

CASE REPORT

Received: 11 May 2025
Last revision: 03 October 2026
Accepted: 06 November 2026
Published: 19 November 2026

Management of Diabetic Ketoacidosis in a Patient with Acute Kidney Injury: A Case Report

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ABSTRACT

Introduction: Diabetic ketoacidosis (DKA) is a life-threatening acute complication of diabetes mellitus (DM) that may be accompanied by acute kidney injury (AKI), particularly in the presence of severe dehydration and precipitating infections. **Case:** A 32-year-old male presented with generalized weakness, polydipsia, and polyuria for two weeks. His random blood glucose level was 803 mg/dL. Further evaluation revealed DKA, stage 1 AKI of presumed pre-renal origin, and suspected urinary tract infection (UTI). **Discussion:** The patient had no previous history of diabetes mellitus or hypertension but had a family history of type 2 diabetes mellitus and reported frequent consumption of sugar-sweetened beverages and a sedentary lifestyle. Management consisted of intravenous fluid resuscitation, insulin infusion, potassium replacement, treatment of the suspected UTI, and close monitoring of metabolic and renal parameters. After three days of treatment, the patient improved clinically and recovered renal function. **Conclusion:** This case highlights the importance of early recognition of DKA and identification of concurrent AKI and potential infectious triggers. Prompt fluid resuscitation, insulin therapy, electrolyte correction, and treatment of precipitating factors are essential to prevent further organ dysfunction and improve clinical outcomes.

Keywords: Acute kidney injury, case report, diabetes mellitus, diabetic ketoacidosis.

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https://doi.org/10.55175/crm3g833
e-ISSN: 2503-2720

INTRODUCTION

Diabetic ketoacidosis (DKA) is one of the most serious acute complications of diabetes mellitus (DM), particularly in patients with type 1 diabetes, but increasingly recognized in type 2 diabetes.[1] It represents a life-threatening medical emergency requiring prompt recognition and management.[2] DKA is also imposes a considerable burden on healthcare systems, especially in low-resource settings. Its importance lies in its rapid onset, complex metabolic derangements, and potential for fatal outcomes if not treated in a timely and structured manner. DKA is often the initial presentation of diabetes in previously undiagnosed individuals, particularly among young adults and children, making it a critical aspect of diabetes care.[3]

Diabetic ketoacidosis (DKA) is an acute metabolic disorder characterized by elevated circulating ketone bodies that progressively lead to severe ketoacidosis accompanied by hyperglycaemia, primarily caused by insulin deficiency.[4] DKA occurs when there is a profound insulin deficiency that not only results in hyperglycaemia and severe dehydration but also leads to increased ketone production and metabolic acidosis.[5] It is considered an acute complication of uncontrolled diabetes mellitus. The lack of insulin triggers enhanced lipolysis and incomplete oxidation of free fatty acids, resulting in the accumulation of ketone bodies and an increase in the anion gap metabolic acidosis.[6] The condition is clinically characterized by a triad of hyperglycemia, acidosis, and ketosis, typically arising from absolute or relative insulin deficiency.[7]

DKA is more commonly observed in patients under the age of 65 and occurs more frequently in females than males.[8] In community-based data from Rochester, United States, the incidence of DKA was reported to be 8 per 1,000 diabetic patients per year across all age groups, with an incidence of 13.4 per 1,000 diabetic patients annually among individuals under the age of 30.[9] Other sources estimate the incidence of DKA to range from 4.6 to 8 per 1,000 diabetic patients per year.[10] Although there are no comprehensive community-based studies on the prevalence of DKA in Indonesia, the 2018 Riskesdas (National Basic Health Research) data reported an 8.5% prevalence of diabetes mellitus based on blood glucose levels.[11] The highest mortality rate due to DKA in Indonesia is observed in males aged 1 to 17 years.[12] Mortality rates for DKA in developed countries range from 5%–10% in large hospitals, but can rise to 25% in clinics with limited resources.[13] Mortality risk increases when DKA is accompanied by conditions such as sepsis, shock, extensive myocardial infarction, advanced age, extremely elevated blood glucose, uremia, or severe acidosis.[14]

Several factors can precipitate the onset of DKA, including infections, acute vascular events, trauma, burns, heat stroke, acute pancreatitis, cholecystitis, and the use of medications such as corticosteroids.[15] In patients with a known diagnosis of diabetes mellitus who are on insulin therapy,

