Case Report

Nursing Care for Patients with Euglycemic Diabetic Ketoacidosis After Cardiac Surgery: A Case Report

Xiaorun Xiang , Sha Zhang , Yao Deng , Ya Wang , Maoting Ye , Wei Sun *

Posted Date: 14 February 2025

doi: 10.20944/preprints202502.1069.v1

Keywords: euglycemic diabetic ketoacidosis; cardiac surgery; postoperative; SGLT2i

Case Report

Nursing Care for Patients with Euglycemic Diabetic Ketoacidosis after Cardiac Surgery: A Case Report

Xiaorun Xiang [1], Sha Zhang [1] Yao Deng [1], Ya Wang [2], Mating Ye [1] and Wei Sun [1,*]

[1] The First Affiliated Hospital of Army Medical University, China

[2] 958 Hospital of Army PLA, China

* Correspondence: sunfamily@tmmu.edu.cn

Abstract: Objective euDKA is a fatal complication of diabetes, but its symptoms overlap with common symptoms after cardiac surgery and are not easily detected. euDKA associated with SGLT2i was reported, highlighting the importance of recognizing novel hypoglycemic agents for this condition in different populations. **Methods** We describe the management of a 58-year-old diabetic man who developed severe nonhyperglycemic ketoacidosis after cardiac surgery. A detailed clinical care assessment and management was performed, including dietary history, physical examination, laboratory evaluation of blood ketone bodies, and results of blood gas analysis for metabolic analysis. **Results** The results showed that the blood ketone body was elevated, accompanied by severe metabolic acidosis and clinical symptoms of polydipsia, polyphagia and polyuria. After massive fluid replacement, small dose insulin treatment, and adjustment of diet structure, the patient's clinical symptoms disappeared, and the blood ketone body laboratory results returned to normal. **Conclusions** This case highlights the association between non-hyperglycemic diabetic ketoacidosis and sodium-glucose cotransporter 2 inhibitors, and emphasizes the importance of thorough medication history and blood test results for accurate diagnosis. As de novo diabetes agents become more popular worldwide, increased awareness of nonhyperglycemic diabetic ketoacidosis is critical to ensure prompt diagnosis and effective management.

Keywords: euglycemic diabetic ketoacidosis; cardiac surgery; postoperative; SGLT2i

1. Introduction

Sodium–glucose cotransporter type 2 inhibitors (SGLT2is) are a new class of hypoglycaemic drugs that are increasingly being used to treat type 2 diabetes; these drugs have very high selectivity, good effects, and are generally well tolerated[1]. The SGLT2is predominantly used in the clinic are gliflozin analogues such as dapagliflozin and empagliflozin. In addition to their hypoglycaemic effect, cardiovascular and renal benefits of SGLT2is have also been demonstrated in diabetic patients with high cardiovascular risk. Euglycemic diabetic ketoacidosis (euDKA) is a fatal complication of type 2 diabetes treated with SGLT2is, and its onset is insidious, making it difficult to diagnose. A retrospective study[2] revealed that in perioperative heart disease patients receiving an SGLT2i for the treatment of diabetes, the incidence of euDKA was approximately 15%, and the mortality in the SGLT2i group was approximately 1.5%. Currently, there is no unified conclusion on the morbidity and mortality of euDKA, but with the increasing use of SGLT2is, an increase in the number of reports of SGLT2i-related euDKA is expected in the future[3].

euDKA refers to normal or slightly elevated blood glucose (<13.9 mmol/L) combined with metabolic acidosis (serum bicarbonate <18 mmol/L and pH < 7.3) and ketosis (>0.6 mmol/L)[4, 5]. Currently, there are few case reports of euDKA after cardiac surgery caused by SGLT2i treatment. It is difficult to identify euDKA in patients after cardiac surgery because the signs and symptoms of euDKA overlap with those of other common conditions, and clinicians providing care for these patients lack the clinical understanding and management experience for this special condition.

 doi:10.20944/preprints202502.1069.v1

During the postoperative monitoring of the patient's condition, the focus of early care evaluation should be on the gastrointestinal symptoms of the patient, the chief patient complaints, the close monitoring of the changes in electrolytes, evaluation to determine whether metabolic acidosis is present, and the timely detection of ketones in the blood or urine. During treatment, on the one hand, a large amount of fluid must be replaced to reduce the concentration of ketone bodies and blood glucose; on the other hand, attention must be paid to the maintenance of blood glucose and serum potassium levels during the fluid replacement process, as well as the avoidance of acute right ventricular failure caused by an excessive volume load, which has brought great challenges to clinical nursing. In 2024, a total of 2 patients with euDKA after cardiac surgery caused by SGLT2i treatment for diabetes were admitted to our department. After effective symptomatic treatment and care, the patients recovered and were discharged from the hospital. Here, we report the case of one of these patients.

