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Original Research Article

HOSPITAL-RECORDED PROPORTION AND FACTORS ASSOCIATED WITH EUGLYCEMIC DIABETIC KETOACIDOSIS AMONG SGLT2 INHIBITOR USERS: A SINGLE-CENTRE RETROSPECTIVE STUDY

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ABSTRACT

Background: SGLT2 inhibitor exposure creates a diagnostic challenge when a patient becomes ketoacidotic without the substantial hyperglycemia usually associated with DKA. Failure to assess ketones and acid-base status may therefore delay recognition. The objective is to estimate the hospital-recorded proportion of euDKA among eligible adults receiving SGLT2 inhibitors at the study centre and describe clinical factors and inpatient outcomes. **Materials and Methods:** Clinical, prescribing and laboratory records from January 2024 to June 2026 were reviewed retrospectively at the study centre. The denominator comprised 236 eligible adults identified in hospital records without separate sampling strata for inpatient, emergency-department or outpatient care. euDKA required glucose <200 mg/dL in a person with diabetes, beta-hydroxybutyrate >=3.0 mmol/L or urine ketones >=2+, and venous pH <7.30 and/or bicarbonate <18 mmol/L. The patient-level proportion was estimated using a Wilson 95% confidence interval (CI); associations were assessed using unadjusted odds ratios (ORs). **Result:** Among 236 eligible adults, 18 met euDKA criteria (7.6%; 95% CI 4.9-11.7). Cases were older than non-cases (58.7 [SD 11.4] vs 54.9 [SD 12.3] years); insulin co-therapy was recorded in 66.7% versus 39.0%. Reduced intake or fasting (66.7%), dehydration (61.1%), and infection (55.6%) were common among cases. Crude ORs were 9.48 (95% CI 3.41-26.31) for dehydration and 7.48 (2.66-21.00) for reduced intake or fasting. Five cases (27.8%) required intensive care; one (5.6%) died in hospital. **Conclusion:** euDKA was confirmed in 7.6% of this selected hospital-record cohort; this estimate is not a population incidence rate. Ketone and acid-base testing should be considered in symptomatic SGLT2 inhibitor users even without marked hyperglycemia.

INTRODUCTION

SGLT2 inhibitors are prescribed for diabetes as well as heart failure and chronic kidney disease, bringing their potential adverse effects into several areas of clinical practice. Among type 2 diabetes patients at high cardiovascular risk, empagliflozin was shown to improve cardiovascular outcomes and survival.[1] Dapagliflozin was beneficial in patients whose heart failure was accompanied by reduced ejection fraction.[2] Dapagliflozin also reduced kidney disease progression in a trial involving patients with chronic kidney disease.[3] Although these drugs provide important benefits, clinicians should remain alert to complications during acute illness.

Ketoacidosis associated with this drug class does not always resemble the hyperglycemic presentation that clinicians expect. Initial clinical observations described episodes occurring with comparatively low glucose concentrations.[4] Subsequent comparative research found an increased risk after the start of SGLT2 inhibitor treatment.[5] A multicentre cohort study also reported an increased risk of diabetic ketoacidosis among SGLT2 inhibitor users.[6] Medication history is therefore important when evaluating patients with suspected ketoacidosis. Confirmation of euDKA requires ketone and acid-base measurements rather than glucose alone.

Glucose concentration may not reflect the severity of ketoacidosis. According to the 2024 consensus on hyperglycemic crises, DKA may be diagnosed from the required ketone and acid-base abnormalities, with glucose values under 200 mg/dL[7] In symptomatic patients receiving an SGLT2 inhibitor, ketone testing

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and blood-gas assessment should be considered even without marked hyperglycemia.

Preceding illness and changes in treatment can further complicate recognition. Clinical literature describes episodes involving poor oral intake, fasting, carbohydrate restriction, dehydration, infection, operative stress or altered insulin therapy.[8] These conditions are not mutually exclusive: a single illness may disrupt eating, fluid intake and medication use at the same time.

