (Patel & Nair, 2022). Currently, it is recommended to discontinue SGLT2i three days before elective surgery (Takemura et al., 2025). International scientific societies also recommend immediate drug withdrawal in the case of emergency procedures, acute fever, or dehydration. In situations requiring bowel preparation, such as colonoscopy, the break should be at least three days, whereas for shorter procedures, such as gastroscopy, discontinuation of the drug only on the day of the procedure is permissible (Milder et al., 2018). In cases of emergency surgery, where prior suspension of therapy was not possible, it becomes crucial to individually determine the risk of acidosis and, if necessary, use perioperative intravenous insulin and glucose infusions (Ito et al., 2022).

Particular attention should be paid to patients undergoing surgical treatment for obesity. In bariatric surgery, risk factors include rigorous low-carbohydrate high-protein diets used in the preoperative period to reduce liver mass, as well as nausea, vomiting, and rapid weight loss after the procedure (Milder et al., 2018). In this group, symptoms of euDKA may manifest for up to six weeks after surgery (Thiruvenkatarajan et al., 2019).

Due to the absence of typical hyperglycemia, traditional glucose monitoring may be insufficient, therefore, in the case of illness or worsening glycemic control, it is recommended to measure ketone body concentrations directly in the blood, as urine tests may be unreliable during SGLT2i use (Chow et al., 2023).

A potential solution to increase the safety of high-risk patients is continuous interstitial fluid ketone monitoring (CKM) technology. The development of wearable devices to date has focused almost exclusively on glucose monitoring, which resulted from the necessity to prevent rapid glucose fluctuations and their direct complications. Nevertheless, the integration of continuous ketone measurement seems a logical extension of current systems, which, with a minimal increase in costs, could significantly improve the patient's health awareness and raise their safety level. It should, however, be noted that CKM is still a novel technology. Despite the theoretical foundations, evidence regarding, among others, the ketone concentration thresholds at which medical intervention is necessary, is still lacking. In the future, the CKM system could be a fundamental

component of continuous glucose monitoring systems for individuals with type 1 diabetes and perhaps for patients taking SGLT2i with risk factors for acidosis. Early detection of such metabolic disorders could reduce the frequency of hospitalizations and the burden on medical care(Kong et al., 2024).

Currently, no specific implications resulting from the long-term use of these drugs have been established, however, a key element remains the awareness of physicians that the risk of euDKA is higher in patients treated for diabetes than in individuals receiving these drugs for heart failure or kidney disease (Koceva & Kravos Tramšek, 2024).

Conclusions

Euglycemic diabetic ketoacidosis constitutes a rare but life-threatening metabolic complication, the incidence of which is increasing with the popularization of SGLT2 inhibitors. The lack of clear hyperglycemia in the clinical presentation often leads to delayed diagnosis, highlighting the necessity of measuring blood ketone body concentrations in

high-risk patients presenting with non-specific symptoms such as nausea or weakness. A key preventive factor is rigorous adherence to perioperative protocols involving the discontinuation of flozins 3 to 4 days before an elective procedure, as well as patient education regarding the avoidance of restrictive diets and proper hydration.

The treatment of euDKA, although based on classic fluid therapy and insulin therapy regimens, requires a specific modification involving the early initiation of glucose infusions to safely continue the insulin administration necessary to inhibit ketogenesis. A promising solution to increase patient safety, especially for those with type 1 diabetes or using SGLT2 inhibitors with present risk factors, is continuous ketone monitoring (CKM) technology, which in the future may become a standard of care comparable to glycemic monitoring systems. Ultimately, therapeutic success in the case of euDKA depends on high diagnostic vigilance of medical personnel and an interdisciplinary approach involving diabetologists, cardiologists, and surgeons.

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