Section: Medicine

Systematic Review Article

CLINICAL PROFILE AND MANAGEMENT OUTCOMES OF EUGLYCEMIC DIABETIC KETOACIDOSIS: A SYSTEMATIC REVIEW

***Habiba Mohammed Usman Dudhmal*[1]**

[1]*MBBS, Medical College for Women and Hospital, Dhaka, Bangladesh*

Received *: 02/02/2026*
Received in revised form *: 18/03/2026*
Accepted *: 05/04/2026*

Corresponding Author:
Dr. Habiba Mohammed Usman Dudhmal,
MBBS, Medical College for Women and Hospital, Dhaka, Bangladesh.
Email: habibausman707@gmail.com

DOI: 10.70034/ijmedph.2026.2.93

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 551-556

ABSTRACT

Background: Euglycemic diabetic ketoacidosis (EDKA) is a variant of diabetic ketoacidosis marked by metabolic acidosis and ketosis despite normal or only mildly elevated blood glucose levels. In contrast to classical diabetic ketoacidosis (DKA), the lack of significant hyperglycemia can obscure recognition and delay timely management. The growing use of sodium–glucose cotransporter-2 (SGLT2) inhibitors, together with triggers such as infection, prolonged fasting, pregnancy, and missed insulin doses, has led to an increasing number of reported cases. Therefore, a systematic review is needed to integrate current evidence and strengthen clinical recognition and understanding of this emerging condition. The objective is to comprehensively evaluate the demographic and clinical characteristics, precipitating factors, management strategies, and outcomes—including recovery, complications, intensive care requirement, and mortality—of patients diagnosed with euglycemic diabetic ketoacidosis.

Materials and Methods: This study was conducted as a systematic review in accordance with PRISMA guidelines. A comprehensive literature search was performed in PubMed/MEDLINE, Scopus, Web of Science, Embase, and the Cochrane Library using relevant keywords related to euglycemic diabetic ketoacidosis. Observational studies, case series, randomized controlled trials, and case reports, review articles, meta-analysis were included. Titles, abstracts, and full-text articles were independently screened according to predefined eligibility criteria.

Results: Euglycemic diabetic ketoacidosis affects all age groups and is increasingly linked to SGLT2 inhibitor use. It presents with milder acidosis and lower glucose levels, which may delay diagnosis. Triggers include reduced intake, infection, pregnancy, insulin omission, surgical stress, low-carbohydrate diets, and Sodium–Glucose Cotransporter-2 inhibitors (SGLT2 inhibitors). Outcomes are comparable to classical DKA with appropriate treatment, though hypoglycemia during therapy is more frequent. Early recognition and prompt management are essential to reduce complications. Management included fluid resuscitation, electrolyte correction, insulin infusion, and dextrose supplementation. Euglycemic DKA required shorter insulin duration but had higher hypoglycemia risk. Mortality was under 5% with standardized treatment.

Conclusion: Euglycemic DKA is a serious diabetic emergency that may be overlooked due to near-normal glucose levels and is increasingly associated with SGLT2 inhibitor use, particularly in perioperative settings. Early recognition, patient education, routine ketone monitoring, and preventive strategies such as the STICH and STOP DKA protocols are essential to reduce risk. Prompt management and structured follow-up remain key to preventing recurrence and improving outcomes.

Keywords: Euglycemic diabetic ketoacidosis, SGLT2 inhibitors, Diabetic ketoacidosis, Type 1 diabetes mellitus, Insulin therapy, Clinical profile, Management outcomes.