SGLT2 inhibition reduces renal glucose reabsorption and maintains urinary glucose loss despite relatively modest circulating glucose concentrations. Full-dose treatment can cause approximately 50-100 g/day of glucose excretion, with the amount varying according to glycemia and kidney function.[9] This glucose loss lowers the stimulus for pancreatic insulin secretion and promotes a relative increase in glucagon activity. Reduced insulin action releases adipose lipolysis, increasing the supply of free fatty acids to the liver. Lower acetyl-CoA carboxylase activity reduces malonyl-CoA inhibition of carnitine palmitoyltransferase I (CPT-I), facilitating mitochondrial fatty-acid beta-oxidation and ketone formation. Clinically significant ketosis and acidosis may therefore occur despite glucose concentrations below 200 mg/dL.[10] Safety surveillance outside clinical trials has also recorded ketoacidosis among patients receiving these agents.[11]

This retrospective review assessed the proportion of SGLT2 inhibitor users meeting the euDKA case definition at the study centre. Medication records, biochemical findings and clinical notes were also examined to describe the circumstances surrounding presentation and short-term hospital outcomes.

Aim: To estimate the hospital-recorded proportion of eligible SGLT2 inhibitor-treated adults at the study centre with an event fulfilling the euDKA case definition.

Primary objective: To determine the number of distinct patients with confirmed euDKA and express this as a proportion of the eligible cohort.

Secondary objectives: To compare the recorded characteristics of cases and non-cases, quantify exploratory unadjusted associations, and describe possible triggers, admission biochemistry and short-term outcomes among confirmed cases.

MATERIALS AND METHODS

Study design and setting: This single-centre retrospective review used prescribing, laboratory and clinical records at a tertiary-care centre in Kerala, India, from January 2024 to June 2026. The study population comprised 236 unique adults with documented SGLT2 inhibitor exposure and adequate records for retrospective assessment. It was assembled from available hospital records rather than defined as a consecutively admitted inpatient cohort. Inpatient, emergency-department and outpatient encounters were not tabulated as separate recruitment strata; the number of eligible patients contributed by each setting cannot be determined from these aggregate data. The denominator therefore represents this selected hospital-record cohort, not all SGLT2 inhibitor users attending the hospital or a population followed over person-time. Accordingly, the estimate is a hospital-recorded proportion, not an inpatient prevalence or a population incidence rate.

Eligibility and case ascertainment: Adults with documented diabetes and ongoing or recent SGLT2 inhibitor exposure were eligible. The extracted records did not reliably identify diabetes subtype; neither restriction to type 2 diabetes nor exclusion of known type 1 diabetes could be confirmed. Duplicate records, records without verified exposure and records with insufficient laboratory documentation for review were excluded. Twenty-nine presentations were investigated for suspected euDKA: 18 met the biochemical definition and 11 did not. The other 207 patients had no suspected episode documented and were not subjected to systematic ketone screening. Thus, the group without confirmed euDKA comprised 11 evaluated non-cases and 207 patients with no recorded suspected episode; it was not a universally biochemically screened group. Each patient was counted once, irrespective of repeated encounters.

Operational definition of euDKA: euDKA required three concurrent elements: plasma glucose <200 mg/dL in a person with documented diabetes; ketosis, defined by beta-hydroxybutyrate >=3.0 mmol/L or urine ketones >=2+; and metabolic acidosis, indicated by venous pH <7.30 and/or serum bicarbonate <18 mmol/L. Venous blood-gas pH was used for case classification and the descriptive biochemical results; arterial pH was not part of the reported case definition. The anion gap was recorded as a descriptive biochemical variable and was not used as a separate diagnostic criterion. Albumin-corrected anion gaps were not systematically calculated. Records with uncertain documentation or a plausible competing explanation underwent reassessment. The same criteria were applied to all suspected events. For the time-to-resolution variable, the endpoint was resolution as documented in the clinical chart; the duration in hours was abstracted as recorded for each confirmed episode. A common venous pH, bicarbonate, blood-ketone clearance, or insulin-transition threshold was not applied retrospectively.

