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Postoperative Transition from Euglycemic Diabetic Ketoacidosis to Diabetic Ketoacidosis: A Case Report

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Abstract

Objective: This study aims to analyze the causes of postoperative transition from euglycemic diabetic ketoacidosis (EDKA) to diabetic ketoacidosis (DKA), summarize clinical nursing experiences, enhance the recognition and management of such complications, and improve patient prognosis. **Methods:** A detailed case analysis was conducted on a patient who developed EDKA early after breast cancer surgery, which subsequently transitioned to DKA. A multidisciplinary team (MDT) consultation was employed to formulate a personalized nursing plan. Specific methods included comprehensive clinical data collection, monitoring of blood glucose, urine ketones, and blood ketone levels; implementing dynamic insulin adjustment strategies; providing dietary education and psychological support; and guiding dietary adjustments through nutritional consultations. **Results:** Through personalized observation, blood glucose management, dietary management, psychological care, and wound care, the patient's blood and urine ketone levels returned to normal, the flap healed well, and blood glucose was maintained within the normal range. The patient is currently undergoing postoperative adjuvant chemotherapy. **Conclusion:** For postoperative patients with unexplained nausea, vomiting, and dehydration, regardless of diabetes history, timely testing of blood glucose, blood ketones, blood urea nitrogen, creatinine, electrolytes, and blood gas analysis can facilitate early detection of EDKA. Additionally, personalized management of blood glucose, diet, psychological care, and wound care is crucial for the prevention and treatment of EDKA.

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Keywords

Breast Cancer, Diabetic Ketoacidosis (DKA), Euglycemic Diabetic Ketoacidosis (EDKA), Blood Glucose, Nursing Care

1. Introduction

Diabetic Ketoacidosis (DKA) is a severe acute complication frequently occurring in diabetic patients, particularly those with type 1 diabetes [1]. It is characterized by the triad of hyperglycemia, elevated ketone levels, and metabolic acidosis, with diagnostic criteria including blood glucose levels over 13.9 mmol/L, metabolic acidosis (arterial blood pH < 7.3, serum HCO_3^- < 18 mmol/L), and positive urine or blood ketones. The prevalence of DKA is rising in correlation with the increasing incidence of diabetes. Clinical manifestations include increased polyuria, excessive thirst, and fatigue. Following metabolic decompensation, patients may experience decreased appetite, nausea, and vomiting, often accompanied by headaches, irritability, and drowsiness. Breathing becomes deep and rapid, with breath that smells like acetone (fruity odor). As the condition progresses, symptoms can include reduced urine output, dry skin and mucous membranes, sunken eyes, a rapid but weak pulse, decreased blood pressure, and cold extremities. In advanced stages, there may be varying degrees of consciousness impairment, leading to coma [2]. In recent years, the incidence of DKA has been rising in parallel with the increasing prevalence of diabetes. Studies from various geographic regions show that the annual incidence of DKA can reach as high as 56‰ per year [3]. Recent estimates report in-hospital mortality rates for DKA among patients with type 1 diabetes as 0.20% and 1.04% for those with type 2 diabetes in developed countries [4] [5]. However, reported mortality rates in low- and middle-income countries are significantly higher, ranging from 26% to 41.3%, likely due to delays in diagnosis and treatment [6]. Therefore, timely detection and treatment are crucial.

However, in certain cases, DKA may be misdiagnosed or diagnosed late. Euglycemic Diabetic Ketoacidosis (EDKA) is an uncommon but serious complication of diabetes, characterized by normal or slightly elevated blood glucose levels (generally <11.1 mmol/L), high anion gap metabolic acidosis, and positive urine or blood ketones. Its diagnosis is often delayed due to normal blood glucose levels [7]. EDKA is a specific type of DKA, accounting for approximately 2.6% to 7% of DKA cases [7]. EDKA has an insidious onset and, compared to DKA, presents with lower blood glucose levels, mild thirst, and dehydration, making early recognition challenging [8]. Major triggers for EDKA include low-carbohydrate ketogenic diets, fasting, pregnancy, acute infections, and surgery [9]. Like DKA, EDKA is a life-threatening acute condition, and early recognition and treatment are crucial. Enhancing healthcare professionals' awareness of EDKA can effectively prevent diagnostic delays.