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hyperglycemic crises may arise due to inadequate insulin dosing or suboptimal diabetes management.[7] Absolute or relative insulin deficiency leads to intracellular starvation in insulin-dependent tissues such as muscle, liver, and adipose tissue. This triggers the release of counter-regulatory hormones including glucagon, catecholamines, cortisol, and growth hormone. The development of hyperglycemia and ketosis in DKA results from insulin deficiency combined with elevated levels of these counter-regulatory hormones. The major metabolic disturbances leading to hyperglycemia include increased gluconeogenesis, accelerated glycogenolysis, and impaired peripheral glucose utilization.[12,16]

Although diabetic ketoacidosis is well recognized as an acute metabolic complication of diabetes mellitus, the simultaneous occurrence of DKA, acute kidney injury, and suspected urinary tract infection in a patient with newly diagnosed type 2 diabetes mellitus presents an important clinical management challenge. This case is clinically significant because it requires prompt identification of a potential precipitating infection while correcting severe metabolic disturbances and maintaining adequate renal perfusion. In this patient, early fluid resuscitation, insulin therapy, electrolyte correction, and treatment of the suspected infection were followed by improvement in both metabolic status and renal function. This case report aims to describe the clinical presentation, diagnostic evaluation, management, and clinical response of a patient with diabetic ketoacidosis complicated by stage 1 acute kidney injury and suspected urinary tract infection.

CASE

A 32-year-old male, was referred to the Emergency Department. The patient reported persistent generalized weakness for one week that significantly interfered with daily activities. The patient denied any previous episodes of similar weakness. No aggravating or relieving factors were identified.

The patient also complained of persistent thirst for the past two weeks, consuming one gallon of water every 3–5 days. He also reported increased micturition frequency, approximately once every hour. One week prior, the patient experienced a transient febrile episode lasting approximately one hour, resolved spontaneously after drinking fruit juice; no temperature measurement or medication was undertaken. No reported changes in appetite, bowel or bladder function, nausea, or vomiting. The patient reported a known allergy to paracetamol.

Weakness occurred after the patient consumed a packaged sweetened beverage on around twelve hours before visit to Primary Health Centre. He subsequently experienced abdominal discomfort followed by persistent fatigue. On the morning of the next day, seven hours before referral to Emergency Department Hospital A, the patient visited Primary Health Center, where a random blood glucose measurement revealed a glucose level of 803 mg/dL. The patient was immediately referred to Emergency Department of Hospital A for further management.

In the Emergency Department of Hospital A, the patient received 1 liter of intravenous 0.9% NaCl over 30 minutes and 8 IU IV insulin bolus. Although his clinical condition slightly improved, blood glucose decreased only to 320 mg/dL. Consequently, the patient was referred Emergency Department of Hospital B after two hours under management in Hospital A. The patient had no known history of diabetes mellitus or hypertension. However, his father had type 2 diabetes mellitus and had been undergoing hemodialysis for six years. Several paternal family members also had a history of type 2 diabetes mellitus.

The patient worked as an administrative staff at a private company, with a sedentary lifestyle without regular physical exercise. He reported active smoking and alcohol consumption, as well as frequent intake of 2 to 3 sweetened packaged beverages per day.

Initial evaluation of hyperglycemic patients should be based on clinical presentation, including metabolic assessment, identification of infectious sources, and comorbidity screening. Initial investigations should include random blood glucose, serum ketones, electrolytes, serum osmolality, arterial blood gas analysis (if ketones are present), renal function tests, HbA1c, and urinalysis. Further workup for precipitating factors such as infection should be performed with complete blood count, chest x-ray, and urine culture.[17]

On initial assessment, the patient was mildly ill, alert (GCS: E4V5M6), with a blood pressure 130/80 mmHg, an axillary temperature 37.2 °C, a pulse rate 85 bpm, an oxygen saturation 97% on room air, and a respiratory rate 18 breaths per minute. Physical examination revealed no signs of anemia or jaundice. Pupillary reflexes were normal. ENT examination was unremarkable, and no cervical lymphadenopathy was noted. Cardiac auscultation revealed a normal heart rate without murmurs. Pulmonary examination revealed regular breath sounds without rales or wheezing. Abdominal examination showed no distention, normal bowel sounds, and no palpable masses, hepatosplenomegaly, or tenderness. Tympanic percussion was present throughout all quadrants. All extremities were warm to the touch.