Variables and clinical factors: Recorded baseline variables were age, sex, diabetes duration, SGLT2 inhibitor, HbA1c, eGFR and insulin co-therapy. Acute circumstances documented during the preceding 14 days were reduced oral intake or fasting, dehydration, infection or fever, vomiting, insulin omission or dose reduction, carbohydrate restriction, and surgery or another procedure. These were chart-derived categories: dehydration required a documented clinical impression of dehydration or volume depletion; reduced intake or fasting required a note of poor oral intake, fasting or nil-by-mouth status; and carbohydrate restriction required

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documentation of a low-carbohydrate dietary pattern. No BUN-to-creatinine ratio threshold, minimum fasting duration or numerical carbohydrate limit was applied retrospectively because these measures were not the basis of the abstracted categories. The circumstances could co-occur and were not independently established as causes. Insulin omission or reduction was also considered within the insulin-treated subgroup because it is applicable only to patients prescribed insulin.

Statistical analysis: Confirmed euDKA was expressed per eligible patient with a Wilson 95% CI. Categorical variables are reported as counts and percentages, and continuous variables as means (SD) or medians (IQR). Separate 2 x 2 comparisons provided crude ORs with Wald 95% CIs. Univariable odds ratios were evaluated for charted acute precipitants and pre-specified high-risk clinical markers (HbA1c >9% and eGFR <60 mL/min/1.73 m²), rather than all baseline demographics in [Table 1]. Effect sizes were reported with 95% confidence intervals rather than null-hypothesis p-values. For insulin omission or reduction, a subgroup analysis restricted the denominator to patients receiving concomitant insulin; the total-cohort comparison was retained for consistency with [Table 4 and Figure 2]. Multivariable adjustment was not undertaken given the 18 confirmed events. As acute circumstances can occur together in a single illness episode, these unadjusted estimates describe associations rather than independent or causal effects. Reporting followed observational-study principles.[12]

RESULTS

Screening began with 250 records. Six duplicate entries, four records without confirmed SGLT2 inhibitor use and four with inadequate laboratory information were excluded, leaving 236 patients for analysis [Figure 1]. Twenty-nine presentations prompted evaluation for euDKA; 18 met the laboratory definition and 11 did not. The other 207 patients had no suspected episode, giving 218 patients without confirmed euDKA. The patient-level proportion was 18/236 (7.6%; Wilson 95% CI 4.9-11.7). Cases had a mean age of 58.7 years (SD 11.4), compared with 54.9 years (SD 12.3) in non-cases. Among the 18 cases, eGFR was below 60 mL/min/1.73 m² in 8 (44.4%), HbA1c exceeded 9% in 12 (66.7%), and 12 (66.7%) were on concomitant insulin. Further baseline details appear in [Table 1].

Marked ketosis and acidosis were observed despite relatively low glucose concentrations. Glucose had a median of 168 mg/dL (IQR 148-181), whereas beta-hydroxybutyrate had a median of 5.4 mmol/L (IQR 4.1-6.8). Mean venous pH was 7.22 (SD 0.07), and mean serum bicarbonate was 12.8 mmol/L (SD 3.4). During admission, 5 of the 18 cases (27.8%) required intensive care and 1 patient (5.6%) died [Table 2]. A proximate cause of the in-hospital death could not be assigned from the clinical details summarised in this review.

Multiple possible precipitants were recorded in 12 of 18 cases (66.7%; [Table 3]). Reduced oral intake or fasting occurred in 12 (66.7%), dehydration in 11 (61.1%), infection or fever in 10 (55.6%), and vomiting in 9 (50.0%). Total-cohort unadjusted ORs were 9.48 (95% CI 3.41-26.31) for dehydration, 7.48 (2.66-21.00) for reduced intake or fasting, 6.47 (2.33-17.97) for insulin omission or reduction, and 5.09 (1.89-13.66) for acute infection or fever. In the clinically relevant subgroup receiving insulin, omission or reduction was documented in 8/12 cases (66.7%) and 24/85 non-cases (28.2%), giving an OR of 5.08 (95% CI 1.40-18.46). [Table 4 and Figure 2] display total-cohort comparisons, with the insulin-omission denominator identified explicitly; the insulin-treated comparison is presented separately in the text and table note. Vomiting (9/18) and alcohol exposure (2/18) are described among confirmed cases in [Table 3], but comparator counts were not tabulated for the 218 patients without confirmed euDKA; no ORs were therefore estimated for these two factors.