INTRODUCTION

Euglycemic diabetic ketoacidosis (EDKA) is defined by high anion gap metabolic acidosis with ketonemia or ketonuria despite blood glucose levels below 200 mg/dL. The absence of marked hyperglycemia can delay diagnosis, requiring strong clinical suspicion and exclusion of alternative causes (Rawla P et al., 2017).[1]

It is linked to conditions such as starvation, infection, pregnancy, alcohol use, liver disease, and SGLT2 inhibitor therapy. It results from insulin deficiency leading to excessive ketone production, and patients commonly present with symptoms such as nausea and fatigue. Management includes intravenous fluids, insulin with glucose support, and treatment of the underlying trigger (Long B et al., 2021).[2]

Euglycemic DKA presents a diagnostic challenge because normal blood glucose levels can mask underlying ketoacidosis, potentially delaying treatment. Despite the absence of marked hyperglycemia, it remains a medical emergency requiring prompt recognition and timely intervention (Modi A et al., 2017).[3]

Euglycemic DKA (eu-DKA) is a life-threatening condition that can occur in both type 1 and type 2 diabetes, characterized by ketoacidosis with blood glucose levels typically below 200 mg/dL, which may delay diagnosis. Its incidence has increased with the widespread use of SGLT2 inhibitors. Other associated triggers include pregnancy, reduced caloric intake, alcohol misuse, prior insulin use, cocaine abuse, pancreatitis, sepsis, and chronic liver disease. Prompt emergency management is essential and involves aggressive fluid resuscitation, correction of electrolyte imbalances, and continuous insulin infusion until resolution of acidosis (Barski L et al., 2019).[4]

Diabetic ketoacidosis (DKA) is characterized by hyperglycemia, metabolic acidosis, and ketosis, whereas hyperosmolar hyperglycemic state (HHS) involves severe hyperglycemia, hyperosmolality, and dehydration with minimal ketosis. Both result from insulin deficiency and elevated counterregulatory hormones. While DKA is more common in type 1 diabetes, it can also occur in type 2 diabetes during acute stress. This statement outlines the diagnosis, management, and prevention of DKA and HHS in adults (Kitabchi AE et al., 2009).[5]

Given the growing recognition of eu-DKA and its potential for delayed diagnosis, a comprehensive synthesis of available evidence is necessary. This systematic review aims to evaluate the clinical characteristics, precipitating factors, management strategies, and outcomes of patients diagnosed with euglycemic diabetic ketoacidosis, thereby providing clinicians with consolidated evidence to facilitate early recognition and optimize management.

Objectives: This systematic review aimed to comprehensively evaluate the demographic and clinical characteristics, precipitating factors, management strategies, and outcomes—including recovery, complications, intensive care requirement, and mortality—of patients diagnosed with euglycemic diabetic ketoacidosis.

MATERIALS AND METHODS

This study was conducted as a systematic review in accordance with PRISMA guidelines. A comprehensive literature search was performed in PubMed/MEDLINE, Scopus, Web of Science, Embase, and the Cochrane Library using relevant keywords Euglycemic diabetic ketoacidosis, SGLT2 inhibitors, Diabetic ketoacidosis, Type 1 diabetes mellitus, Insulin therapy, Clinical profile, Management outcomes. Observational studies, case series, randomized controlled trials, case reports, review articles, meta-analysis were included. Irrelevant studies, studies without extractable data or clearly defined outcome measures, conference abstracts, were excluded. Titles, abstracts, and full-text articles were independently screened according to predefined eligibility criteria. Data were extracted using a standardized format including study characteristics, patient demographics, precipitating factors, laboratory findings, treatment modalities, and clinical outcomes. Quality assessment was performed using appropriate appraisal tools, and a qualitative synthesis was conducted.

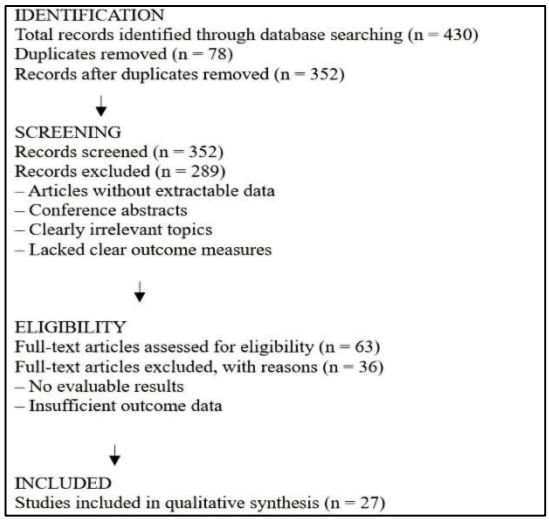

Figure 1: PRISMA Flow chart of Study Selection Process.