This article introduces a detailed case analysis of a patient who developed EDKA

early after breast cancer surgery, which subsequently transitioned to DKA. Through multidisciplinary discussions, the study explores the reasons for the postoperative transition from EDKA to DKA and summarizes clinical nursing experiences. The aim is to enhance clinical recognition and management capabilities for such complications.

2. Clinical Data

The patient is a 55-year-old female who walked into the hospital on July 24, 2024, with a diagnosis of a mass in the right breast. She has a medical history of type II diabetes for 18 years, managed with oral medications including gliflozins, metformin, atorvastatin, and weekly subcutaneous injections of dulaglutide, although her blood sugar control has been poor. Ten years ago, she suffered a cerebral infarction, which was not treated; she experiences limb numbness and has been taking mecobalamin. She also has a history of hepatitis B and reported a history of depression since 2023, for which she has been taking sertraline as prescribed. Upon admission, the patient underwent comprehensive preoperative examinations. Following medical advice, blood glucose was monitored four times daily (QID). Abnormal preoperative test results included glucosuria at 4+ mmol/L with negative ketone bodies, blood glucose at 13.93 mmol/L, an anion gap of 16.9, total carbon dioxide at 22.4 mmol/L, and uric acid at 450.5 μmol/L. The patient's blood glucose control was inadequate before surgery, as detailed in Table 1, which shows that post-breakfast blood glucose levels were normal, but control at other times was generally poor. Additionally, the patient experienced abnormal blood glucose levels the day before surgery due to overeating in anticipation of fasting on the surgery day.

Table 1. Preoperative abnormal blood glucose conditions and management measures for patients.

Date	Fasting Blood Glucose (mmol/L)	Post-Breakfast Blood Glucose (mmol/L)	Post-Dinner Blood Glucose (mmol/L)	Post-Dinner Blood Glucose (mmol/L)
7-24 (Day of Admission)	/	/	/	• 18.3, patient forgot to take hypoglycemic medication, instructed to take it. • Rechecked 2 h later: 13.5, no further action.
7-25	8.6		• 14.6, no action taken • Rechecked 2 h later: 12.6	• 14.9, no action taken
7-26	8.6		• 13.3, no action taken	
7-27	7.2			• 12.2, no action taken
7-28	8.1			
7-29 (Day Before Surgery)	7.3		• 15.3, no action taken	• 17.9, no action taken • Rechecked 1 h later: 13.1

On July 30, the patient underwent surgery under general anesthesia, which included right breast-conserving, subcutaneous gland resection, right axillary lymph node biopsy, and right breast implant insertion. On the second postoperative day, the implant flap exhibited ecchymosis over an area of approximately 9 ×

4 cm. On the third postoperative day, she developed symptoms such as vomiting, runny nose, dizziness, constipation, dry mouth, and decreased limb strength, with postprandial blood glucose maintained between 9.1 and 12 mmol/L. On the fourth postoperative day, symptoms including dry mouth, nausea, vomiting, dizziness, and finger numbness were noted, with a peak blood glucose level of 17.7 mmol/L. Blood gas analysis indicated severe uncompensated metabolic acidosis combined with respiratory alkalosis, likely related to acidosis from vomiting, diabetic nephropathy, and possibly diabetic ketoacidosis. She was diagnosed with DKA, leading to a critical condition alert, and was transferred to the intensive care unit (ICU) on August 4th. On August 6th, after receiving treatment in the ICU, the patient was transferred back to the ward in stable condition with generally good health and stable vital signs. On the same day, a multidisciplinary team (MDT) discussion was conducted to retrospectively analyze the case and provide guidance on subsequent diagnosis, treatment, and nursing care. During this period, the patient recovered well. For further treatment, after discussions with the family, the patient was transferred to the endocrinology department of a general hospital on August 12th.