2. Case Details

A 58-year-old male with a 23-year history of type 2 diabetes was admitted to the hospital due to “syncope after strenuous exercise”, and the diagnosis was “coronary atherosclerotic heart disease”. Before surgery, the patient was given 25 mg of oral empagliflozin (tablet) once per day. The drug was discontinued 2 days before surgery, and the preoperative fasting time was 10 hours. The patient underwent coronary artery bypass grafting (anterior descending artery, circumflex branch, and right coronary artery) with extracorporeal circulation (cardiopulmonary bypass (CPB)) for 113 minutes. The operation lasted 5.5 hours. The patient was transferred to the cardiac surgery intensive care unit (ICU) after surgery, and the tracheal tube was removed after 26 hours. After surgery, the patient was fasted for 40 hours. Empagliflozin (25 mg) was taken orally once/day from the 4th to 5th days after surgery. On the 4th day after surgery, the patient developed wheezing and shortness of breath, polydipsia, polyuria, a fast heart rate, high anion gap metabolic acidosis combined with high partial pressure of CO_2 (PCO_2) respiratory alkalosis, and blood glucose <13.9 mmol/L as determined by blood gas analysis. After being diagnosed with euDKA, the patient received a continuous intravenous infusion of 10% glucose and insulin, and crystalloids were used for fluid resuscitation. After 2 days of treatment, the patient’s clinical symptoms completely resolved. The patient was transferred to the external care unit on the 6th day after surgery, and the patient was discharged from the hospital on the 20th day after surgery.

3. Treatment and Nursing Care

3.1. Cessation of SGLT2i Treatment and Changing of the Hypoglycaemic Agent

Elimination of predisposing factors is an important part of the treatment of euDKA. When euDKA is suspected, treatment with an SGLT2i, including canagliflozin, empagliflozin, and dapagliflozin, should be discontinued immediately[6]; however, treatment with these drugs can be restarted after euDKA symptoms resolve[7]. The nursing care measures taken for this patient and recommended measures were as follows: (1) SGLT2i treatment was stopped. (2) The oral hypoglycaemic agent was changed to subcutaneous insulin injection (regimen: insulin aspart 30 injection, 4 U before breakfast; insulin aspart 30 injection, before lunch; insulin aspart 30 injection, 6 U before dinner; and insulin glargine injection, 14 U once per night). (3) Each time the insulin pen was used, the pen was checked to ensure there was enough insulin in the pen for a full dose and whether the agent had deteriorated. (4) According to the type and duration of action of subcutaneous insulin, the patient was monitored for adverse reactions, such as hypoglycaemia, allergies, subcutaneous lipoatrophy or hyperplasia at the injection site, oedema, and blurred vision. Insulin aspart is a fast-acting insulin analogue that takes effect 10–15 minutes after injection. Insulin aspart reaches a peak at 1~2 hours and lasts for 4~6 hours. Insulin glargine is a long-acting insulin analogue that takes effect 2~3 days after injection, has no peak and lasts for 30 hours. Insulin lispro is a short-acting insulin analogue that takes effect in 10~15 minutes, reaching a peak at 1~1.5 hours and lasting

