Review Article

Perioperative Diabetic Ketoacidosis in Type 2 Diabetes: Risk and Prevention in the Era of SGLT2 Inhibitors

The American Surgeon™
2026, Vol. 0(0) 1-8

DOI: 10.1177/00031348261443329
journals.sagepub.com/home/asu
Sage

Pao-Jen Kuo, MD[1], Yuan-HaoYen, MD[1], and Ching-Hua Hsieh, MD, PhD[1]

Abstract

Diabetic ketoacidosis (DKA) typically affects patients with type 1 diabetes but can also occur in type 2 diabetes under severe stress or relative insulin deficiency. The postoperative phase constitutes a high-risk interval for diabetic ketoacidosis in individuals with type 2 diabetes, attributable to surgical stress, altered medication routines, and metabolic changes. The introduction of sodium-glucose cotransporter-2 (SGLT2) inhibitors has added complexity, as these medications can precipitate euglycemic DKA where ketoacidosis occurs without marked hyperglycemia. This review examines postoperative DKA in type 2 diabetes, focusing on pathophysiology, clinical outcomes, prevention strategies, and management approaches. The pathophysiology involves insulin deficiency, counterregulatory hormone excess, and SGLT2 inhibitor effects that promote ketogenesis. Risk factors include emergency surgery, poor glycemic control, insulin-dependent status, and SGLT2 inhibitor use. Prevention strategies include optimizing preoperative glycemic control, discontinuing SGLT2 inhibitors at least 3 days before surgery, maintaining basal insulin during the perioperative period, and monitoring for ketosis. Management requires aggressive fluid resuscitation, insulin therapy, electrolyte management, and addressing any precipitating factors. Early recognition is essential, particularly for euglycemic DKA which may be overlooked due to near-normal blood glucose levels. With proper preventive measures and timely management, most cases of postoperative DKA in type 2 diabetes can be avoided or successfully treated.

Keywords

diabetic ketoacidosis, type 2 diabetes, SGLT2 inhibitors, euglycemic DKA, perioperative management

Introduction

Diabetic ketoacidosis (DKA) is classically associated with absolute insulin deficiency in type 1 diabetes but can occur in type 2 diabetes during severe stress or relative insulin deficiency,[1] leading to prolonged hospitalization, ICU admissions, and increased mortality.[1,2] Postoperative DKA can present atypically, mimicking sepsis or typical post-surgical metabolic changes, contributing to low clinical awareness. Clinicians may not suspect DKA in postoperative type 2 diabetes patients, particularly when glucose levels are near-normal (euglycemic DKA), resulting in misdiagnosis or delayed treatment. The increasing use of sodium-glucose cotransporter-2 (SGLT2) inhibitors has introduced a new dimension, with numerous reports of perioperative euglycemic DKA.[3] This knowledge gap—compounded by rising SGLT2 inhibitor use—necessitates a comprehensive review to inform practitioners about this underrecognized yet severe complication.

Following PRISMA guidelines, we conducted a structured literature search using PubMed with broad keyword combinations (eg, "postoperative diabetic ketoacidosis, "type 2 diabetes, "surgery, and "SGLT2 inhibitors"). Inclusion criteria, defined a priori, encompassed clinical articles (case reports, case series, cohort studies, and reviews) discussing postoperative DKA in type 2 diabetes, particularly euglycemic DKA and SGLT2 inhibitor-related cases. We excluded animal

[1]Department of Plastic Surgery, Kaohsiung Chang Gung Memorial Hospital, Chang Gung University and College of Medicine, Kaohsiung, Taiwan

Corresponding Author:
Ching-Hua Hsieh, MD, PhD, Department of Plastic Surgery, Kaohsiung Chang Gung Memorial Hospital, Chang Gung University and College of Medicine, No. 123, Dapi Road, Niao-Song District, Kaohsiung 833, Taiwan.
Email: m93chinghua@gmail.com

studies, pediatric cases, articles focusing solely on type 1 diabetes, and non-English publications. Three reviewers independently screened titles, abstracts, and full texts, resolving discrepancies through consensus. This systematic approach ensures comprehensive, unbiased literature collection while maintaining transparency and reproducibility of our search strategy.

Pathophysiology of Postoperative DKA in Type 2 Diabetes

Diabetic ketoacidosis results from absolute or relative insulin deficiency combined with excess counter-regulatory hormones (glucagon, catecholamines, cortisol, growth hormone).[3] In type 2 diabetes, residual insulin secretion may become insufficient under acute stress such as surgery or infection, precipitating ketosis.[2] Surgical stress elevates catecholamines and cortisol, antagonizing insulin action and promoting lipolysis and gluconeogenesis.[3] Free fatty acids released from adipose tissue are hepatically oxidized to ketone bodies (β-hydroxybutyrate and acetoacetate).[3] Concurrently, stress-induced hyperglycemia causes osmotic diuresis, dehydration, and electrolyte losses, impairing insulin signaling and renal ketone clearance—perpetuating a cycle of rising ketones and worsening metabolic acidosis.[4] Beyond metabolic derangements, DKA also triggers significant systemic inflammation. In rat models, DKA induces elevated serum cytokines (TNF-α, IL-1β) and increased cerebral inflammatory markers during ketosis.[5] Notably, some neuroinflammatory changes persist after DKA resolution,[5] indicating lasting pathophysiological consequences beyond the acute metabolic crisis.

