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CASE REPORT

"SGLT2 Inhibitor-Associated Diabetic Ketoacidosis in a Pediatric Patient: An Off- Label Use Case Report"

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Abstract: A 7-year-old boy presented with new-onset type one diabetes mellitus (T1DM) and moderate diabetic ketoacidosis (DKA). The patient presented with polyuria, polydipsia, and weight loss along with reduced activity and excessive sleepiness. Initial blood tests revealed elevated blood glucose levels, high anion-gap metabolic acidosis, and ketonuria. The patient was treated with fluid resuscitation, insulin infusion, and electrolyte replacement, which resolved within three days. During medication reconciliation, the patient was administered empagliflozin, a sodium-glucose co-transporter 2 (SGLT2) inhibitor, by a family member without medical supervision. This case highlights the challenges in managing pediatric diabetes, potential risks of unauthorized medication use, and need for comprehensive family education in diabetes care. This also raises questions regarding the safety and benefits of SGLT2 inhibitors in children under 10 years of age with type 1 diabetes mellitus, an area that requires further research.

Keywords: diabetic ketoacidosis, euglycemic diabetic ketoacidosis, pediatric diabetes, off-label drug use, type 1 diabetes mellitus, type 2 diabetes mellitus, adolescent diabetes care, adjunctive therapy, insulin therapy, sodium-glucose co-transporter 2 inhibitors

Introduction

Diabetes mellitus (DM) is a chronic metabolic condition that disturbs glucose metabolism in the blood.[1] Diabetes mellitus (DM) is classified into several types, which include type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM).[1] The prevalence of diabetes is increasing among pediatric patients worldwide and in the Kingdom of Saudi Arabia (KSA).[2] Its prevalence may differ according to the type of DM.[2] Mobasseri et al reported that the prevalence of type one diabetes mellitus is 95 cases per 100,000 individuals, with an incidence of 15 cases per 100,000 based on the documented global occurrence.[2] The incidence of type one DM has increased significantly in Saudi Arabia. Based on the last available data which present on Alhabishi study mentioned that ", the incidence rate in KSA is 29 cases per 100,000 individuals, whereas the prevalence rate is 109.5 per 100,000".[3] The International Diabetes Federation (IDF) reported that the annual incidence of type one DM in Saudi Arabian pediatric groups (age from 0 to 19 years) is 46,468.9 (44%) out of 10,525,192.0 in 2024.[4] The main etiologies of diabetes mellitus type 1 include autoimmune disease, while type 2 DM include insulin resistance, overweight (obesity with a large waist size), and sedentary lifestyle.[5] One of the acute complications of uncontrolled diabetes mellitus is a diabetic ketoacidosis (DKA) life-threatening condition caused by hyperglycemia, ketonemia, ketonuria, and metabolic acidosis.[5] The mortality rate is reported to be 5.2% after a single event of diabetic ketoacidosis, which increases 6-fold with five or more admissions due to diabetic ketoacidosis (DKA).[6] Multiple factors, including younger age, low socioeconomic status, and poor glycemic control, lead to the development of DKA and affect its severity.[5]

The management of both type 1 and type 2 (DM) extends beyond pharmacological treatment and requires comprehensive lifestyle modifications.[7] This includes regular physical activity, adherence to a balanced and nutritious diet, and the ability to monitor daily intake of carbohydrates, saturated and unsaturated fats, and proteins.[7] Achieving and maintaining a healthy weight also plays a critical role in reducing the risk of diabetes-related complications and addressing the associated

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psychological or stress-related concerns.[7] Insulin therapy remains the cornerstone of treatment for pediatric patients with type 1 DM and is approved by the US Food and Drug Administration (FDA).[8] Another pharmacological option for type I DM combined with obesity is metformin administered for a short duration and at small doses.[8] In recent years, there has been growing emphasis on the autoimmune pathogenesis of the disease, highlighting the importance of early detection and screening. The FDA has approved teplizumab, a humanized monoclonal anti-CD3 antibody, for use in delaying the onset of clinical type 1 DM in children aged ≥ 8 years.[9]