 doi:10.20944/preprints202502.1069.v1

for 4~5 hours. (5) Unopened insulin should be refrigerated at 2~8 °C, and insulin in use at room temperature (25~30 °C) is stable for 42 days. Violent shaking, extreme cold temperatures and overheating should be avoided during use. (6) During long-term subcutaneous insulin injection, attention should be paid to the rotation and selection of injection sites, and the deltoid, gluteus maximus, and anterior thigh muscles; abdomen; and loose skin of the upper arm should be chosen for injection in turn. (7) Injection should be performed at the same time every day, with "major rotation" of the injection sites; injection at the same site also requires minor rotation. This patient started to take empagliflozin orally on the 4th day after surgery. When elevated ketone bodies were observed 5 days after surgery, treatment with empagliflozin was immediately stopped, and empagliflozin was replaced by insulin subcutaneous injection. The patient's blood glucose level subsequently stabilized in the cardiac surgery ICU, and there was no recurrence of euDKA or DKA. On postoperative day 6, the patient was transferred to the cardiac surgery ICU. Blood glucose control via the original regimen was not satisfactory. On postoperative day 8, a subcutaneous injection of insulin was given to increase the concentration. On postoperative day 12, the pancreatic islets were targeted by changing to insulin lispro subcutaneous injection, and the dose was increased again. However, the effect was still not satisfactory. On the 13th day after surgery, the patient was treated with intensive insulin pump therapy after multidisciplinary consultation, and his blood glucose level stabilized. No adverse reactions or skin lesions at the injection sites occurred in the patient during subcutaneous insulin injection treatment.

3.2. Implementation of a Fluid Replacement Strategy with the Protection of Right Ventricular Function as the Core

Right ventricular function impairment is common after cardiac surgery, and volume assessment and adjustment are important in the prevention and treatment of cardiac dysfunction[8]. The treatments of euDKA and DKA are similar, with fluid replacement being the primary and main rescue measure. The use of balanced crystalloid fluids (such as PlasmaLyte or Ringer's lactic acid) for fluid replacement is recommended because normal saline may lead to hyperchloremia and anion gap metabolic acidosis. Compared with normal saline, balanced fluids can shorten the time to remission in DKA patients[9]. The initial recommended treatment is resuscitation with 1–2 L of crystalloid solution in the first 1–2 hours and 4000–600 ml of fluid replacement over 24 hours[10]. However, fluid intake needs to be restricted after cardiac surgery. How to reduce blood glucose and ketone levels during fluid replacement without impairing right ventricular function has been a challenge in nursing care. Because the patient developed euDKA while in the cardiac surgery ICU, the urinary catheter, invasive arterial catheter, and deep venous catheter were still in place, which is favourable for euDKA treatment. The nursing care measures implemented were as follows: (1) A nurse-led fluid management team was formed, including a doctor who was responsible for evaluating the laboratory results and cardiac ultrasound findings, volume loading, and adjusting medications. In addition, two nurses were responsible for the qualitative fluid management strategy, observing the effects of fluid replacement and reporting these effects in a timely manner. Moreover, one nursing team leader was responsible for overall supervision, the handling of abnormal results, volume assessment, and ensuring the accuracy of fluid replacement solution properties. (2) Isotonic fluid and 10% glucose injection were chosen for the intravenous fluid to avoid hypoglycaemia during fluid replacement. (3) The value and waveform of invasive arterial blood pressure and central venous pressure were dynamically monitored using a pressure detection kit. Central venous pressure was measured hourly. The systolic blood pressure was maintained at >100 mmHg, and the central venous pressure was maintained at 6~10 mmHg. (4) The electronic record sheet was used to record the amount of fluid intake and urine output every hour, and the changes in urine volume were closely monitored. (5) An infusion pump was used assist the nurse in precisely adjusting the infusion rate and volume. (6) The patient was helped into the supine or semi-recumbent position for cardiac ultrasound assessment. The hospital gown was removed to expose the anterior region of the heart. After the examination, the skin was cleaned to remove the conductive paste. (7) The patient was also assisted into the supine

 doi:10.20944/preprints202502.1069.v1

position to undergo bedside computed tomography (CT) examination, and the CT scans were immediately photographed and sent to the doctor for timely assessment of pulmonary oedema. (8) At each nurse shift change, nurses evaluated the patient for oedema, especially oedema of the lower extremities, hands, and face. (9) The lungs were auscultated to determine whether there were wet and dry rales in the lungs and whether the breath sounds were turbid. Sputum colour, volume, and nature were observed. The patient's fluid intake and urine output after 10 hours of treatment were -630 ml, the systolic arterial pressure was 131 mmHg, the central venous pressure was 8 mmHg, and the urine output was 30 ml. In this case, the fluid intake and urine output of this patient during the 1st and 2nd days of treatment were >1,000 ml, and during this time, no adverse reactions caused by volume imbalance occurred.