A notable postoperative DKA had been observed in insulin-dependent type 2 diabetes receiving bariatric surgery.[6] Rapid weight loss and drastically reduced caloric intake after bariatric procedures can decrease insulin requirements, and if insulin therapy is not appropriately adjusted or is stopped too soon, DKA can result.[7] The stress of surgery and anesthesia itself is a precipitant: pain and stress hormone release can provoke prolonged hyperglycemia, while anesthesia may mask early symptoms of hyperglycemia or ketosis. Furthermore, inadvertent withholding of basal insulin on the day of surgery may further predispose DKA.

SGLT2 inhibitors have fundamentally altered the understanding of DKA pathophysiology. By reducing renal glucose reabsorption and causing glucosuria, these agents lower blood glucose without a commensurate rise in insulin secretion.[8,9] This creates a distinct metabolic state: insulin levels fall and glucagon rises, tilting metabolism toward ketosis despite near-normal glucose.[10,11] Euglycemic DKA (eu-DKA)—defined by normal or mildly elevated blood glucose (often <250 mg/dL)—is strongly associated with SGLT2 inhibitor use.[3] Several mechanisms underlie SGLT2 inhibitor-induced ketosis[12,13,14]: glycosuria lowers insulin release, disinhibiting lipolysis; reduced insulin and elevated glucagon promote fatty acid oxidation and ketone production; and SGLT2 inhibitors may directly stimulate α-cell glucagon secretion, as demonstrated in human islet cell studies. Perioperatively, these drugs compound ketogenic risk through caloric loss, appetite suppression, and reduced oral intake.[3] Surgical fasting and physiological stress further elevate counter-regulatory hormones, exacerbating the tendency toward ketoacidosis.[2,15] Consequently, a type 2 diabetic patient on an SGLT2 inhibitor may develop postoperative DKA despite apparently controlled glucose.[3] Many reported postoperative eu-DKA cases were initially misdiagnosed for this reason (Table 1).

Risk Factors and Clinical Outcomes

A 2023 cohort study examined over 147,000 surgical patients with type 2 diabetes, finding that SGLT2 inhibitor users had a significantly higher 30-day postoperative DKA incidence (IRR: 6.33; 95% CI: 5.57-7.18) compared to non-users.[22] Within SGLT2-treated patients, HbA1c ≥ 8% and preoperative insulin use each conferred approximately triple the DKA risk[23,24]—consistent with the observation that insulin-deficient, poorly controlled type 2 diabetics are most vulnerable under surgical stress. DKA risk also varied markedly by surgical urgency. Elective surgery carried an approximately six-fold increased DKA incidence with preoperative SGLT2 inhibitor use, while emergency surgery yielded an IRR of ∼24 compared to elective procedures.[22] However, a large 2025 study by Dixit et al of ∼34,000 emergency surgery patients found no statistically significant difference in 14-day postoperative DKA between SGLT2 inhibitor users (3.8%) and non-users (3.5%) after covariate adjustment.[25] This discrepancy likely reflects methodological differences, including surgical type, analytic approach, outcome definitions, follow-up duration (14 vs 30 days), and population characteristics.[25] These findings support differentiated perioperative management: for elective surgery, discontinuing SGLT2 inhibitors at least 3 days preoperatively effectively eliminates added DKA risk; for emergency surgery, aggressive insulin therapy and vigilant metabolic monitoring are essential.[26,27,28]

When postoperative DKA does occur in a type 2 diabetic patient, the consequences can be severe. Among SGLT2 inhibitor users, patients who developed postoperative DKA more frequently required invasive mechanical ventilation, had more acute renal failure necessitating dialysis, developed more infections/sepsis,

Table 1. Published Cases of Postoperative Euglycemic DKA Associated With SGLT2 Inhibitors