The Pharmacological agents approved by the FDA for the treatment of pediatric patients with T2DM include metformin, sodium glucose transporter 2 (SGLT2) inhibitors (not in all pediatric age groups), Sulfonylureas, PPAR-γ agonists, glucagon-like peptide (GLP)-1 receptor agonists, and dipeptidyl peptidase-4 (DPP-4) inhibitors.[7] The use of metformin in the pediatric population is an excellent choice for targeting both T2DM and obesity.[10] It was approved for the treatment of DM for those older than 10 years, while 6 years and above were approved for weight reduction.[10] In all cases, renal dose adjustment may be required, and the risk of lactic acidosis may increase.[10] Recently, a sodium glucose co-transporter (SGLT-2 inhibitor), a group of medications used to lower blood glucose by excreting glucose in the urine, was shown to allow diuresis, which helps in glycemic control and blood pressure.[11] SGLT-2 inhibits the reabsorption of sodium and glucose from proximal tubules, Its action is insulin-independent.[11] It has been used in the management of diabetes type 2 in adults, with cardio-and renal protection advantages and minimal risk of developing hypoglycemia.[11] The FDA approved its use in children aged 10 years and older as an adjective therapy for T2DM. There are clinical considerations regarding the initiation of SGLT-2 inhibitors, which involve the correction of any electrolyte or volume abnormalities before adding this agent in their treatment plan and a gradual decrease in the dose of insulin or other medications to avoid the risk of hypoglycemia.[10] The recommended dose in children aged 10 years and older is to be started with 10 mg orally once per day, and then gradually increase the dose to reach a maximum of 25 mg.[10] This may differ based on child tolerance and glucose controlling.[10] SGLT-2 inhibitors have not been approved by the FDA for renal or cardiovascular protection in children under 10 years of age, as current evidence is insufficient to support their safety and efficacy in this age group.[7,10]

This case study demonstrates both the advantages and risks associated with the administration of SGLT2 inhibitors in the treatment of pediatric Type 1 DM. While insulin remains the cornerstone of therapy for pediatric Type 1 DM, emerging treatments, such as SGLT-2 inhibitors, provide a supplementary option owing to their potential benefits. Although the patient tolerated empagliflozin without significant adverse effects, the subsequent development of diabetic ketoacidosis (DKA) highlights the potential risks associated with use of sodium-glucose co-transporter 2 (SGLT2) inhibitors in pediatric patients with type 1 diabetes mellitus (T1DM) underscore the importance of close monitoring and early detection strategies when administering these agents to young patient with Type 1 DM patients.

Case Presentation

A 7-year-old boy was brought to the emergency department (ED) by his family because of reduced activity, excessive sleepiness, and a two-week history of polydipsia, polyuria, and weight loss (the exact amount not specified). No associated abdominal pain, vomiting, diarrhea, or symptoms suggestive of infection (respiratory tract, skin, soft tissue, or urinary tract) were observed. The family initially visited a primary health clinic because of worsening symptoms, and a random blood glucose (RBG) level of 320 mg/dL was detected. The patient was referred to a specialized hospital without immediate treatment. Upon further symptom progression and persistently high blood glucose levels, the patient was transferred to the ED of a maternity facility and a children's hospital before his scheduled appointment.

Past Medical and Family History

The patient had ocular trauma at four years of age (post-car accident status and post-artificial eye transplant). The vaccinations were updated according to the patient's parents, and the patient did not receive any chronic medication. Family history: older brother's known case of Type one DM; no other autoimmune diseases in the family.

Examination Findings (EF)

1. General: Ill-appearing dehydrated but conscious, alert, and oriented (Glasgow Coma Scale (GCS) score: 15/15).

2. Vitals: Stable (HR 111 bpm, BP 129/66 mmHg, RR 17/min, SpO_2 98% on room air).
3. Anthropometrics: Weight 23.2 kg, height 120 cm.
4. Systemic Exam:
   - ENT/Chest/CVS: Normal; no added sounds, S1S2 +0, CRT <3 s.
   - Abdominal: soft, no hepatosplenomegaly.
   - CNS: Normal tone/reflexes; no signs of cerebral edema.