3.3. Administration of Low-Dose Insulin Therapy

For euDKA patients with a serum potassium level greater than 3.5 mmol/L, it is recommend to start insulin therapy immediately at a rate of 0.05–0.1 units/kg/hour to inhibit lipolysis and ketone body production[11]. However, the blood glucose level of this patient with euDKA was relatively low, and fluid replacement + low-dose insulin therapy could further reduce the blood glucose level. Furthermore, it is recommended to maintain blood glucose levels at 8.88–9.99 mmol/L after cardiac surgery[8]. Therefore, combined intravenous glucose injection was needed to avoid hypoglycaemia. Simultaneous intravenous infusions of 5% glucose and insulin are used routinely. If hypoglycaemia still occurs after the 5% glucose infusion, the use of 10% glucose is recommended[12-14]. If insulin infusion does not resolve ketosis, the volume of insulin infusion[9, 15] should be increased. The nursing care measures implemented for this patient were as follows: (1) Short-acting insulin was continuously intravenously infused along with 5% glucose injection (1 U of insulin was added for every 2~4 g of glucose). (2) The blood glucose levels were remeasured every 1~2 hours in the early stage and every 4~6 hours in the later stage. The insulin dose was adjusted according to the blood glucose levels. When the blood glucose level was <10 mmol/L, the treatment was changed to insulin plus 10% glucose injection. (3) The blood ketone levels were reevaluated every 2 hours. After the ketone level returned to normal, the insulin regimen was adjusted, according to the patient's condition, to a subcutaneous injection of short-acting insulin. In this patient, when euDKA was diagnosed on the 5th day after surgery, his blood glucose level was 12.5 mmol/L, and his blood ketone level was 6.3 mmol/L. Considering that the body was in a high-metabolic state after cardiac surgery, a 12-U injection of human insulin plus 500 ml of 10% glucose was used for treatment. During the treatment, the blood glucose level fluctuated at 7–13 mmol/L, and the blood ketone level continued to decrease. The patient's blood ketone levels returned to normal on the 8th day after surgery. No adverse reactions related to insulin treatment occurred during this period.

3.4. Correction of Electrolyte and Acid–Base Disorders

Because diuresis promotes the excretion of potassium ions, euDKA is often accompanied by hypokalaemia. For patients with a serum potassium level of 3.5–5.5 mmol/L, it is recommended to provide a potassium supplement at a dose of 10 mmol/L. If the serum potassium level is >5.5 mmol/L, the supplement may not be needed temporarily, but if the serum potassium level is <3.5 mmol/L, the potassium supplement should be continued until the serum potassium level is > 3.5 mmol/L before the start of insulin infusion[9, 16, 17]. The infusion of sodium bicarbonate is usually unnecessary because metabolic acidosis can be corrected after adequate fluid replacement + insulin therapy, but whether to provide a sodium bicarbonate supplement for patients with severe acidosis (pH <6.9) is controversial[9, 18, 19]. Combination therapy with glucose and insulin promotes the entry of potassium ions from the blood into the cells, thereby reducing the blood potassium level. Hypokalaemia can cause arrhythmias[20]; therefore, strict maintenance of serum potassium levels is required after cardiac surgery. During treatment, the serum potassium level, pH, residual base in whole blood, anion gap, and PCO_2 should be closely monitored. The recommended nursing measures and the measures implemented for this patient were as follows: (1) Potassium supplementation

 doi:10.20944/preprints202502.1069.v1

should be performed immediately in patients with severe hypokalaemia before treatment, and insulin therapy should be started when the serum potassium level is >3.5 mmol/L. (2) After the start of treatment, when the urine output is greater than 40 ml/hour and the serum potassium level is lower than 5.2 mmol/L, potassium supplementation can be started to maintain a serum potassium level >4.0 mmol/L. (3) Blood gas analysis was conducted every 2 hours to determine the serum potassium level, and the rate and amount of potassium supplement infusion were adjusted based on the electrocardiogram (ECG) findings and urine output. (4) The pH, anion gap, residual base in whole blood, and serum bicarbonate levels were reevaluated every 2 hours to determine the degree of metabolic acidosis. Whether to provide a base supplement was determined on the basis of on the results of these evaluations. (5) When intravenous infusion of sodium bicarbonate solution is used for the treatment of severe acidosis, the infusion rate should not be too fast to avoid cerebral oedema. When this patient was diagnosed with euDKA, his blood potassium level was >4.0 mmol/L. His blood potassium level decreased to 3.3 mmol/L after 4 hours of treatment. He was immediately given a 10% potassium chloride injection (20 ml) via intravenous infusion pump at a bolus speed of 8 ml/hour for 4 hours. The serum potassium level then increased to 4.3 mmol/L and was maintained within the ideal range. At the time of euDKA diagnosis, the blood gas analysis results showed a pH of 7.49, a PCO_2 of 23 mmHg, an anion gap of 28 mmol/L, and a residual base in whole blood of -8.3 mmol/L. The patient had severe metabolic acidosis combined with respiratory alkalosis, and the patient did not receive base replacement. The metabolic acidosis was relieved after 4 hours of fluid replacement plus insulin treatment, and the patient did not develop metabolic acidosis again.