At a follow-up on September 3, the patient reported that she received symptomatic treatment at the other hospital until her ketone levels normalized. Following discharge, her blood glucose has been managed effectively with oral antidiabetic medications and insulin injections, and her appetite and intake have returned to normal, although ecchymosis at the breast wound site remains. At follow-up on September 11, her flap has healed well, her blood glucose is well controlled, and she is undergoing postoperative adjuvant chemotherapy. Acute changes and the treatment course are detailed in Table 2.

Table 2. The acute progression and treatment of postoperative diabetic ketoacidosis in patients.

	Date and Time	Symptoms	Positive Findings	Blood Glucose (mmol/L)	Urinary Ketones (mmol/L)	Blood Gas Analysis	Treatment Measures
Postoperative Day 3	Aug 2, 09:00	Vomiting, runny nose, dizziness, constipation	None	9.1	/	/	Paracetamol, lactulose, etc.
	Aug 2, 15:30	Dry mouth, worsening dizziness, upper limb muscle strength: grade 4, lower limb: grade 1, vomiting	• AG: 29.5↑ • TCO_2: 4 mmol/L↓ • UA: 747.1 μmol/L↑	9.7	/	/	Oral concentrated sodium, levofloxacin, etc.
Postoperative Day 4	Aug 3, 04:00	Vomited approximately 300ml of pale yellow fluid	None	12	/	/	None
	Aug 3, 09:00	Noticeable wheezing, moderate alertness, ~32 breaths/min, heavy breathing sounds, dizziness, normal muscle tension, weak fast pulse, pupils equal and round, normal light reflex, dry mouth, BP: 161/84 mmHg, HR: 131 bpm	• D-Dimer: 1.1 μg/mL↑ • Sodium: 133.1 mmol/L↓ • AG: 31.5↑ • TCO_2: 2 mmol/L↓ • UA: 858.3 μmol/L↑ **Chest X-ray:** No significant abnormalities in the heart and lungs. **ECG:** • Sinus tachycardia • ST-T changes • Prolonged QTc interval • T-P wave fusion	14.9	/	/	Cardiac monitoring, low-flow oxygen, oral concentrated sodium chloride, electrolyte glucose infusions, 5% dextrose saline infusion, 24-hour input-output monitoring

Continued

	Aug 3, 13:30	Acidosis symptoms	/	17.7	4+	• Blood pH:6.94 • PCO_2: 11.4 mmHg • BE: −28.3 mmol/L • Severe uncompensated metabolic acidosis with respiratory alkalosis	ICU consultation, catheterization, furosemide diuretics, rapid bicarbonate infusion with follow-up blood gas and urinalysis, subcutaneous insulin for blood sugar control, nil by mouth except fluids, esmolol for heart rate control, critical condition alert
	Aug 3, 16:00	Suspected diabetic ketoacidosis	• Na: 147.9 mmol/L↑ • Cl: 109.9 mmol/L↑	9.7	4+	• Blood pH: 7.32 • PCO_2: 17.4 mmHg • BE: −14.8 mmol/L	500 ml compound sodium chloride + 1g potassium chloride IV
	Aug 3, Evening	Slow pupillary light reflex	• WBC: 17.74 × 10^9/L↑ • Sodium: 157.1 mmol/L↑ • Chloride: 118.9 mmol/L↑ • AG: 33.5↑ • TCO_2: 10.5 mmol/L↓ • UA: 1015.5 μmol/L↑	7.3	4+	• Blood pH: 7.32 • PCO_2: 21.9 mmHg • BE: −13.1 mmol/L	Cranial CT, 5% Glucose saline 250 ml + insuline R 3u at 50 ml/h IV infusion
Postoperative Day 5	Transferring to ICU Aug 4, 08:30	Sleepy but responsive, sluggish pupillary light reflex, upper limb muscle strength: grade 2, lower limb: grade 1, BP normal, HR max: 154 bpm, O_2 saturation normal	• WBC: 20.78 × 10^9/L↑ • Sodium: 161.3 mmol/L↑ • Chloride: 127.2 mmol/L↑ • AG: 26.5↑ • TCO_2: 6.4 mmol/L↓	10.7	4+	Blood pH: 7.08 PCO_2: 11.9 mmHg BE: −24.3 mmol/L	Transferred to ICU, continuous cardiac monitoring and oxygen therapy, gastric tube placement, CVC
Postoperative Day 6	ICU Day 1 Aug 5	Improved consciousness, follows simple commands	• WBC: 11.79 × 10^9/L↑ • Sodium: 146.4 mmol/L • Chloride: 111 mmol/L↑ • AG: 11.9 • TCO_2: 22.7 mmol/L↓ • UrA: 589.9 μmol/L↑	9.2	1+	Roughly normal	Esmolol micro-infusion, 5% glucose 500 ml + 1 g potassium chloride + 6u insulin IV, gastric tube potassium replenishment, comprehensive chest and cranial CT
Postoperative Day 7	ICU to Ward Aug 6, 10:00	Alert, responsive, upper and lower limb muscle strength: grade 4, normal heart rate	• WBC: 11.0579 × 10^9/L↑	9.2	Weak positive	None	Transfer to ward, cardiac monitoring, oxygen therapy, oral antidiabetic medication, pre-meal and bedtime insulin injection, additional insulin based on post-meal blood sugar, apply alcohol compress for wound hematoma
Postoperative Day 8	ICU to Ward Day 1 Aug 7	Alert, responsive	• Phosphorus: 0.8 mmol/L↓	4.7	Weak positive	None	Phosphate supplementation, blood sugar checks before meals and 2 h post-meal, 5% glucose saline 500 ml + 1 g potassium chloride + 6u insulin IV daily