Initial Investigations

1. RBG: 394 mg/dL.
2. Venous Blood Gas (VBG): pH 7.18, pCO_2 29, HCO_3^- 11.4 (high-anion-gap metabolic acidosis, AG 19).
3. Urine dipstick: Glucose +3, Ketones +3.
4. Biochemistry:
   - High anion gap (19); otherwise, unremarkable electrolytes (Na^+ 132, K^+ 3.7, Cl^-) 102).
   - Normal renal function (BUN 4.2, Cr 66.3 µmol/L) and CBC (WBC 9.53, Hgb 14.6, PLT 286).

Impression and Management

New-onset Type I DM with moderate diabetic ketoacidosis (DKA), according to the Saudi Ministry of Health protocol. Laboratory investigations and management during the hospital stay are shown in Table 1.

Treatment

1. Fluid resuscitation with 0.9% NS was followed by transition to dextrose/half-normal saline when RBG dropped to 250 mg/dL.
2. Regular insulin infusion was initiated with electrolyte replacement and close monitoring (hourly glucose and 2-hourly venous blood gas/renal panels).
3. No sodium bicarbonate was administered.

Day 3: DKA resolved (closed anion gap, improving acidosis), tolerating oral intake and transitioning to a subcutaneous basal bolus insulin regimen.

Day 4: transfer to the pediatric ward. Diabetes education was provided to families. Medication Reconciliation: Parents disclosed unreported empagliflozin use (10 mg orally once per day for 3 days, then increased to 15 mg orally once per day for 3 days, then 25 mg orally once per day for 4 days, for a total of 10 days while he was on this medication). It was given by the family member to lower his blood glucose level before going to the hospital. The Toxicology Center

Table 1 Summary of Laboratory and Management During the PICU Stay

TimeLine e	RBS (mg/ dL)	BUN (mmol/L)	Cr (µmol/L)	Na (mmol/L)	K (mm ol/L)	Cl (mmol/L)	Ca (mmol/L)	Mg (mmol/L)	pH	HCO_3 (mmol/L)	Treatment
ER	394	4.2	66.3	132	3.7	102	NA	NA	7.18	11.4	NS + KCl (40), insulin
PICU - 2h	237.6	4.2	54.8	135	3	105	2.03	0.8	7.16	8.9	NS + KCl (40), insulin
PICU - 6h	133.2	3.8	34.8	137	3.1	107	1.93	0.7	7.21	14.4	D10NS + KCl (60), insulin
PICU - 24h	73.8	2.7	52	138	2.8	109	1.99	NA	NA	NA	D10NS + KCl (60), insulin
PICU - 43h	255.6	3.44	22.9	137	4.2	102	2.05	0.83	7.33	19.1	D10NS + KCl (60), insulin, KCl IV correction
Dischar ge from PICU to ward	266	3.4	22.9	137	4.2	102	NA	0.83	7.33	19.1	Insulin infusion stopped and shifted to insulin regimen (Basal and bolus)

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advised observation only (no pediatric patients under the age of 10 years with data dosing for SGLT2 inhibitors). On day 5, the patient was discharged with basal-bolus insulin (glargine 5 units at bedtime and glulisine 4 units with meals). Multidisciplinary teams participated in educating parents about the recognition of hypo/hyperglycemia, insulin administration/storage, and glucose monitoring. Social work involvement: No abuse concerns and reinforced parental education.

Follow-Up

- Endocrinology clinic (1-week post-discharge)
- BG within the target, no hypoglycemic episodes.
- Continued same insulin regimen; initiated continuous glucose monitoring (CGM)
- Referred to clinical nutrition for dietary counseling.
- Ophthalmology referral for evaluation of prior ocular trauma.

Discussion

Managing Type I DM in young children presents significant challenges owing to their dependence on caregivers for daily management, emotional support, and decision-making regarding long-term health outcomes.[12] Achieving optimal glycemic control in this population is particularly difficult and is influenced by behavioral variability, physiological development, and environmental factors.[12] A study conducted in Italy demonstrated a correlation between family socioeconomic status and glycemic control in children despite clinical guidelines emphasizing the importance of tight glycemic regulation to prevent long-term complications.[13] Recent updates from the International Society for Pediatric and Adolescent Diabetes (ISPAD) have introduced individualized glycemic targets based on access to advanced diabetes technologies. For children utilizing continuous glucose monitors (CGMs) and automated insulin delivery systems, the recommended HbA1c target has been lowered to 6.4% with the aim of reducing the risk of acute and chronic complications. Conversely, for those without access to such technologies, the target remains 7.5%.[14]