3.5. Monitoring for euDKA-Specific Symptoms

The presence of euDKA should be considered when diabetes patients have the following clinical symptoms: nausea, vomiting, diffuse abdominal pain, polyuria, polydipsia, weight loss, dehydration, weakness, fatigue, tachycardia, shortness of breath and Kussmaul breathing, and changes in mental status[21, 22]. Studies[23, 24] have shown that euDKA patients often complain of nausea and vomiting, and patients also experience gastrointestinal reactions after cardiac surgery[25]. Therefore, close monitoring of patients with euDKA-related symptoms during treatment and the differentiation of these symptoms from common symptoms after cardiac surgery are one of the focuses of nursing evaluation. The recommended nursing care measures are as follows: (1) Be proactive about asking the patient if he/she has any discomfort, such as fatigue and shortness of breath, to detect the abnormalities in a timely manner. (2) Pay attention to gastrointestinal symptoms such as nausea, vomiting, and abdominal pain. (3) Evaluate patients for symptoms such as polydipsia, polyphagia, polyuria, and weight loss. (4) Evaluate the patient's state of consciousness after each nursing shift, and report any abnormalities in a timely manner. This patient complained of nausea and shortness of breath 4 days after surgery. Gastrointestinal reactions are common after cardiac surgery; however, we were not sufficiently vigilant. Four days after surgery, polydipsia and polyuria occurred; the 24-hour urine output was 5670 ml, the drinking volume was 2100 ml, and the fluid intake and urine output were -1264 ml. However, due to the relatively low blood glucose level, the ketoacidosis was masked, and the patient was worried about diabetes insipidus; therefore, a routine urine test was performed. The routine urinalysis results showed urinary ketone levels of 3+, and the diagnosis was euDKA. Combination therapy with fluid replacement and insulin was started immediately. After treatment, the patient's urine output returned to normal levels, and there was no recurrence of gastrointestinal reactions.

3.6. Prevention of Causes and Complications

The main inducers of euDKA include surgical stress, shock, infection, alcohol abuse, chronic liver disease, hunger, improper diet, and pregnancy[3, 26, 27]. If not treated in a timely manner, euDKA can lead to hypovolemic shock, renal failure, respiratory failure, acute gastric dilatation, cerebral oedema, coma, or even death[28, 29]. The nursing care measures implemented for this patient were as follows: (1) A piperacillin sodium and tazobactam sodium 4.5 g plus 100 ml normal

 doi:10.20944/preprints202502.1069.v1

saline intravenous drip was administered once every 8 hours to prevent infection. (2) An omeprazole sodium 40 mg push injection was administered intravenously once per day, and a pantoprazole sodium 40 mg enteric-coated tablet was orally administered once per day to protect gastrointestinal function. (3) A glutathione injection (1.8 g plus 100 ml of normal saline) was administered via intravenous drip once per day to protect liver function. (4) The concept of asepsis was followed in nursing operations through strict hand hygiene, with handwashing before and after patient contact. (5) The restrictive diet was adjusted after cardiac surgery. Total daily intake was not limited, and the patient could choose low-glycaemic index (GI) foods and eat less but more often (5–6 meals a day). (6) When eating, the head of the bed was adjusted to an angle of >45 degrees to avoid accidental aspiration, and the patient was not allowed to lie down on their back until 2 hours after a meal. (7) The patient was monitored for manifestations of liver function damage, such as anorexia, yellow staining of the skin and sclera, and changes in urine colours. (8) The patient was monitored for gastrointestinal symptoms such as nausea and vomiting. (9) The patient's state of consciousness was assessed after each nursing shift. The patient in this study experienced anorexia before surgery and fasted with water only early after surgery. After the patient resumed their diet, due to the strict volume requirements after cardiac surgery, the daily food intake was restricted, and the liquid diet volume was approximately 300 ml per meal. He had nutritional disorders and was diagnosed with euDKA. After that, the patient's diet was not restricted, but the amount of each meal was adjusted, and the number of meals was increased. During this period, the patient experienced no adverse reactions due to excess volume load. The treatment options included anti-infection treatment, liver and gastrointestinal function protective treatment, myocardial polarization, and subcutaneous injection of insulin. No complication of euDKA occurred in this patient during hospitalization.