Note: TCO_2: Total Carbon Dioxide, AG: Anion Gap, UA: Uric Acid, ECG: Electrocardiogram, BE: Base Excess, WBC: White Blood Cells.

3. Discussion

The incidence of EDKA is low, but it is difficult to detect early and can have serious consequences. This study presents the treatment process for a patient who developed EDKA early after breast cancer surgery, which then transitioned to DKA. It emphasizes the importance of monitoring the patient's condition and highlights the successful management of the situation through personalized blood glucose, dietary, psychological, and wound care. This approach ultimately led to the successful rescue and recovery of the patient.

3.1. Condition Monitoring

In this case, the patient exhibited symptoms such as vomiting, dizziness, and altered mental status on the third postoperative day. At the time, the patient's blood glucose level was only 9.1 mmol/L, and given the history of cerebral infarction, there was an initial suspicion of a reaction to postoperative anesthetics or a recurrence of cerebral infarction. Consequently, a cranial CT scan was performed, which showed normal results. However, the patient continued to experience dry mouth and numbness in the fingers despite consuming a large amount of water. Further tests, including D-dimer, routine biochemistry, and ECG, revealed elevated white blood cells and electrolyte imbalances. However, abnormalities in the anion gap and total carbon dioxide in routine biochemistry were overlooked, despite significant changes compared to admission values. An increased anion gap is clinically significant and is associated with metabolic acidosis [10]. Approximately 95% of serum total carbon dioxide is in the form of HCO_3^-, so a decrease in total carbon dioxide indicates metabolic acidosis or respiratory alkalosis [11]. Consequently, despite electrolyte supplementation and anti-inflammatory symptomatic treatment, the patient's condition deteriorated, resulting in drowsiness and increased heart rate. A subsequent urinalysis revealed 4+ ketones, and blood gas analysis indicated severe uncompensated metabolic acidosis with respiratory alkalosis.

In this case, the patient's postoperative symptoms of nausea, vomiting, and dizziness were initially mistaken for reactions to anesthetics or a recurrence of cerebral infarction. However, because blood glucose levels did not significantly rise, the diagnosis of DKA was delayed. In discussing DKA and euglycemic diabetic ketoacidosis (EDKA), understanding the distinctions and diagnostic challenges of these conditions is crucial. DKA typically features high blood glucose, high ketones, and metabolic acidosis, and is a common acute complication in diabetic patients. EDKA, on the other hand, is a rare but severe complication characterized by normal or only slightly elevated blood glucose levels. Its symptoms can include dizziness, nausea, vomiting, dehydration, and abdominal pain, though they may not be as typical as those of DKA.