RESULTS

Clinical Profile

Demographic variables: Munro JF et al., 1973, studied that among 211 episodes of diabetic decompensation, 37 involved severe euglycemic ketoacidosis (glucose <300 mg/dL, bicarbonate ≤10 mEq/L). Most patients were young with insulin-dependent diabetes, and vomiting with reduced carbohydrate intake despite continued insulin was common. Treatment included fluids, electrolytes,

insulin with dextrose support, minimal bicarbonate use, and close monitoring. All patients survived.[6]

In the retrospective study by Sell J et al., 2023, of 629 adult DKA patients, 44 had euglycemic DKA (<250 mg/dL). Compared with hyperglycemic cases, they presented with milder acidosis, lower glucose and potassium levels, and required shorter insulin infusion. Common triggers included prior insulin use, reduced oral intake, and SGLT2 inhibitors. Hypoglycemia during treatment was more frequent in the euglycemic group, while hypokalemia rates were similar.[7]

Over 10 years, 11 episodes (2%) of DKA occurred in pregnancies complicated by diabetes despite close monitoring. About one-third had glucose levels below 200 mg/dL, and most presented with nausea, vomiting, and poor intake. Two cases required cesarean delivery for fetal distress, and one fetal death occurred. These findings emphasize that normal or near-normal glucose levels do not rule out DKA in pregnancy, and symptomatic patients should be evaluated for ketosis (Cullen MT et al., 1996).[8]

Euglycemic DKA is frequently reported in middle-aged women with type 2 diabetes, particularly those treated with SGLT2 inhibitors, often in combination with metformin. Use of SGLT2 inhibitors has been associated with an approximately 3.7-fold higher risk of DKA compared with other antidiabetic therapies (Dutta S et al., 2022).[9]

Euglycemic DKA occurs in patients on SGLT2 inhibitors, especially those with low body mass index (BMI), and may be triggered by illness, reduced intake, vomiting, dehydration, or decreased insulin use. A 34-year-old man with type 2 diabetes on dapagliflozin–metformin developed eu-DKA despite glucose levels below 250 mg/dL and improved with intravenous insulin and fluids. Early recognition is crucial, as management mirrors standard DKA treatment with hydration and insulin therapy (Sarno MJF et al., 2023).[10]

Precipitating factors: Common triggers of euglycemic DKA, such as caloric restriction and surgical stress, along with the absence of significant hyperglycemia, may delay diagnosis and increase the risk of serious complications. Therefore, clinicians should carefully review antidiabetic medications and consider eu-DKA in any patient with diabetes presenting with high anion gap metabolic acidosis. As SGLT2 inhibitor–associated DKA frequently occurs in the perioperative period, appropriate preoperative discontinuation of these agents is essential to reduce risk (Goto S et al., 2021).[11]

SGLT2 inhibitors are antidiabetic agents that lower blood glucose by blocking renal glucose reabsorption, leading to glucosuria. Beyond glycemic control, they promote weight loss, reduce blood pressure and uric acid levels, and offer cardiovascular and renal protection in high-risk patients with type 2 diabetes (Qiu H et al., 2017).[12]

SGLT2 inhibitors alter the insulin-to-glucagon ratio in a way that promotes ketone production, increasing the risk of ketosis in susceptible individuals. However, the degree of insulin deficiency and resistance associated with these agents is milder than that seen in classical diabetic ketoacidosis, resulting in less marked hepatic glucose overproduction and peripheral underutilization. Because these drugs also reduce renal glucose reabsorption, blood glucose levels often remain only mildly elevated. This relative absence of significant hyperglycemia can delay recognition of SGLT2 inhibitor–associated ketoacidosis, as patients and clinicians commonly rely on plasma glucose levels for routine monitoring (Palmer BF et al., 2016).[13]

With the widespread use of SGLT2 inhibitors, euglycemic ketoacidosis (eu-KA) has gained renewed attention, as these agents can trigger ketoacidosis despite normal or mildly elevated blood glucose levels. Eu-KA may also occur in non-diabetic states such as pregnancy, caloric restriction, glycogen storage disorders, impaired gluconeogenesis (e.g., alcohol misuse or chronic liver disease), and cocaine use. The absence of significant hyperglycemia makes diagnosis challenging (Bonora BM et al., 2020).[14]