Sodium-glucose co-transporter 2 (SGLT2) inhibitors have shown efficacy in improving glycemic control and maintaining a favorable safety profile in approved age groups of pediatric patients with type 2 diabetes; however, their role in Type I DM remains unclear.[15] A systematic review assessing the use of SGLT2 inhibitors in combination with insulin in patients with Type I DM excluded pediatric patients. The review reported improved glycemic control, reduced insulin requirements, and weight loss but also noted an increased incidence of diabetic ketoacidosis (DKA) in the SGLT2 inhibitor group.[16] SGLT2 inhibitors offer several benefits, including improved glycemic control, weight loss, reduced glucose variability, and lower blood pressure, without increasing the risk of severe hypoglycemia.[17] One of the known side effects of SGLT2 inhibitors is euglycemic DKA, and studies have shown an increase in the development of DKA in adult patients with type one DM while using SGLT-2 as adjustment therapy; however, there is a lack of data in the pediatric population.[18] The STICH protocol, as proposed by Garg et al (2018), is a clinical safety framework designed to reduce the risk of diabetic ketoacidosis (DKA) in patients with type 1 Diabetes Mellitus (T1DM) receiving SGLT2 inhibitors as an adjunctive therapy.[15] The protocol outlines five key steps: first, screening patients at an increased risk for DKA, including those with a history of DKA, alcohol use, low-carbohydrate diets, or poor treatment adherence; second, educating patients on the signs and symptoms of DKA and the importance of regular ketone monitoring.[15] The third step is to ensure adequate insulin therapy, particularly by maintaining sufficient basal insulin levels.[15] The fourth step is to promote appropriate carbohydrate intake, especially during illness or caloric restriction, to minimize the risk of ketosis, and the fifth step is to encourage hydration to support renal clearance of ketones and prevent dehydration.[15] Although this protocol has shown promise in lowering the risk of DKA among adult patients, its use in pediatric populations remains uncertain. Differences in metabolic rates, insulin sensitivity, and adherence behaviors in children necessitate a more individualized and cautious approach when considering SGLT inhibitor therapy in younger individuals.[15]

Dapagliflozin is effective in a retrospective study on the management of proteinuria in chronic kidney disease (CKD).[19] The study demonstrated stabilization of the decline in GFR and a reduction in systolic blood pressure, with no adverse effects observed in the pediatric population.[19]

In the present case, the patient recovered from DKA without the need for sodium bicarbonate correction, which may be attributed to the pharmacological effects of empagliflozin administered 10 days prior to the DKA event. The patient exhibited normal renal and hepatic functions. Most children with severe DKA present with hypernatremia, while in this case, hyponatremia is a known side effect of empagliflozin.[20] While the brief 10-day unintentional exposure to empagliflozin restricts conclusions about its efficacy, the absence of adverse effects and the maintenance of stable renal function despite the patient's elevated risk for acute kidney injury suggest that short-term use may be well tolerated in pediatric cases. However, larger, longer-term studies are necessary to confirm the safety of this population.[20]

Pharmacokinetic data indicate that empagliflozin, an SGLT2 inhibitor, exhibits similar pharmacokinetics in children and adults.[20] The drug is primarily excreted through urine (54.4%) and feces (41.2%), with a half-life of approximately 12.4 hours, 86% protein binding, and a time to peak plasma concentration of 1.5 hours.[20] In patients with impaired renal or hepatic function, drug clearance decreases as the area under the curve (AUC) increases.[20] Scott E Schaeffer mentioned that the symptoms of overdose with SGLT-2 inhibitors include nausea, vomiting, dizziness, hypoglycemia, confusion, hypertension, tachycardia, and urinary incontinence and toxicity symptoms present with mental status changes, polyuria, or tachypnea for people whose ingestion of 6000 mg of canagliflozin and 150 mg of ertugliflozin.[21] The toxic dose of canagliflozin was 20 times the maximum dose, while ertugliflozin was 10 times the maximum dose.[21] There is no clear data about the toxic dose of empagliflozin in human studies, while in animal studies, it is mentioned as 700 mg/kg/day (72–154 times the maximum clinical dose).[22] However, in this case, the child did not reach this amount of empagliflozin to be considered toxic or overdose. However, the absence of signs and symptoms of empagliflozin toxicity may be related to its good tolerability. The patient presented with signs and symptoms of DKA as a new diagnosis of T1DM.