In EDKA, normal blood glucose levels can lead to misdiagnosis or missed diagnosis. Symptoms similar to typical DKA—such as nausea, vomiting, abdominal pain, rapid breathing, and altered consciousness—can be confused with other conditions like infection, chronic liver disease, pregnancy, or alcohol consumption, which increases the risk of misdiagnosis. Therefore, in diabetic patients, it's important to promptly recognize early symptoms like vomiting, dizziness, changes in mental status, and increased heart and respiration rates, especially in the early postoperative phase. These symptoms may resemble those of other diseases, so vigilance is necessary. For patients with unexplained nausea, vomiting, or dehydration, regardless of diabetes history, the possibility of this condition should be considered. Immediate testing for blood glucose, blood ketones, blood urea nitrogen, creatinine, electrolytes, and a blood gas analysis can confirm or

exclude this diagnosis. Special attention should be given to the anion gap and serum total carbon dioxide to identify the root cause early on.

3.2. Blood Glucose Management

Upon admission, the patient was prescribed blood glucose monitoring four times a day (QID) and instructed to continue taking their own oral antidiabetic medications (SGLT2 inhibitors, metformin, and atorvastatin). Preoperative fasting blood glucose (≤8.7 mmol/L) and postprandial blood glucose values (≤14.9 mmol/L) were not stable, which may have contributed to the risk of postoperative DKA. Post-surgery, the patient resumed blood glucose monitoring as per preoperative instructions, with oral antidiabetic medication continued. During the first three days post-surgery, blood glucose levels were controlled between 9.1 and 12 mmol/L. On the fourth postoperative day, the patient developed DKA and was transferred to the ICU; after stabilization, they returned to the ward. Subsequently, the protocol was changed to monitor blood glucose every two hours, with additional checks two hours post-meal to promptly adjust insulin dosages. When glucose levels dropped below 14 mmol/L, the doctor was notified to immediately initiate a glucose polarization solution of 10% glucose at a rate of 125 ml/h to prevent hypoglycemia and hypokalemia. Polarization solutions, typically composed of glucose, insulin, and potassium, effectively reduce blood glucose levels, replenish potassium ions, and improve cellular metabolism [12]. By dynamically adjusting insulin doses, the patient's fasting blood glucose was eventually maintained at 5.6 - 6.9 mmol/L, and postprandial glucose was controlled at 5.3 - 8.9 mmol/L.

This case emphasizes the importance of perioperative blood glucose management. A comprehensive preoperative assessment should be conducted for diabetic patients, including evaluations of glycated hemoglobin (HbA1c) and routine urinalysis, to assess past blood glucose control. The patient's inadequate blood glucose management at admission and significant fluctuations postoperatively increased the risk of DKA. The Centre for Perioperative Care (CPOC) recommends that the key to successful perioperative management plans lies in frequent monitoring of glucose, electrolytes, and fluid concentrations [13]. In this case, insufficient perioperative blood glucose monitoring failed to adequately capture glucose fluctuations, impacting the adjustment of treatment plans. For diabetic inpatients undergoing surgery, it is crucial to refine preoperative blood glucose monitoring protocols, including checks with each meal and fasting. During hospitalization, insulin should be uniformly used to ensure blood glucose control within 8 - 10 mmol/L before proceeding with surgery.

3.3. Dietary Management

In this case, although the patient's blood glucose was normal immediately after surgery, inadequate food intake and improper dietary choices, especially the excessive restriction of carbohydrates, led to insufficient insulin utilization and

ineffective blood glucose use. As a result, the body started breaking down fats for energy, producing a large amount of ketones [14]. Based on the patient's height, weight, and basal metabolic rate, the required caloric intake was calculated to be around 1500 to 1800 kcal, with daily protein intake generally between 0.8 to 1.2 grams per kilogram of body weight, translating to approximately 51.7 to 77.5 grams per day. Since the patient's actual dietary structure was far from adequate, we advised starting with small, easily digestible meals and gradually increasing intake. Foods rich in dietary fiber, such as vegetables and whole grains, and high-quality protein sources, such as chicken, fish, eggs, and dairy, were recommended to help repair tissues and slow blood glucose elevation. It was suggested that the patient follow a quantitative dietary principle, with grains and whole grains making up one-third of each meal, and carbohydrates accounting for 40% - 60% of the intake. The recommended eating order is to consume vegetables before the main course, adjusting based on blood glucose levels. Through these interventions, the patient was able to maintain a balanced intake and output, move around independently, regain appetite, and achieve good blood glucose control.