Author (year)	Type of surgery	SGLT2 inhibitor used	Onset of DKA	Presentation	Outcome
Wang and Isom (2020)[16]	Cerebral revascularization (moyamoya disease)	Empagliflozin	24 hours postoperatively	High anion-gap metabolic acidosis	Full recovery
Osafehinti et al (2021)[17]	Coronary artery bypass graft	Empagliflozin	Few hours postoperatively	Elevated anion-gap metabolic acidosis	Full recovery
Gomez-Sanchez et al (2021)[18]	Femoral endarterectomy and bypass with graft	Empagliflozin	Postoperative day 2	Acute delirium, hypotension, severe metabolic acidosis	Full recovery
Chandrakumar et al (2021)[3]	Glaucoma surgery	Ertugliflozin	Immediate postoperatively	Reduced oral intake, fatigue, euglycemic high-gap acidosis	Full recovery
Smith et al (2021)[19]	Laparoscopic sleeve gastrectomy	Canagliflozin	Postoperative day 3	Tachycardia with later developed altered mental status, tachypnea, and anion-gap acidosis	Full recovery
Ritchie and Dixon (2022)[20]	Total hip arthroplasty for fracture	Empagliflozin	Intraoperative	High anion-gap acidosis requiring critical care and IV insulin therapy	Full recovery (prolonged)
Bazan et al (2025)[21]	Case series (3 cases): Laparoscopic cholecystectomy; hip hemiarthroplasty; appendectomy and cystectomy	Empagliflozin (2 cases) and Canagliflozin (1 case)	Ranged from 4 hours to 3 days postoperatively	All presented with elevated anion-gap metabolic acidosis and ketosis	Full recovery
Petersen et al (2023)[1]	Percutaneous coronary intervention for NSTEMI	Empagliflozin	Postoperative day 2	Dyspnea, nausea, vomiting; tachycardia and hypertension; high anion-gap acidosis	Full recovery (prolonged)
Chen et al (2025)[23]	Laparoscopic distal pancreatectomy	Dapagliflozin	Within a few days postoperatively	Euglycemic DKA despite holding SGLT2 inhibitor preoperatively; normal/mild glucose levels caused delayed recognition of high-gap acidosis	Full recovery

had longer lengths of hospital stay, and higher in-hospital mortality than those who did not.[22] Diabetic ketoacidosis not only necessitates ICU admission for insulin infusion and close monitoring but also precipitates complications like aspiration pneumonia or bloodstream infections.[29,30] Beyond immediate outcomes, 1 study found that type 2 diabetes patients who experienced DKA had a 1.55-times higher risk of stroke in the following years compared to diabetic patients without DKA.[31] The risk was especially elevated in the first 6 months after the DKA episode.[31]

Prevention of Postoperative DKA

To prevent postoperative DKA, preoperative optimization of diabetic patients is the first step. This includes achieving the best feasible glycemic control in the weeks leading up to elective surgery,[32] as high HbA1c is linked to DKA risk and also to poor wound healing and infection.[33] Elective surgeries should ideally be deferred in patients with severely uncontrolled diabetes until glycemia is stabilized.[32] Patients on insulin should receive clear instructions and adjustments for the day of surgery: notably, do not withhold basal insulin entirely.[34] Clinical experience and studies from bariatric surgery suggest that continuing a reduced dose of basal insulin on the morning of surgery is far safer than stopping all insulin.[35]

A crucial preventive measure is the management of SGLT2 inhibitors. In March 2020, the U.S. Food and Drug Administration (FDA) issued updated guidance recommending holding SGLT2 inhibitors for at least 3 days before

scheduled surgeries and at least 4 days for the longer-acting ertugliflozin.[36] A systematic review conducted in 2023 encompassing 99 documented cases of SGLT2 inhibitor-associated perioperative ketoacidosis provides additional support for this recommendation: The review concluded that adhering to a ≥3-day discontinuation prior to surgery could prevent most SGLT2-related DKA episodes.[26]

Given the risk of eu-DKA, routine ketone screening has been proposed for high-risk patients preoperatively.[37] Though large trials are lacking, elevated preoperative ketones may justify postponing elective surgery to treat incipient ketosis.[38,39] For urgent or emergency cases where SGLT2 inhibitors were recently taken, postoperative acid-base and ketone monitoring is essential, with some experts recommending prophylactic insulin and dextrose infusions perioperatively.[40] Serial serum bicarbonate monitoring alongside low-dose insulin and dextrose supplementation is advised, as rising bicarbonate confirms metabolic recovery.[41,42] Diabetic ketoacidosis resolution is indicated when bicarbonate normalizes to ≥18 mEq/L with appropriate clinical improvement.[43]

EMR-based alerts for SGLT2 inhibitor users have demonstrated measurable benefit; 1 study reported that a perioperative precaution pop-up reduced postoperative DKA incidence, with the IRR falling from ∼4.06 to 2.97.[22] Patient education is equally important—patients should be counseled preoperatively on holding SGLT2 inhibitors, maintaining basal insulin, staying hydrated, and reporting symptoms such as nausea, abdominal pain, or excessive fatigue.