In conclusion, the expanding use of SGLT2 inhibitors in adult type two DM management and their emerging application in pediatric populations for renal protection underscore the evolving landscape of diabetes care.[23] Although these developments are promising, the potential risk of euglycemic DKA necessitates close monitoring and early detection. Further comprehensive research is essential to establish the safety and efficacy of SGLT2 inhibitors for managing type one DM, particularly in younger pediatric patients.

Conclusion

This case report highlights the challenge of managing type 1 Diabetes Mellitus (T1DM) in young pediatric patients, where achieving optimal glycemic control is complicated by dependence on caregivers or family members, and the influence of socioeconomic factors. The development of diabetic ketoacidosis (DKA) following off-label empagliflozin use demonstrates the potential risks of SGLT2 inhibitors in children under 10 years of age, a population for whom these agents lack regulatory approval. Notably, the patient recovered without requiring sodium bicarbonate and maintained normal renal and hepatic functions. Although this single case does not permit conclusions regarding causality or generalizable safety, it highlights the need for further research to evaluate long-term outcomes, develop diabetic ketoacidosis (DKA) risk-mitigation strategies, and establish evidence-based protocols for the safe use, optimal dosing, and monitoring of SGLT2 inhibitors as adjunctive therapy in this specific pediatric age group.

Abbreviations

T1DM, Type One Diabetes Mellitus; T2DM, Type Two Diabetes Mellitus; SGLT2, Sodium-Glucose Cotransporter 2 (SGLT2) Inhibitor; DKA, Diabetic Ketoacidosis; DM, Diabetes Mellitus; KSA, Kingdom of Saudi Arabia; IDF, International Diabetes Federation; FDA, Food and Drug Administration; CD3, cluster of differentiation 3; PPAR-γ agonists, Peroxisome Proliferator-Activated Receptor Gamma Agonists; GLP-1, Glucagon-Like Peptide; DPP-4, Dipeptidyl Peptidase-4; ED, Emergency Department; RBG, Random Blood Glucose; EF, Examination Findings; GCS, Glasgow Coma Scale; BP, Blood pressure; HR, Heart Rate; RR, Respiratory Rate; SpO_2, Saturation of Oxygen; ENT, Ear, Nose, Throat; CVS, Cardiovascular system; S1S2, First heart sound, Second heart sound; CRT, Continuous renal replacements therapy; CNS, Central nervous system; VBG, Venous blood gas; pH, Potential of hydrogen; pCO2, Partial pressure of carbon dioxide; HCO3, Bicarbonate; AG, Anion gap; BG, Blood glucose; Na, Sodium; K, Potassium; Cl, Chloride; BUN, Blood Urea Nitrogen; Cr, Serum Creatinine; CBC, Complete Blood Count; WBC, White blood cell count; Hgb, Hemoglobin level; PLT, Platelet count; 0.9%NS, 0.9% Normal Saline; CGM, Continuous Glucose

Monitoring; HbA1c, Glycosylated Hemoglobin; ISPAD, International Society for Pediatric and Adolescent Diabetes; STICH, Screen, Teach, Insulin, Carbohydrate, Hydration; CKD, Chronic Kidney Disease; eGFR, Estimated Glomerular Filtration Rate; AUC, Aurea Under the Curve.

Data Sharing Statement

The data supporting the findings of this study are available from the corresponding author upon request.

Patient Consent

Written informed consent was obtained from the patient's guardian for inclusion in the case report and publication. The Ministry of Health does not require institutional approval for the publication of case reports.

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During the preparation of this work, we used Paparpal, an artificial intelligence tool, in the writing process to check the grammar. After using these paperpal services, we reviewed and edited the content as needed and took full responsibility for the content of the published article.

Author Contributions

All authors contributed significantly to this case report, including conception, data acquisition, and interpretation. All authors were involved in drafting, revising, and critically reviewing the manuscript. All authors approved the final version to be published, agreed to the journal of submission, and took full responsibility for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this work.

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