Table 1: Baseline characteristics of the cohort by euDKA status

Characteristic	Overall (n = 236)	euDKA (n = 18)	No euDKA (n = 218)
Age, years, mean (SD)	55.2 (12.2)	58.7 (11.4)	54.9 (12.3)
Female sex, n (%)	98 (41.5)	11 (61.1)	87 (39.9)
Diabetes duration, years, median (IQR)	7 (4-11)	8 (5-13)	7 (4-11)
HbA1c >9%, n (%)	86 (36.4)	12 (66.7)	74 (33.9)
Insulin co-therapy, n (%)	97 (41.1)	12 (66.7)	85 (39.0)
Reduced eGFR (<60 mL/min/1.73 m²), n (%)	57 (24.2)	8 (44.4)	49 (22.5)
Empagliflozin use, n (%)	124 (52.5)	10 (55.6)	114 (52.3)
Dapagliflozin use, n (%)	96 (40.7)	7 (38.9)	89 (40.8)
Other SGLT2 inhibitor use, n (%)	16 (6.8)	1 (5.6)	15 (6.9)

Values are presented as n (%), mean (SD) or median (IQR) where applicable. Abbreviations: HbA1c, glycated hemoglobin; eGFR, estimated glomerular filtration rate. The SGLT2 inhibitor categories do not overlap.

Table 2: Biochemical profile and short-term outcomes of confirmed euDKA cases

Measure	euDKA cases (n = 18)
Plasma glucose at presentation, mg/dL, median (IQR)	168 (148-181)
Beta-hydroxybutyrate, mmol/L, median (IQR)	5.4 (4.1-6.8)
Venous pH, mean (SD)	7.22 (0.07)
Serum bicarbonate, mmol/L, mean (SD)	12.8 (3.4)

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Serum anion gap, mmol/L, mean (SD)	24.6 (5.2)
Serum creatinine, mg/dL, median (IQR)	1.5 (1.1-2.0)
Intensive care admission, n (%)	5 (27.8)
Time to documented euDKA resolution, hours, median (IQR)	26 (18-34)
Hospital stay, days, median (IQR)	5 (4-7)
In-hospital mortality, n (%)	1 (5.6)

Continuous data are given as median (IQR) or mean (SD); categorical data are given as n (%). ICU: intensive care unit.

Table 3: Documented clinical precipitants in confirmed euDKA cases

Factor recorded among euDKA cases	n / 18 (%)
Reduced oral intake or fasting	12 (66.7)
Dehydration	11 (61.1)
Acute infection or fever	10 (55.6)
Vomiting	9 (50.0)
Insulin omission or reduction	8 (44.4)
Carbohydrate restriction	6 (33.3)
Recent surgery or procedure	5 (27.8)
Alcohol exposure	2 (11.1)
More than one precipitating factor	12 (66.7)

The denominator is 18 confirmed euDKA patients. A patient could have more than one precipitating circumstance; percentages are therefore not additive.