Early postoperative feeding or IV dextrose minimizes prolonged catabolic fasting. SGLT2 inhibitors should not be restarted until the patient is eating regularly and free of surgical complications.[44,45] Emerging risk stratification tools, including a 2024 algorithm incorporating surgery type, urgency, HbA1c, glucose, renal function, and SGLT2 inhibitor use, can guide intensity of monitoring and prophylactic intervention.[46]

Within an Enhanced Recovery After Surgery (ERAS) framework, multidisciplinary coordination among anesthesia, surgery, and endocrinology is essential. Shared protocols for preoperative SGLT2 inhibitor cessation, intraoperative glucose control, and postoperative surveillance—combined with early endocrinologist involvement and routine EMR alerts—align with ERAS principles to maintain perioperative metabolic stability.[47] Regular postoperative debriefs analyzing DKA events further support continuous system improvement.

Management of Postoperative DKA

Clinicians should suspect DKA in any post-surgical diabetic patient who develops symptoms like unexplained nausea, vomiting, abdominal pain, tachypnea, or an altered mental status—even if blood glucose is not extremely high. If DKA is even remotely suspected, prompt measurement of serum electrolytes, blood gas, and ketone levels (or serum β-hydroxybutyrate) should be done.

The diagnostic criteria for DKA remain the same: presence of a metabolic acidosis (anion gap acidosis with bicarbonate typically ≤18 mEq/L, blood pH ≤7.30) with positive ketones, and usually hyperglycemia >250 mg/dL—though in euglycemic DKA, glucose may be only mildly elevated (sometimes 120-250 mg/dL).[3]

Acute management principles include the following:

1. Fluid Resuscitation: Postoperative DKA patients are often significantly dehydrated from preoperative fasting, intraoperative losses, and DKA's osmotic diuresis. Restoring circulation with IV fluids is the first priority. Isotonic saline is typically used initially unless heart failure is present. Diabetic ketoacidosis guidelines suggest starting with 1L in the first hour and assessing hemodynamics. In postoperative patients, consider using balanced crystalloid like Lactated Ringer's for large volumes to avoid exacerbating hyperchloremic acidosis. Once blood glucose drops below ∼200-250 mg/dL on insulin, add dextrose-containing fluids to prevent hypoglycemia while continuing ketone clearance.
2. Insulin Therapy: Insulin is the cornerstone of DKA treatment, suppressing ketogenesis and lowering blood glucose.[48] In nearly all postoperative DKA cases, IV insulin infusion is recommended for rapid titration and is standard for moderate-to-severe DKA. A typical regimen starts at 0.1 units/kg/hour after an initial IV bolus, with adjustments to maintain glucose decline of 50-75 mg/dL per hour. Insulin should continue until anion gap closure, not merely glucose normalization. In eu-DKA or during treatment, glucose may normalize before acidosis resolves, necessitating continued insulin with glucose supplementation to prevent hypoglycemia.
3. Electrolyte Management: DKA causes total-body electrolyte depletion, especially potassium. Even if serum potassium appears normal or elevated initially, total body potassium is depleted, and insulin therapy drives potassium intracellularly, risking severe hypokalemia. Postoperative DKA patients may have additional electrolyte disturbances from perioperative fluid shifts or nasogastric suction. Potassium must be repleted aggressively once levels fall below ∼5.0 mEq/L with adequate urine output. Typically, IV potassium (20-40 mEq per liter) is started with insulin unless K+ exceeds 5.2 mEq/L. If potassium is < 3.3 mEq/L, temporarily hold