The growing use of SGLT2 inhibitors has led to increased recognition of euglycemic DKA, a rare but serious complication. It results from glucosuria-induced carbohydrate deficit and a raised glucagon–insulin ratio, promoting ketogenesis despite normal glucose levels, which may delay diagnosis. Early recognition, identification of triggers, and prompt management are essential for prevention and improved outcomes (Koceva A et al., 2024).[15]

The absence of marked hyperglycemia can delay recognition, particularly in emergency and critical care settings. Although uncommon, it occurs in both type 1 and type 2 diabetes and has become more frequent with the use of SGLT2 inhibitors. Other triggers include pregnancy, prolonged fasting, bariatric surgery, gastroparesis, insulin pump failure, cocaine use, chronic liver disease, and glycogen storage disorders (Nasa P et al., 2021).[16]

In a study by Mistry S et al., 2020, found that two patients with type 2 diabetes developed euglycemic DKA while using SGLT2 inhibitors in combination with a ketogenic diet. One case occurred after a single dose of empagliflozin, and the other within a week of starting a ketogenic diet while on therapy. Although this association has been reported, it may be underrecognized. Clinicians should advise patients on SGLT2 inhibitors to avoid very low-carbohydrate diets and maintain a high index of suspicion for DKA in symptomatic individuals.[17]

Fukuyama Y et al., 2020, reported a case of a 54-year-old woman taking canagliflozin developed euglycemic ketoacidosis after six days on a strict low-carbohydrate diet and was later diagnosed with type 1 diabetes. This case emphasizes caution when combining SGLT2 inhibitors with low-carbohydrate diets due to increased DKA risk.[18]

FDA [Food and Drug Administration] Adverse Event Reporting System (FAERS) recorded 259 cases of acidosis with SGLT2 inhibitors, including 192 cases of ketoacidosis, compared to 477 acidosis cases with

Dipeptidyl Peptidase-4 (DPP-4) inhibitors, of which 71 were ketoacidosis. When adjusted for estimated patient exposure, SGLT2 inhibitors were associated with an approximately 14-fold higher risk of acidosis. Among 51 reports with available metabolic data, 20 involved type 1 diabetes, 25 type 2 diabetes, and 6 unspecified diabetes. After excluding type 1 diabetes, the risk of acidosis in type 2 diabetes remained about seven times higher with SGLT2 inhibitors. Notably, 71% of cases presented as euglycemic ketoacidosis (Blau JE et al., 2017).[19]

In a retrospective review by Sell J et al., 2023, found that among 629 DKA cases, 44 were euglycemic and 585 hyperglycemic. Patients with euglycemic DKA presented with milder acidosis (higher pH and bicarbonate, lower anion gap) and lower initial glucose (195 vs 561 mg/dL) and potassium levels. Common triggers included prior insulin use (57%), reduced intake with ongoing insulin therapy (29%), and SGLT2 inhibitors (14%). They required a shorter duration of insulin infusion (13.5 vs 19.4 hours), with similar time to metabolic recovery. However, hypoglycemia during treatment was more frequent in euglycemic DKA, while hypokalemia rates were comparable.[7]

Table 1: Key finding of precipitating factors associated with euglycemic diabetic ketoacidosis