Table 4: Unadjusted associations of recorded clinical factors with euDKA status

Potential associated factor	euDKA, n/18 (%)	No euDKA, n/218 (%)	Crude OR (95% CI)
Acute precipitating circumstances (preceding 14 days)			
Dehydration	11/18 (61.1)	31/218 (14.2)	9.48 (3.41-26.31)
Reduced oral intake or fasting	12/18 (66.7)	46/218 (21.1)	7.48 (2.66-21.00)
Insulin omission or reduction (total cohort denominator)	8/18 (44.4)	24/218 (11.0)	6.47 (2.33-17.97)
Acute infection or fever	10/18 (55.6)	43/218 (19.7)	5.09 (1.89-13.66)
Carbohydrate restriction	6/18 (33.3)	22/218 (10.1)	4.45 (1.52-13.05)
Recent surgery or procedure	5/18 (27.8)	18/218 (8.3)	4.27 (1.37-13.34)
Baseline clinical characteristics			
HbA1c >9%	12/18 (66.7)	74/218 (33.9)	3.89 (1.40-10.79)
Reduced eGFR (<60 mL/min/1.73 m²)	8/18 (44.4)	49/218 (22.5)	2.76 (1.03-7.37)

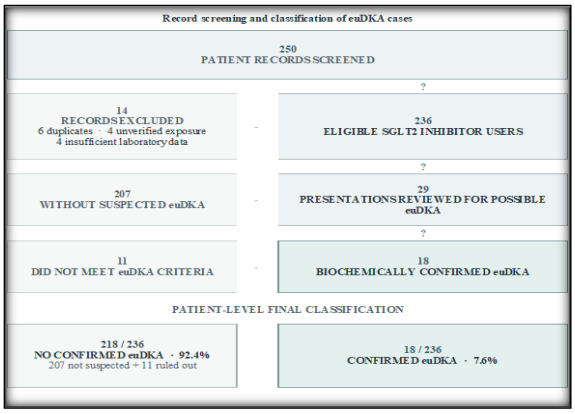


Figure 1: This diagram follows record screening through final euDKA classification. From 250 screened records, 14 were excluded and 236 patients were eligible. Among 29 presentations investigated for possible euDKA, 18 met the study definition and 11 were ruled out. With 207 additional patients not suspected of euDKA, the non-case group comprised 218 patients.

CI, confidence interval; OR, odds ratio. Univariable estimates were calculated from the indicated 2 x 2 counts. Insulin omission/reduction is a conditional exposure relevant to patients prescribed insulin. The insulin-omission row uses total-cohort denominators, also used in Figure 2; among insulin-treated patients only, the comparison was 8/12 versus 24/85 (OR 5.08; 95% CI 1.40-18.46). HbA1c and reduced eGFR are baseline characteristics; other factors are acute circumstances recorded during the preceding 14 days. Vomiting and alcohol exposure appear in Table 3 as case-only summaries because corresponding non-case counts were not tabulated for these comparisons. The No euDKA group indicates no recorded confirmed event, not a uniformly negative biochemical screen.

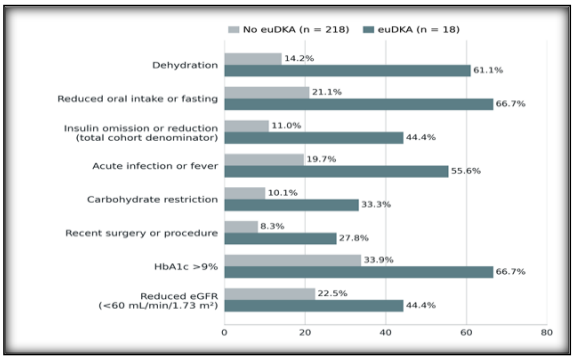


Figure 2: Bars display the percentage of patients with each documented factor in the confirmed euDKA (n = 18) and no-euDKA (n = 218) groups. The insulin omission or reduction bars use total-cohort denominators (18 and 218); conditional comparisons among insulin users are reported in Table 4. The underlying counts and crude associations are listed in Table 4.