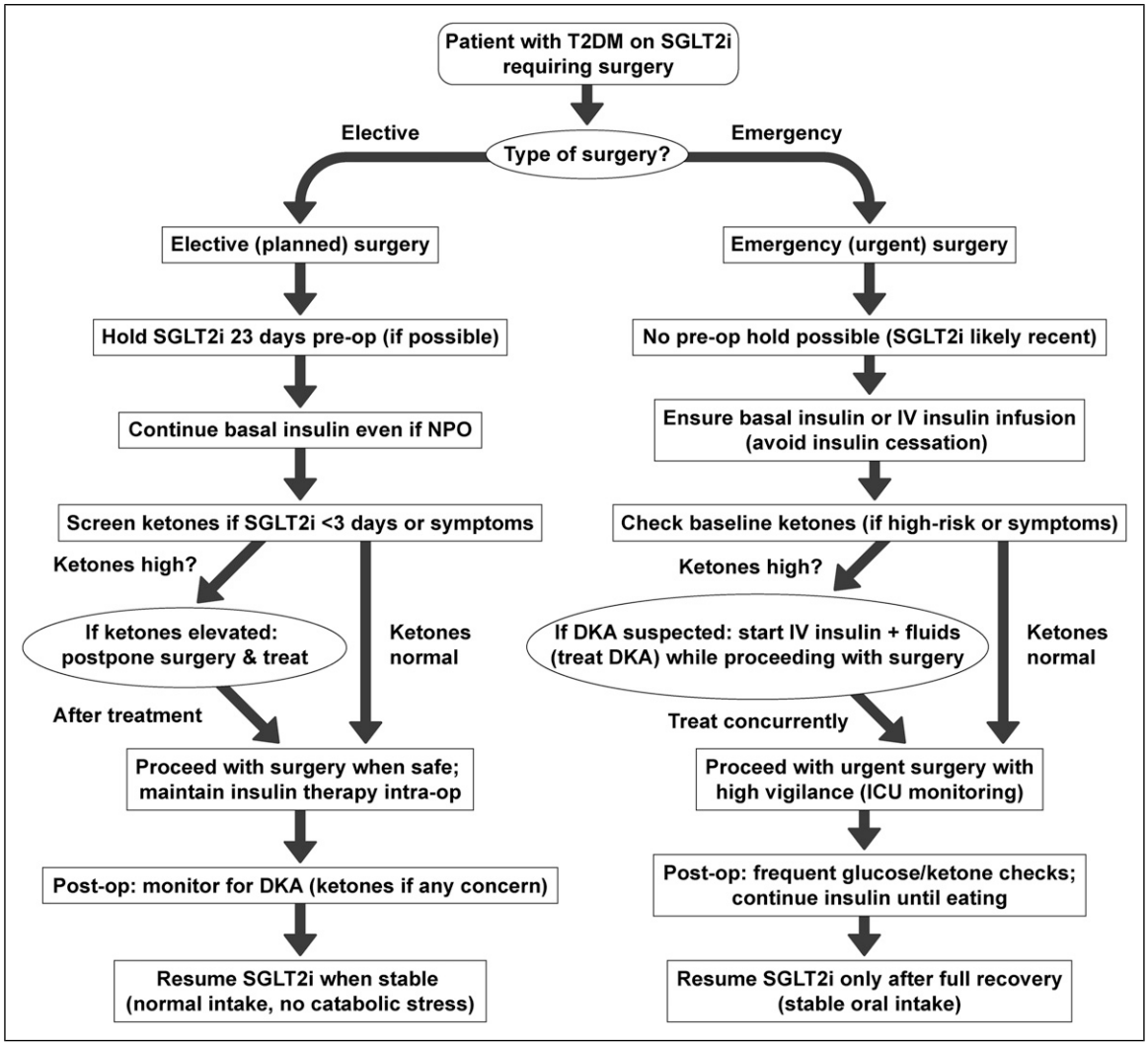

Figure 1. Algorithm of perioperative management for patients with type 2 diabetes on SGLT2 inhibitors

insulin and replete potassium first. Bicarbonate therapy is generally reserved for severe acidosis (pH <6.9) due to risks of paradoxical CNS acidosis and electrolyte shifts.

4. Addressing Precipitating Factors: Management must include treating trigger factors like infection or inadequate insulin dosing. For example, if an infection such as pneumonia or wound infection is identified, starting broad-spectrum antibiotics early is lifesaving in conjunction with DKA treatment. If the cause was an SGLT2 inhibitor that was inadvertently continued, that medication is of course stopped.

For patients with euglycemic DKA on SGLT2 inhibitors, one nuance is that they may not require as much insulin to control glucose, but they do require insulin to suppress ketogenesis.[49,50] Some reports have found that type 2 diabetes patients with eu-DKA can sometimes be managed with lower-intensity protocols. For instance, the case by Chandrakumar et al described a postoperative eu-DKA patient on ertugliflozin who was managed successfully without an insulin drip with fluids and carbohydrate-containing diet to stimulate endogenous insulin secretion. This requires preservation of beta-cell function and close monitoring.[3]

The safest approach in postoperative DKA is IV insulin until resolution, given unpredictable oral intake and postoperative stress. While mild cases in type 2 diabetics might resolve conservatively, insulin should not be withheld in type 1 diabetics or insulin-dependent type 2 diabetics, as endogenous recovery is impossible. Once DKA resolves (anion gap closed, bicarbonate normalized), transition to subcutaneous insulin carefully to prevent rebound ketoacidosis: overlap IV insulin with subcutaneous basal insulin for 1-2 hours.[51] A common error is stopping insulin infusion when patients resume eating without adequate long-acting insulin coverage, risking ketosis recurrence.

Perioperative Management Algorithm for SGLT2 Inhibitors

Algorithm delineating perioperative management for patients with type 2 diabetes on SGLT2 inhibitors, contrasting elective vs emergency surgery pathways (Figure 1). For elective surgeries, SGLT2 inhibitors should be held at least 3 days preoperatively per FDA guidance. Basal insulin should be maintained even when patients are fasting (NPO) to prevent insulin deficiency. Preoperative ketone screening is recommended if the SGLT2 inhibitor was taken within 3 days of surgery or if the patient has symptoms or high-risk features. If ketones are elevated, elective surgery should be postponed and insulin and fluids initiated to treat incipient ketoacidosis. Only proceed with surgery once ketosis is resolved and adequate insulin therapy is in place.