Author (Year)	Key Findings
Goto S et al., 2021	Caloric restriction and surgical stress trigger eu-DKA; absence of marked hyperglycemia delays diagnosis. Preoperative SGLT2 inhibitor discontinuation recommended.
Qiu H et al., 2017	SGLT2 inhibitors reduce renal glucose reabsorption; promote weight loss, lower BP and uric acid, and provide cardiovascular and renal protection.
Palmer BF et al., 2016	SGLT2 alter increase glucagon–insulin ratio, promoting ketogenesis with only mild hyperglycemia, delaying recognition.
Bonora BM et al., 2020	euKA linked to SGLT2 inhibitors and non-diabetic states (pregnancy, fasting, alcohol misuse, liver disease, cocaine use); diagnosis challenging due to absence of significant hyperglycemia.
Koceva A et al., 2024	eu-DKA results from glucosuria-induced carbohydrate deficit and raised glucagon–insulin ratio; early recognition essential.
Nasa P et al., 2021	Absence of significant hyperglycemia may delay diagnosis, especially in emergency settings. Though rare, it occurs in both type 1 and type 2 diabetes and has increased with SGLT2 inhibitor use. Triggers include pregnancy, fasting, bariatric surgery, gastroparesis, pump failure, cocaine use, liver disease.
Mistry S et al., 2020	SGLT2 inhibitors + ketogenic diet precipitated eu-DKA; avoid very low-carbohydrate diets.
Fukuyama Y et al., 2020	Canagliflozin + strict low-carbohydrate diet triggered eu-DKA; caution advised.
Blau JE et al., 2017	FAERS: ~14-fold higher acidosis risk with SGLT2 inhibitors; 71% presented as eu-DKA.
Sell J et al., 2023	44 out of 629 DKA cases were euglycemic; milder acidosis, lower glucose, required shorter insulin infusion; hypoglycemia more frequent during treatment.

Management Outcomes: Management consisted of aggressive fluid and electrolyte replacement along with high-dose insulin therapy administered with adequate carbohydrate supplementation, often using 10% dextrose infusion. Bicarbonate therapy was either avoided or used cautiously, and treatment response was monitored through serial plasma bicarbonate measurements. All patients recovered without mortality (Munro JF et al., 1973).[6]

The increased use of SGLT2 inhibitors in type 2 diabetes has led to a rise in euglycemic DKA, though other triggers include pregnancy, reduced caloric intake, alcohol excess, insulin dose changes, pancreatitis, sepsis, and chronic liver disease. Both eu-DKA and classical DKA require urgent management with prompt fluid resuscitation, electrolyte correction, and continuous insulin infusion until metabolic parameters normalize. Because glucose levels are often only mildly elevated, higher-concentration dextrose solutions may be necessary to permit adequate insulin therapy and resolution of acidosis (Barski L et al., 2019).[4]

Diagnosis requires a high index of suspicion and confirmation of metabolic acidosis with elevated ketones while excluding other causes of high anion gap acidosis. Management follows standard DKA principles, including fluid resuscitation, electrolyte correction, and insulin therapy, with concurrent dextrose infusion to resolve ketosis and prevent hypoglycemia (Nasa P et al., 2021).[16]

DKA is a serious complication in pregnancy, associated with maternal and fetal risk. Euglycemic DKA, defined by ketoacidosis with normal glucose levels, is rare and usually seen in type 1 diabetes. This case reports a 37-year-old woman with type 2 diabetes who developed euglycemic DKA (78 mg/dL) during pregnancy and improved with intravenous dextrose and insulin. Prompt recognition is essential even in type 2 diabetes (Tarif N et al., 2007).[20]

Compared with hyperglycemic DKA managed under the same protocol, patients with euglycemic DKA required nearly six fewer hours of insulin infusion but had more than a threefold higher risk of hypoglycemia. Further studies are needed to refine treatment approaches and reduce complications, particularly treatment-related hypoglycemia (Sell J et al., 2023).[7]

Joseph F et al., 2009, described a case of euglycemic DKA in a patient with type 1 diabetes precipitated by prolonged starvation due to severe depression. Despite significant acidosis and ketonuria, blood glucose was only 105 mg/dL. The patient improved with fluid resuscitation and correction of acidosis, followed by treatment of underlying depression. The case underscores the need to assess acid–base status in ill diabetic patients even without hyperglycemia

and to consider psychiatric causes, such as depression-related anorexia, as potential triggers.[21]

DKA can occur in both type 1 and type 2 diabetes, and SGLT2 inhibitors have led to recognition of euglycemic DKA, where ketoacidosis develops without marked hyperglycemia. Management includes fluids, electrolytes, insulin, and early glucose infusion, though recovery may be prolonged due to the drug's lasting effect (Mehta AE et al., 2025).[22]