4. Discussion

There is no systematic investigation of SGLT2i-related DKA. A meta-analysis published in 2020 showed that in T2D patients, SGLT2i can increase the risk of DKA by 2.13 times compared with placebo or other hypoglycemic drugs other than insulin [30]. Among patients with type 1 diabetes enrolled in SGLT2i clinical trials, up to 9.4% developed ketosis and up to 6% developed DKA[31]. According to the EMA announcement, as of May 2015, a total of 101 T2D patients with DKA treated with SGLT2i have been recorded in the EudraVigilance system worldwide, with an estimated exposure of more than half a million patients/year. However, the proportion of DKA caused by SGLT2i seems to be significantly higher than that observed in clinical trials, which may be related to the lack of understanding of the risk factors and early clinical manifestations of DKA during the use of this drug.

The causes of euDKA in diabetic patients include hunger, low-calorie diet, alcoholism, pregnancy, chronic liver disease, insulin reduction and SGLT2i use [29]. SGLT2i users may develop DKA when they have the following risk factors [32], including: T1D or latent autoimmune diabetes in patients with T1D or late-onset autoimmune diabetes adults,LADA), previous history of DKA, excessive alcohol or illicit drug use, pregnancy, low-carbohydrate or ketogenic diets, excessive SGLT2i dosing, too rapid insulin dose reduction, and insulin infusion failure. The development of DKA may be accelerated in these patients when the following predisposing factors [32]occur: vomiting, hypovolemia or dehydration, acute infection, acute phase of illness, surgery, strenuous or prolonged exercise, insulin pump failure or insulin infusion failure, or sudden interruption of insulin therapy. In the case reported in this article, the patient had fasting after surgery, postoperative stress, and diet was not fully recovered, and SGLT2i drugs were added to reduce glucose, resulting in euDKA.

The clinical manifestations of DKA include: thirst, polydipsia, polyuria, dehydration, nausea, vomiting, diarrhea, loss of appetite, abdominal pain, deep and rapid breathing, irritability, slow reaction and lethargy[4]. The clinical manifestations of most patients with DKA caused by SGLT2i are not typical, only mild dizziness, nausea, vomiting, abdominal pain or fatigue, and some patients even feel slightly uncomfortable with normal or slightly increased blood glucose levels[10]. It has

 doi:10.20944/preprints202502.1069.v1

been reported that about 30% of DKA patients caused by SGLT2i are hyperglycemic DKA and 70% are euglycemic DKA[33]. Due to the atypical clinical manifestations of DKA with normal blood glucose, clinicians need to be more sensitive to identify and diagnose. Therefore, when patients are in the acute phase of the disease, undergoing surgery, having reduced food and fluid intake, dehydration, alcoholism, and sudden reduction in insulin dosage, and when they have unexplained acidosis but normal blood glucose, it is necessary to be alert to the possibility of euDKA caused by SGLT2i.

When DKA occurs during the use of SGLT2i, treatment measures include discontinuation of SGLT2i, rehydration, administration of insulin, removal of incentives, and prevention and treatment of complications[6], and the treatment principles are basically the same as those for other DKA. After the patient developed euDKA, SGLT2i was stopped in time, and supplemented with fluid and alkali supplementation, insulin hypoglycemic therapy, and symptomatic and supportive treatment, the patient's DKA was relieved. In order to prevent DKA caused by SGLT2i, we should first identify whether the patient has high-risk factors, and try to avoid using SGLT2i when the patient has high-risk factors.