For emergency surgeries, holding the SGLT2 inhibitor is not feasible, so emphasis shifts to active management: ensure basal or IV insulin is continued to avoid relative insulin deficiency, check baseline and postoperatively ketones if the patient is high-risk or symptomatic, and have a low threshold to initiate IV insulin and fluids intraoperatively if DKA is suspected. Postoperatively, all patients on recent SGLT2 inhibitor are monitored closely for euglycemic DKA by frequent glucose and ketone checks.

Resumption of SGLT2 inhibitors in both scenarios is deferred until the patient is fully stable postoperatively—that is, normal oral intake has resumed, no surgical complications or active infection, and the patient's catabolic stress response has abated. This conservative delay in restarting SGLT2 inhibitor (often several days postoperatively) is advised to minimize recurrence of ketosis.

Conclusion

Postoperative DKA in patients with type 2 diabetes is an uncommon but life-threatening complication, increasingly associated with SGLT2 inhibitor therapy and often presenting as euglycemic DKA. Clinician awareness is crucial, as these cases may occur with only modest hyperglycemia, requiring a high index of suspicion and prompt recognition. Prevention strategies are paramount: current recommendations include optimizing glycemic control before surgery, discontinuing SGLT2 inhibitors at least 3 days preoperatively, maintaining basal insulin during the perioperative period, and monitoring ketones in high-risk patients. If DKA does occur, immediate aggressive management with intravenous fluids, insulin infusion, and electrolyte repletion is essential. The rising incidence linked to SGLT2 inhibitors necessitates protocolized perioperative management and electronic medical record alerts to flag at-risk patients; by implementing these safeguards and staying vigilant, clinicians and institutions can ensure early intervention, reduce postoperative DKA, and improve surgical outcomes.

Acknowledgment

Thanks for the resource support from Laboratory Animal Center, Kaohsiung Chang Gung Memorial Hospital.

ORCID iD

Ching-Hua Hsieh https://orcid.org/0000-0002-0945-2746

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was supported by grants from CORPG8M0591 and CORPG8M0592.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

References

1. Petersen C, Gyabaah F, Sotelo J, Yohanna S, Deoker A. A case of prolonged recovery for Post-percutaneous coronary intervention (PCI) sodium-glucose Cotransporter-2 (SGLT2) inhibitor-induced euglycemic diabetic ketoacidosis in a 28-Year-Old. *Cureus*. 2023;15(9):e45180.
2. Chen CC, Feng TY, Wang SC, Chen TH, Chou SJ, Jan HC. SGLT-2 inhibitor induced euglycemic diabetic ketoacidosis in post laparoscopic distal pancreatectomy: a case report. *Int J Surg Case Rep*. 2025;128:111006.
3. Chandrakumar HP, Chillumuntala S, Singh G, McFarlane SI. Postoperative euglycemic ketoacidosis in type 2 diabetes associated with sodium-glucose cotransporter 2 inhibitor: insights into pathogenesis and management strategy. *Cureus*. 2021;13(6):e15533.
4. Castellanos L, Tuffaha M, Koren D, Levitsky L. Management of diabetic ketoacidosis in children and adolescents with type 1 diabetes mellitus. *Pediatr Drugs*. 2020; 22(4):357-367.
5. Glaser N, Chu S, Hung B, et al. Acute and chronic neuroinflammation is triggered by diabetic ketoacidosis in a rat model. *BMJ Open Diabetes Research & Care*. 2020;8(2): e001793.
6. Andalib A, Elbahrawy A, Alshlwi S, et al. Diabetic ketoacidosis following bariatric surgery in patients with type 2 diabetes. *Diabetes Care*. 2016;39(8):e121-e122.
7. Aminian A, Kashyap SR, Burguera B, et al. Incidence and clinical features of diabetic ketoacidosis after bariatric and metabolic surgery. *Diabetes Care*. 2016; 39(4):e50-e53.
8. Chae H, Augustin R, Gatineau E, et al. SGLT2 is not expressed in pancreatic α- and β-cells, and its inhibition