In type 1 diabetes, the likelihood of developing DKA and the therapeutic effects of SGLT2 inhibitors are influenced by factors such as baseline body mass index, degree of insulin resistance, the extent of insulin dose reduction relative to underlying insulin sensitivity, and the presence of volume depletion. Careful consideration of these variables may help identify patients who are most likely to benefit from therapy while minimizing the risk of DKA (Musso G et al., 2020).[23]

CONCLUSION

Euglycemic ketoacidosis can be difficult to recognize, as normal or only mildly elevated blood glucose levels may obscure the diagnosis and delay appropriate management (Bonora BM et al., 2020).[14]

DKA and euglycemic DKA are serious diabetic emergencies. While classic DKA presents with marked hyperglycemia, euglycemic DKA occurs with near-normal glucose levels and is often linked to SGLT2 inhibitors, making diagnosis more challenging (Ju HH, 2025).[24]

As SGLT2 inhibitor–related DKA commonly occurs in the perioperative setting, appropriate preoperative discontinuation of these agents is essential to reduce risk (Goto S et al., 2021).[11]

For patients with type 1 diabetes using SGLT2 inhibitors with insulin (off-label), clear education on DKA risk, routine ketone monitoring, and early symptom recognition is essential. Use of the STICH protocol (Stop SGLT2 inhibitor, Take insulin, Ingest carbohydrates, Hydrate) can help reduce risk and support safer use if approved by regulatory authorities (Garg SK et al., 2018).[25]

Management includes prompt clinical assessment, correction of metabolic abnormalities, treatment of underlying and coexisting conditions, optimization of long-term diabetes care, and strategies to prevent recurrence (Fasanmade OA et al., 2008).[26]

Diabetic ketoacidosis is a known complication of type 1 diabetes, and the risk rises with SGLT inhibitor use. To balance benefits while reducing this risk, the "STOP DKA Protocol" was developed as a simple, practical strategy to help prevent DKA in patients with type 1 diabetes receiving SGLT inhibitors (Goldenberg RM et al., 2019).[27]

Euglycemic diabetic ketoacidosis is a serious and often underrecognized complication, particularly associated with SGLT2 inhibitor use. The absence of marked hyperglycemia can delay diagnosis and treatment, especially in perioperative settings and in patients with type 1 diabetes using these agents off-label.

Risk reduction requires appropriate patient selection, preoperative discontinuation of SGLT2 inhibitors, patient education, ketone monitoring, and early symptom recognition. Protocols such as STICH and STOP DKA provide practical preventive strategies. Prompt diagnosis, standardized management, and ongoing preventive education are essential to reduce morbidity and recurrence.

Future studies should be undertaken to establish clear associations between SGLT2 inhibitor use and the incidence of euglycemic DKA, and to identify independent risk factors and high-risk populations. Prospective research is needed to optimize perioperative management, ketone monitoring strategies, and preventive protocols. Long-term outcome data will help balance therapeutic benefits with metabolic safety.

Acknowledgment

The author gratefully acknowledges the researchers and investigators whose published studies provided the basis for this systematic review. Author also extends sincere appreciation to mentors and colleagues for their guidance, constructive feedback, and ongoing support, which significantly strengthened the quality and rigor of this work. The author declares no conflicts of interest and confirms that this study received no external funding.