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DISCUSSION

Eighteen of 236 eligible patients met the euDKA definition. As ketone testing reflected routine clinical practice rather than systematic screening across all SGLT2 inhibitor users, the 7.6% figure describes the hospital-recorded proportion in this selected cohort, not incidence in the broader treated population. Multiple clinical circumstances were documented in 12 cases. Dehydration, reduced intake, vomiting and changes in insulin use can arise together during acute illness; accordingly, the crude ORs should not be interpreted as independent or causal effects. The effect of reduced carbohydrate availability on ketogenesis provides a biological explanation for this pattern.[10]

Perioperative fasting and physiological stress may contribute to euDKA. Surgery or another procedure preceded five confirmed cases, with a crude OR of 4.27 (95% CI 1.37-13.34). This comparison is based on few exposed patients and cannot distinguish procedure-related circumstances from underlying differences between the groups. Published perioperative case reports identify a similar setting for SGLT2 inhibitor-associated ketoacidosis.[13]

In cases, median presenting glucose was 168 mg/dL despite substantial ketosis and acidosis. In an SGLT2 inhibitor recipient with vomiting, abdominal symptoms or rapid breathing, ketone and blood-gas assessment should not depend on the presence of marked hyperglycemia.[14] Management requires fluid replacement and insulin to suppress ketogenesis; because glucose may already be below 200 mg/dL, 5%-10% dextrose should accompany intravenous insulin from the outset, with close monitoring of glucose and potassium.[7] Requiring plasma beta-hydroxybutyrate <0.6 mmol/L together with both venous pH >=7.30 and serum bicarbonate >=18.0 mmol/L is a stricter measure of biochemical normalization, not the 2024 consensus definition. The consensus specifies ketone clearance with either venous pH >=7.30 or bicarbonate >=18 mmol/L; the anion gap is not recommended for determining resolution.[7] Five patients needed intensive care and one died in hospital. The observational design does not allow the intensive care admissions or in-hospital death to be attributed directly to SGLT2 inhibitor treatment.

During acute illness, patients should receive practical advice about fluid and carbohydrate intake, appropriate insulin dosing and temporary interruption of SGLT2 inhibitors under clinical guidance rather than stopping insulin independently.[15] A structured sick-day plan should cover fever, vomiting, dehydration, poor oral intake and prolonged fasting; it should specify when to hold an SGLT2 inhibitor, when to check blood ketones where feasible, and when symptoms warrant urgent medical evaluation.[15] Insulin-treated patients should not discontinue basal insulin solely because they are eating less and should seek individual advice on dose adjustment.[15] Before elective surgery, commonly recommended interruption periods are at least three days for empagliflozin, dapagliflozin and canagliflozin, and four days for ertugliflozin.[16] Glucosuria can persist for at least 3-5 days after discontinuation and sometimes considerably longer, so post-withdrawal symptoms still require assessment for ketoacidosis.[17] The median 26-hour chart-recorded resolution interval cannot establish a drug-specific delay or be interpreted as time to a uniform biochemical endpoint.

Study limitations: The retrospective design depended on the completeness of medication histories, laboratory measurements and clinician documentation. The hospital records were not divided into inpatient, emergency-department and outpatient strata, so the contribution from each care setting and the representativeness of the denominator cannot be established. No suspected euDKA event was recorded for 207 eligible patients, and routine ketone screening was not performed across the full cohort; unrecognised events are therefore possible. Only 18 events were confirmed, yielding wide CIs and limiting comparisons to unadjusted associations that may reflect overlapping illness-related triggers and residual confounding. Dehydration, fasting and carbohydrate restriction were classified from charted descriptions rather than uniformly applied numerical thresholds. Albumin-adjusted anion gaps were not systematically assessed, and routine C-peptide or islet-autoantibody testing was not included to exclude unrecognised autoimmune diabetes. Diabetes subtype was not consistently available, and the recorded time to resolution was based on clinical documentation rather than a standard biochemical endpoint. Vomiting and alcohol exposure could not be compared with non-cases because corresponding comparator counts were not tabulated.

CONCLUSION

Within the eligible study-centre cohort, 18 of 236 SGLT2 inhibitor users (7.6%) had biochemically confirmed euDKA. Coexisting illness-related circumstances and the discrepancy between glucose and ketone-acid-base findings were prominent features of the documented cases. Clinical assessment should therefore be driven by suggestive symptoms rather than glucose alone, while advice for acute illness and planned procedures remains important. The 7.6% estimate should not be extrapolated to all patients receiving SGLT2 inhibitors.

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