does not directly affect glucagon and insulin secretion in rodents and humans. *Mol Metabol*. 2020;42:101071.
9. DeFronzo RA, Norton L, Abdul-Ghani M. Renal, metabolic and cardiovascular considerations of SGLT2 inhibition. *Nat Rev Nephrol*. 2017;13(1):11-26.
10. Dutta D, Khandelwal D. Sodium glucose transporter 2 inhibition, euglycemic ketosis and bone mineral loss: refining clinical practices. *Indian Journal of Endocrinology and Metabolism*. 2015;19:854-855.
11. Bloomgarden ZT. Sodium–glucose cotransporter 2 inhibitors and diabetic ketoacidosis. *J Diabetes*. 2016;8(2):175-176.
12. Berger J, Finck B. Beyond ketosis: the search for the mechanism underlying SGLT2-inhibitor benefit continues. *J Clin Investig*. 2024;134(24):e187097.
13. Wallenius K, Kroon T, Hagstedt T, et al. The SGLT2 inhibitor dapagliflozin promotes systemic FFA mobilization, enhances hepatic β-oxidation, and induces ketosis. *JLR (J Lipid Res)*. 2022;63(3):100176.
14. Yaribeygi H, Maleki M, Butler AE, Jamialahmadi T, Sahebkar A. New insights into cellular links between sodium–glucose cotransporter-2 inhibitors and ketogenesis. *J Cell Biochem*. 2022;123(12):1879-1890.
15. Steinhorn B, Wiener-Kronish J. Dose-dependent relationship between SGLT2 inhibitor hold time and risk for postoperative anion gap acidosis: a single-centre retrospective analysis. *Br J Anaesth*. 2023;131(4):682-686.
16. Wang KM, Isom RT. SGLT2 inhibitor-induced euglycemic diabetic ketoacidosis: a case report. *Kidney Medicine*. 2020; 2(2):218-221.
17. Osafehinti D, Okoli O, Karam J. A case of SGLT2 inhibitor-associated euglycemic diabetic ketoacidosis following coronary artery bypass surgery. *AACE Clinical Case Reports*. 2020;7(1):20-22.
18. Gomez-Sanchez CM, Wu BX, Gotts JE, Chang RW. Euglycemic diabetic ketoacidosis following major vascular surgery is a new item on the differential for postoperative acidosis. *Journal of Vascular Surgery Cases and Innovative Techniques*. 2021;7(4):778-780.
19. Smith A, Holtrop J, Sadoun M. Post-operative euglycemic diabetic ketoacidosis in a patient with SGLT-2 inhibitor use and recent sleeve gastrectomy. *Cureus*. 2021;13(4):e14297.
20. Ritchie DT, Dixon J. SGLT-2 inhibitor associated euglycaemic diabetic ketoacidosis in an orthopaedic trauma patient. *BMJ Case Rep*. 2022;15(9):e250233.
21. Bazan DZ, Esqueda L, Ibrahim A, et al. Sodium-glucose Cotransporter-2 inhibitors and euglycemic diabetic ketoacidosis: a case series of three post-surgical patients. *Cureus*. 2025;17(5):e84665.
22. Lui DTW, Wu T, Au ICH, et al. A population-based study of SGLT2 inhibitor-associated postoperative diabetic ketoacidosis in patients with type 2 diabetes. *Drug Saf*. 2023; 46(1):53-64.
23. Chow E, Clement S, Garg R. Euglycemic diabetic ketoacidosis in the era of SGLT-2 inhibitors. *BMJ Open Diabetes Research & Care*. 2023;11(5):e003666.
24. Morace C, Lorello G, Bellone F, et al. Ketoacidosis and SGLT2 inhibitors: a narrative review. *Metabolites*. 2024; 14(5):264.
25. Dixit AA, Bateman BT, Hawn MT, Odden MC, Sun EC. Preoperative SGLT2 inhibitor use and postoperative diabetic ketoacidosis. *JAMA Surg*. 2025;160(4):423-430.
26. Seki H, Ideno S, Shiga T, et al. Sodium-glucose cotransporter 2 inhibitor-associated perioperative ketoacidosis: a systematic review of case reports. *J Anesth*. 2023; 37(3):465-473.
27. Mehta PB, Robinson A, Burkhardt D, Rushakoff RJ. Inpatient perioperative euglycemic diabetic ketoacidosis due to sodium-glucose Cotransporter-2 inhibitors - lessons from a case series and strategies to decrease incidence. *Endocr Pract*. 2022;28(9):884-888.
28. Crowley K, Scanaill P, Hermanides J, Buggy DJ. Current practice in the perioperative management of patients with diabetes mellitus: a narrative review. *Br J Anaesth*. 2023; 131(2):242-252.
29. Dunn BK, Coore H, Bongu N, et al. Treatment challenges and controversies in the management of critically ill diabetic ketoacidosis (DKA) patients in intensive care units. *Cureus*. 2024;16(9):e68785.
30. Tian B, Cao X, Cheng J, et al. Aspiration pneumonia in diabetic ketoacidosis: a decade-long analysis of occurrence, associated risk factors and clinical outcomes in 530,700 US hospitalizations. *Diabetol Metab Syndr*. 2025;17(1):395.
31. Chen YL, Weng SF, Yang CY, Wang JJ, Tien KJ. Long-term risk of stroke in type 2 diabetes patients with diabetic ketoacidosis: a population-based, propensity score-matched, longitudinal follow-up study. *Diabetes Metab*. 2017;43(3):223-228.
32. Fahda MJF, Gostevcic N, Hammad YM, Ahmed SMG. Preoperative management of diabetes mellitus: a comparative narrative review of the recommendations of three professional organizations with hamad medical corporation guidelines. *Qatar Med J*. 2025;2025(1):23.
33. Shivganesh BRD, Karim HMR, Agrawal N, Kumar M. The relation of preoperative HbA1c level with intraoperative and postoperative complications in Type-2 diabetic patients: an observational study. *Cureus*. 2024; 16(7):e64487.
34. Diabetes care in the hospital: standards of care in Diabetes-2024. *Diabetes Care*. 2024;47(Suppl 1):S295-s306.
35. Priya V, Bais PS, Rastogi A, Shamim R, Aggarwal A, Patro A. Peri-operative management of diabetes mellitus - a survey of current practices among Indian anesthesiologists. *J Anaesthesiol Clin Pharmacol*. 2023;39(3):411-421.
36. Gregory S. Perioperative management of glucose-lowering drugs: comment. *Anesthesiology*. 2021;134(2):349-350.
37. Muermann MM, Retief PJ. Perioperative management of patients with diabetes mellitus: ketone screening. *Can J Anaesth*. 2021;68(6):923-924.
38. Bhasin N, Sharma MK, Yadav N, Kumawat MK. Diabetes ketoacidosis is a cause of concern in sodium-glucose Co-