REFERENCES

1. Rawla P, Vellipuram AR, Bandaru SS, Pradeep Raj J. Euglycemic diabetic ketoacidosis: a diagnostic and therapeutic dilemma. Endocrinol Diabetes Metab Case Rep. 2017 Sep 4;2017:17-0081. doi: 10.1530/EDM-17-0081. PMID: 28924481; PMCID: PMC5592704.
2. Long B, Lentz S, Koyfman A, Gottlieb M. Euglycemic diabetic ketoacidosis: Etiologies, evaluation, and management. Am J Emerg Med. 2021 Jun;44:157-160. doi: 10.1016/j.ajem.2021.02.015. Epub 2021 Feb 16. PMID: 33626481.
3. Modi A, Agrawal A, Morgan F. Euglycemic Diabetic Ketoacidosis: A Review. Curr Diabetes Rev. 2017;13(3):315-321. doi: 10.2174/1573399812666160421121307. PMID: 27097605.
4. Barski L, Eshkoli T, Brandstaetter E, Jotkowitz A. Euglycemic diabetic ketoacidosis. Eur J Intern Med. 2019 May;63:9-14. doi: 10.1016/j.ejim.2019.03.014. Epub 2019 Mar 23. PMID: 30910328.
5. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care. 2009 Jul;32(7):1335-43. doi: 10.2337/dc09-9032. PMID: 19564476; PMCID: PMC2699725.
6. Munro JF, Campbell IW, McCuish AC, Duncan LJ. Euglycaemic diabetic ketoacidosis. Br Med J. 1973 Jun 9;2(5866):578-80. doi: 10.1136/bmj.2.5866.578. PMID: 4197425; PMCID: PMC1592207.
7. Sell J, Haas NL, Korley FK, Cranford JA, Bassin BS. Euglycemic Diabetic Ketoacidosis: Experience with 44 Patients and Comparison to Hyperglycemic Diabetic Ketoacidosis. West J Emerg Med. 2023 Nov;24(6):1049-1055. doi: 10.5811/westjem.60361. PMID: 38165186; PMCID: PMC10754195.
8. Cullen MT, Reece EA, Homko CJ, Sivan E. The changing presentations of diabetic ketoacidosis during pregnancy. Am J Perinatol. 1996 Oct;13(7):449-51. doi: 10.1055/s-2007-994386. PMID: 8960616.