Inadequate food intake and improper diet are among the triggering factors for EDKA, and in some cases (such as prolonged starvation), the body's metabolic state may continue to deteriorate, leading to DKA. Therefore, the care team should enhance dietary education for patients, ensuring a reasonable distribution of carbohydrate, protein, and fat intake to prevent the occurrence of DKA.

3.4. Psychological Care

In this case, the patient has a history of depression and has been on long-term treatment with sertraline. Due to sertraline's peak plasma concentration time of 4.5 - 8.4 hours, caution is necessary to avoid nocturnal hypoglycemic reactions [15]. The patient previously took sertraline after dinner and reported experiencing nocturnal hypoglycemia. Therefore, the patient was advised to switch sertraline intake to daytime, resulting in improved nighttime blood glucose stability, with fluctuations between 6.8 - 10.1 mmol/L, and alleviation of hypoglycemic symptoms. When using sertraline, it should not be combined with monoamine oxidase inhibitors to prevent severe adverse reactions [16]. Additionally, when used in conjunction with antidiabetic medications, it is crucial to closely monitor the patient's blood glucose levels and adjust medication dosages as needed [17].

3.5. Wound Management

In diabetic patients, the risk of postoperative complications following breast cancer surgery with implant placement increases, leading to prolonged hospital stays and significantly higher rates of wound infection and necrosis [18]. In this case, the patient developed extensive bruising on the implant flap post-surgery. Although cultures of body fluid samples showed no bacterial or fungal infections, elevated levels of white blood cells and interleukins were noted, prompting symptomatic treatment with levofloxacin. The issues of high tension, poor blood

supply, and bruising in the surgical area were initially addressed by applying epidermal growth factor (EGF) to the wound, but with limited success. After a MDT discussion, the pros and cons of interventions such as heat lamps, Hirudoid cream, and alcohol compresses were analyzed. EGF primarily promotes the growth and repair of epidermal cells [19], but may not be effective for patients with bruised flaps due to issues like vascular embolism that hinder blood supply improvement. Heat lamps could increase local temperature, exacerbate tissue edema, and potentially impair flap perfusion, which is not conducive to recovery [20]. Hirudoid cream can help improve blood circulation and reduce bruising [21]. Alcohol compresses work through vasodilation, anti-inflammatory, cooling, and penetration effects. They dilate local blood vessels, improve circulation, alleviate bruising, and have antibacterial and anti-inflammatory properties; their volatility aids in cooling, reducing swelling and pain; and alcohol penetration softens tissue and enhances drug absorption. During use, it is important to prevent skin dryness or irritation and ensure the patient is not allergic to alcohol. Considering effectiveness, feasibility, and cost, and confirming the patient was not allergic to alcohol, daily alcohol compresses were chosen. The results showed reduced flap swelling, accelerated local blood circulation, and a decrease in the extent of bruising.

4. Conclusion

This study emphasizes the importance of early detection methods for euglycemic diabetic ketoacidosis (EDKA) in patients with unexplained nausea, vomiting, and dehydration, regardless of their diabetes history. Key tests include blood glucose, ketones, urea nitrogen, creatinine, electrolytes, and blood gas analysis. It also offers recommendations for preventing and treating EDKA, focusing on blood glucose management, dietary adjustments, psychological support, and wound care. These strategies aim to enhance clinical recognition and management of EDKA, thereby improving patient outcomes.

Conflicts of Interest

The authors declare no conflict of interest.

Ethical Approval

The procedures used in this study adhered to the tenets of the Declaration of Helsinki. In this case, the patient was informed and agreed to use their data as a research report, and the article did not involve the patient's real name, photo and other privacy.

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