Transporter-2 inhibitors medication. *International Journal of Life Science and Pharma Research*. 2022;13:55-67.
39. Thiruvenkatarajan V, Nanjappa N, Sembu M, Meyer E, Van Wijk R, Jesudason D. An analysis of the Australian therapeutic goods administration database of adverse event notifications of diabetic ketoacidosis associated with sodium-glucose cotransporter-2 inhibitors in surgical patients. *Anaesth Intensive Care*. 2020;48(1):70-72.
40. Ito T, Sugasawa G, Suzuki F, et al. Insulin and glucose infusion could prevent euglycemic diabetic ketoacidosis associated with sodium-glucose cotransporter 2 inhibitors. *Indian J Thorac Cardiovasc Surg*. 2022;38(1):87-91.
41. Gosmanov AR, Gosmanova EO, Dillard-Cannon E. Management of adult diabetic ketoacidosis. *Diabetes Metab Syndr Obes*. 2014;7:255-264.
42. Eledrisi MS, Elzouki AN. Management of diabetic ketoacidosis in adults: a narrative review. *Saudi Journal of Medicine & Medical Sciences*. 2020;8(3):165-173.
43. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. *Diabetes Care*. 2009;32(7):1335-1343.
44. Kietaibl A, Fasching P, Glaser K, Petter-Puchner A. New diabetic medication sodium-glucose Cotransporter-2 inhibitors can induce euglycemic ketoacidosis and mimic surgical diseases: a case report and review of literature. *Front Surg*. 2022;9:828649.
45. Viswanath NG, Sharma M, Bandlamudi N, et al. Sodium-glucose Co-transporter-2 inhibitors induced diabetic ketoacidosis in patients undergoing bariatric surgery: a systematic review of case reports and case series. *Obes Surg*. 2022;33(1):339-344.
46. Kather FAA, Iyengar JJ, Esfandiari NH, et al. 1130-P: enhancing safety—managing individuals on SGLT-2 inhibitors during the perioperative period. *Diabetes*. 2024; 73(Supplement 1):1130-P.
47. Zhou J, Zhang P, Tan Z, et al. Enhanced recovery after surgery in elective cesarean section patients with gestational diabetes mellitus does not lead to glucose-related maternal and neonatal complications. *Front Endocrinol*. 2024;15:1403754.
48. Chow L, Valesky W. Subcutaneous rapid-acting insulin analogs for diabetic ketoacidosis. *Acad Emerg Med*. 2021; 28(6):700-702.
49. TakallooBakhtiari A, Butina JP, Funderburk SG, Brundavanam H. Euglycemic DKA associated with SGLT2 inhibitors. *Journal of the Endocrine Society*. 2021; 5(Supplement_1):A382.
50. Chalmers S, Moraes A. 326: severe euglycemic ketoacidosis IN NON-INSULIN-DEPENDENT diabetes. *Crit Care Med*. 2020;48:326.
51. Welch AA, Toro-Tobon D, Rachmasari KN, et al. Improving intravenous and subcutaneous insulin overlap during treatment of diabetic ketoacidosis: a quality improvement project. *Mayo Clin Proc: Innovations, Quality & Outcomes*. 2024;8(3):293-300.

Appendix

Abbreviations

CI	Confidence interval
DKA	Diabetic ketoacidosis
eu-DKA	Euglycemic diabetic ketoacidosis
FDA	Food and Drug Administration
GI	Gastrointestinal
HbA1c	Hemoglobin A1c
ICU	Intensive care unit
IRR	Incidence rate ratio
IV	Intravenous
SGLT2	Sodium-glucose cotransporter-2