9. Dutta S, Kumar T, Singh S, Ambwani S, Charan J, Varthya SB. Euglycemic diabetic ketoacidosis associated with SGLT2 inhibitors: A systematic review and quantitative analysis. J Family Med Prim Care. 2022 Mar;11(3):927-940. doi: 10.4103/jfmpc.jfmpc_644_21. Epub 2022 Mar 10. PMID: 35495849; PMCID: PMC9051698.
10. Sarno MJF, Hernandez DPF, Matulac MO. "Normal but Catastrophic" Euglycemic Diabetic Ketoacidosis Precipitated by Sodium-Glucose Cotransporter-2 Inhibitor Use: A Case Report. Cureus. 2023 Nov 22;15(11):e49236. doi: 10.7759/cureus.49236. PMID: 38143642; PMCID: PMC10740381.
11. Goto S, Ishikawa JY, Idei M, Iwabuchi M, Namekawa M, Nomura T. Life-Threatening Complications Related to Delayed Diagnosis of Euglycemic Diabetic Ketoacidosis Associated with Sodium-Glucose Cotransporter-2 Inhibitors: A Report of 2 Cases. Am J Case Rep. 2021 Mar 16;22:e929773. doi: 10.12659/AJCR.929773. PMID: 33723205; PMCID: PMC7980085.
12. Qiu H, Novikov A, Vallon V. Ketosis and diabetic ketoacidosis in response to SGLT2 inhibitors: Basic mechanisms and therapeutic perspectives. Diabetes Metab Res Rev. 2017 Jul;33(5). doi: 10.1002/dmrr.2886. Epub 2017 Feb 23. PMID: 28099783.
13. Palmer BF, Clegg DJ, Taylor SI, Weir MR. Diabetic ketoacidosis, sodium glucose transporter-2 inhibitors and the kidney. J Diabetes Complications. 2016 Aug;30(6):1162-6. doi: 10.1016/j.jdiacomp.2016.05.008. Epub 2016 May 10. PMID: 27240541.
14. Bonora BM, Avogaro A, Fadini GP. Euglycemic Ketoacidosis. Curr Diab Rep. 2020 May 19;20(7):25. doi: 10.1007/s11892-020-01307-x. PMID: 32424730.
15. Koceva A, Kravos Tramšek NA. From Sweet to Sour: SGLT-2-Inhibitor-Induced Euglycemic Diabetic Ketoacidosis. J Pers Med. 2024 Jun 21;14(7):665. doi: 10.3390/jpm14070665. PMID: 39063919; PMCID: PMC11277626.
16. Nasa P, Chaudhary S, Shrivastava PK, Singh A. Euglycemic diabetic ketoacidosis: A missed diagnosis. World J Diabetes. 2021 May 15;12(5):514-523. doi: 10.4239/wjd.v12.i5.514. PMID: 33995841; PMCID: PMC8107974.
17. Mistry S, Eschler DC. Euglycemic Diabetic Ketoacidosis Caused by SGLT2 Inhibitors and a Ketogenic Diet: A Case Series and Review of Literature. AACE Clin Case Rep. 2020 Dec 28;7(1):17-19. doi: 10.1016/j.aace.2020.11.009. PMID: 33851013; PMCID: PMC7924151.
18. Fukuyama Y, Numata K, Yoshino K, Santanda T, Funakoshi H. Euglycemic diabetic ketoacidosis due to a strict low-carbohydrate diet during treatment with sodium-glucose cotransporter 2 inhibitors. Acute Med Surg. 2020 Jan 16;7(1):e480. doi: 10.1002/ams2.480. PMID: 31988792; PMCID: PMC6971428.
19. Blau JE, Tella SH, Taylor SI, Rother KI. Ketoacidosis associated with SGLT2 inhibitor treatment: Analysis of FAERS data. Diabetes Metab Res Rev. 2017 Nov;33(8):10.1002/dmrr.2924. doi: 10.1002/dmrr.2924. Epub 2017 Sep 29. PMID: 28736981; PMCID: PMC5950709.
20. Tarif N, Al Badr W. Euglycemic diabetic ketoacidosis in pregnancy. Saudi J Kidney Dis Transpl. 2007 Nov;18(4):590-3. PMID: 17951948.
21. Joseph F, Anderson L, Goenka N, Vora J. Starvation-induced true diabetic euglycemic ketoacidosis in severe depression. J Gen Intern Med. 2009 Jan;24(1):129-31. doi: 10.1007/s11606-008-0829-0. Epub 2008 Oct 31. PMID: 18975036; PMCID: PMC2607495.
22. Mehta AE, Zimmerman R. Classic diabetic ketoacidosis and the euglycemic variant: Something old, something new. Cleve Clin J Med. 2025 Jan 2;92(1):33-39. doi: 10.3949/ccjm.92a.24075. PMID: 39746727.
23. Musso G, Sircana A, Saba F, Cassader M, Gambino R. Assessing the risk of ketoacidosis due to sodium-glucose cotransporter (SGLT)-2 inhibitors in patients with type 1 diabetes: A meta-analysis and meta-regression. PLoS Med. 2020 Dec 29;17(12):e1003461. doi: 10.1371/journal.pmed.1003461. PMID: 33373368; PMCID: PMC7771708.
24. Ju HH. Euglycemic Diabetic Ketoacidosis: How Is It Different from Diabetic Ketoacidosis. Crit Care Nurs Clin North Am. 2025 Mar;37(1):157-165. doi: 10.1016/j.cnc.2024.08.004. Epub 2024 Sep 23. PMID: 39890347.
25. Garg SK, Peters AL, Buse JB, Danne T. Strategy for Mitigating DKA Risk in Patients with Type 1 Diabetes on Adjunctive Treatment with SGLT Inhibitors: A STICH Protocol. Diabetes Technol Ther. 2018 Sep;20(9):571-575. doi: 10.1089/dia.2018.0246. Epub 2018 Aug 21. PMID: 30129772.
26. Fasanmade OA, Odeniyi IA, Ogbera AO. Diabetic ketoacidosis: diagnosis and management. Afr J Med Med Sci. 2008 Jun;37(2):99-105. PMID: 18939392.
27. Goldenberg RM, Gilbert JD, Hramiak IM, Woo VC, Zinman B. Sodium-glucose co-transporter inhibitors, their role in type 1 diabetes treatment and a risk mitigation strategy for preventing diabetic ketoacidosis: The STOP DKA Protocol. Diabetes Obes Metab. 2019 Oct;21(10):2192-2202. doi: 10.1111/dom.13811. Epub 2019 Jun 30. PMID: 31183975.