5. Conclusions

Most euDKA patients can recover with correct diagnosis and treatment. To detect euDKA in a timely manner, nurses should accurately identify euDKA-specific symptoms and triggers, closely monitor changes in circulatory indicators such as blood pressure, and dynamically monitor changes in electrolytes, the blood glucose level, the serum potassium level, pH, and the serum bicarbonate level during the course of clinical nursing care. Nurses should determine whether the patient has metabolic acidosis and perform urine or blood analysis for ketones in a timely manner. For euDKA after cardiac surgery caused by SGLT2i treatment, during fluid replacement, changes in serum potassium levels should be closely monitored, the status of the cardiac volume load should be dynamically evaluated, and the doctor should be notified immediately of any abnormality for timely treatment to promote the early recovery of the patient.

Author Contributions: Conceptualization, X.X.R. and W.Y.; methodology, Z.S.; validation, D.Y., Z.S.; formal analysis, W.Y.; investigation, X.X.R.; resources, D.Y.; data curation, X.X.R.; writing—original draft preparation, X.X.R; writing—review and editing, Z.S.; supervision, S.W. and Y.M.T. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Ethical review was waived as this single case report presents clinical observations from routine medical care without experimental interventions.

Informed Consent Statement: Informed consent was obtained from the patient involved in the study. Written informed consent has been obtained from the patient to publish this paper.

Data Availability Statement: Data are contained within the article.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Koceva A, Kravos T N. From Sweet to Sour: SGLT-2-Inhibitor-Induced Euglycemic Diabetic Ketoacidosis[J]. J Pers Med, 2024,14(7).
2. Auerbach J S, Gershengorn H B, Aljure O D, et al. Postcardiac Surgery Euglycemic Diabetic Ketoacidosis in Patients on Sodium-Glucose Cotransporter 2 Inhibitors[J]. J Cardiothorac Vasc Anesth, 2023,37(6):956-963.
3. Bonora B M, Avogaro A, Fadini G P. Euglycemic Ketoacidosis[J]. Curr Diab Rep, 2020,20(7):25.
4. Long B, Lentz S, Koyfman A, et al. Euglycemic diabetic ketoacidosis: Etiologies, evaluation, and management[J]. Am J Emerg Med, 2021,44:157-160.

 doi:10.20944/preprints202502.1069.v1

5. Danne T, Garg S, Peters A L, et al. International Consensus on Risk Management of Diabetic Ketoacidosis in Patients With Type 1 Diabetes Treated With Sodium-Glucose Cotransporter (SGLT) Inhibitors[J]. Diabetes Care, 2019,42(6):1147-1154.
6. Douros A, Lix L M, Fralick M, et al. Sodium-Glucose Cotransporter-2 Inhibitors and the Risk for Diabetic Ketoacidosis : A Multicenter Cohort Study[J]. Ann Intern Med, 2020,173(6):417-425.
7. Yuen K C, Tritos N A, Samson S L, et al. AMERICAN ASSOCIATION OF CLINICAL ENDOCRINOLOGISTS AND AMERICAN COLLEGE OF ENDOCRINOLOGY DISEASE STATE CLINICAL REVIEW: UPDATE ON GROWTH HORMONE STIMULATION TESTING AND PROPOSED REVISED CUT-POINT FOR THE GLUCAGON STIMULATION TEST IN THE DIAGNOSIS OF ADULT GROWTH HORMONE DEFICIENCY[J]. Endocr Pract, 2016,22(10):1235-1244.
8. Engelman D T, Ben A W, Williams J B, et al. Guidelines for Perioperative Care in Cardiac Surgery: Enhanced Recovery After Surgery Society Recommendations[J]. JAMA Surg, 2019,154(8):755-766.
9. Long B, Lentz S, Koyfman A, et al. Euglycemic diabetic ketoacidosis: Etiologies, evaluation, and management[J]. Am J Emerg Med, 2021,44:157-160.
10. Modi A, Agrawal A, Morgan F. Euglycemic Diabetic Ketoacidosis: A Review[J]. Curr Diabetes Rev, 2017,13(3):315-321.
11. Pontes J, de Melo C S, Arantes F, et al. Perioperative euglycemic diabetic ketoacidosis following use of SGLT-2 inhibitors after cardiac surgery[J]. J Clin Anesth, 2021,71:110201.
12. Yu X, Zhang S, Zhang L. Newer Perspectives of Mechanisms for Euglycemic Diabetic Ketoacidosis[J]. Int J Endocrinol, 2018,2018:7074868.
13. Bonora B M, Avogaro A, Fadini G P. Euglycemic Ketoacidosis[J]. Curr Diab Rep, 2020,20(7):25.
14. Sell J, Haas N L, Korley F K, et al. Euglycemic Diabetic Ketoacidosis: Experience with 44 Patients and Comparison to Hyperglycemic Diabetic Ketoacidosis[J]. West J Emerg Med, 2023,24(6):1049-1055.
15. Cardoso L, Vicente N, Rodrigues D, et al. Controversies in the management of hyperglycaemic emergencies in adults with diabetes[J]. Metabolism, 2017,68:43-54.
16. Van Ness-Otunnu R, Hack J B. Hyperglycemic crisis[J]. J Emerg Med, 2013,45(5):797-805.
17. Fayfman M, Pasquel F J, Umpierrez G E. Management of Hyperglycemic Crises: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State[J]. Med Clin North Am, 2017,101(3):587-606.
18. Dhatariya K K. The management of diabetic ketoacidosis in adults-An updated guideline from the Joint British Diabetes Society for Inpatient Care[J]. Diabet Med, 2022,39(6):e14788.
19. Calimag A, Chlebek S, Lerma E V, et al. Diabetic ketoacidosis[J]. Dis Mon, 2023,69(3):101418.
20. Palmer B F, Carrero J J, Clegg D J, et al. Clinical Management of Hyperkalemia[J]. Mayo Clin Proc, 2021,96(3):744-762.
21. Kitabchi A E, Umpierrez G E, Murphy M B, et al. Hyperglycemic crises in adult patients with diabetes: a consensus statement from the American Diabetes Association[J]. Diabetes Care, 2006,29(12):2739-2748.
22. Plewa M C, Bryant M, King-Thiele R. Euglycemic Diabetic Ketoacidosis[J]. 2025.
23. Dagdeviren M, Akkan T, Ertugrul D T. Re-emergence of a forgotten diabetes complication: Euglycemic diabetic ketoacidosis[J]. Turk J Emerg Med, 2024,24(1):1-7.
24. Milder D A, Milder T Y, Kam P. Sodium-glucose co-transporter type-2 inhibitors: pharmacology and peri-operative considerations[J]. Anaesthesia, 2018,73(8):1008-1018.
25. Parra M F, Brown M L, Staffa S J, et al. Post-operative vomiting and enhanced recovery after congenital cardiac surgery[J]. Cardiol Young, 2023,33(2):260-265.
26. Nasa P, Chaudhary S, Shrivastava P K, et al. Euglycemic diabetic ketoacidosis: A missed diagnosis[J]. World J Diabetes, 2021,12(5):514-523.
27. Barski L, Eshkoli T, Brandstaetter E, et al. Euglycemic diabetic ketoacidosis[J]. Eur J Intern Med, 2019,63:9-14.
28. Goto S, Ishikawa J Y, Idei M, et al. Life-Threatening Complications Related to Delayed Diagnosis of Euglycemic Diabetic Ketoacidosis Associated with Sodium-Glucose Cotransporter-2 Inhibitors: A Report of 2 Cases[J]. Am J Case Rep, 2021,22:e929773.
29. Chow E, Clement S, Garg R. Euglycemic diabetic ketoacidosis in the era of SGLT-2 inhibitors[J]. BMJ Open Diabetes Res Care, 2023,11(5).

 doi:10.20944/preprints202502.1069.v1

30. Liu J, Li L, Li S, et al. Sodium-glucose co-transporter-2 inhibitors and the risk of diabetic ketoacidosis in patients with type 2 diabetes: A systematic review and meta-analysis of randomized controlled trials[J]. Diabetes Obes Metab, 2020,22(9):1619-1627.
31. Henry R R, Thakkar P, Tong C, et al. Efficacy and Safety of Canagliflozin, a Sodium-Glucose Cotransporter 2 Inhibitor, as Add-on to Insulin in Patients With Type 1 Diabetes[J]. Diabetes Care, 2015,38(12):2258-2265.
32. Goldenberg R M, Berard L D, Cheng A, et al. SGLT2 Inhibitor-associated Diabetic Ketoacidosis: Clinical Review and Recommendations for Prevention and Diagnosis[J]. Clin Ther, 2016,38(12):2654-2664.
33. Goldenberg R M, Berard L D, Cheng A, et al. SGLT2 Inhibitor-associated Diabetic Ketoacidosis: Clinical Review and Recommendations for Prevention and Diagnosis[J]. Clin Ther, 2016,38(12):2654-2664.