Laboratory results at admission to Hospital B showed a random blood glucose of 337 mg/dL. Complete blood count was within normal limits. Blood chemistry revealed elevated ALT (48.00 U/L), increased serum creatinine (1.40 mg/dL), and decreased estimated glomerular filtration rate (eGFR) of 60.01 mL/min/1.73 m^2. Arterial blood gas analysis showed metabolic acidosis with a pH of 7.24 and reduced HCO_3^- at 10.70 mmol/L. Urinalysis revealed glycosuria (4+), ketonuria (3+), and the presence of bacteria (130.30/µL), suggestive of a urinary tract infection. Chest x-ray showed no abnormalities in the lungs or heart.

Based on the patient's history, physical examination, and supporting investigations, the diagnosis was diabetic ketoacidosis (DKA), type 2 diabetes mellitus with stage 1 acute kidney injury (AKI) of pre-renal origin, and urinary tract infection (UTI). The management plan included monitoring blood glucose, glycemic control, treatment of diabetes mellitus, management of UTI, and AKI treatment.

In this case, initial fluid resuscitation included 1,000 mL NaCl 0.9% IV infusion over 30




minutes, followed by 500 mL over 30 minutes, 1,000 mL over 1 hour, 500 mL over another hour, and 250 mL in the final hour. Maintenance fluids used NaCl 0.9% at 20 drops/min. Starting from the second hour, insulin infusion (drip) was initiated at 4 units/hour. Two hours after referred to Hospital B, the patient's blood glucose was 400 mg/dL. Glucose was monitored hourly. Two hours after the referral, blood glucose had decreased to 302 mg/dL, and the insulin drip rate was reduced to 2 units/hour. A glucose reduction rate of 50–75 mg/dL per hour was maintained to achieve controlled glucose lowering.

For long-term diabetes management, the patient was placed on a diabetic diet of 1,900 kcal/day and insulin glargine 28 units subcutaneously every 24 hours. To treat the suspected urinary tract infection, the patient received cefixime 200 mg every 12 hours for 7 days.

DISCUSSION

The diagnosis of diabetes mellitus in this patient was established based on a markedly elevated random blood glucose level of 803 mg/dL. This value meets the diagnostic criteria set by the Indonesian Society of Endocrinology (Perkumpulan Endokrinologi Indonesia/PERKENI), which include the following:[18]

1. Fasting plasma glucose ≥ 126 mg/dL after at least 8 hours of fasting, OR
2. Random plasma glucose ≥ 200 mg/dL accompanied by classic symptoms of diabetes mellitus, OR
3. Plasma glucose ≥ 200 mg/dL measured 2 hours after a 75-gram oral glucose tolerance test (OGTT), OR
4. Hemoglobin A1c (HbA1c) ≥ 6.5%.

Laboratory findings showed metabolic acidosis with a pH of 7.24 and a bicarbonate level of 10.70 mmol/L, along with positive urine ketones (3+), supporting the diagnosis of diabetic ketoacidosis (DKA). The patient also presented with elevated serum creatinine of 1.40 mg/dL and reduced eGFR of 60.01 mL/min/1.73 m², indicating renal impairment.

Diabetic ketoacidosis (DKA) is a life-threatening acute complication of diabetes mellitus (DM). It is characterized by a triad of hyperglycemia, ketonemia, and metabolic acidosis. DKA most commonly occurs in individuals with newly diagnosed diabetes, particularly type 1, but can also arise in type 2 diabetes under stress conditions.[18] Several risk factors are associated with the development of DKA, including newly diagnosed diabetes, non-adherence to insulin therapy, infections, myocardial infarction, trauma, thyrotoxicosis, the use of antipsychotic medications, and acute abdominal illnesses.[3] Among infectious triggers, pneumonia and urinary tract infections are the most frequently reported.[4] Clinical manifestations of DKA include polyuria, polydipsia, dehydration, gastrointestinal symptoms, Kussmaul respiration, and, in severe cases, altered mental status.[19]

In diabetic ketoacidosis (DKA), insulin deficiency is the central pathological mechanism leading to hyperglycemia and subsequent loss of fluids and electrolytes through osmotic diuresis, resulting in extracellular fluid depletion. In parallel, the lack of insulin triggers an upregulation of counter-regulatory hormones such as glucagon, catecholamines, cortisol, and growth hormone. These hormones signal the body to generate alternative energy sources, primarily through lipolysis. This process involves the activation of lipase enzymes that break down adipose tissue into free fatty acids. Free fatty acids are subsequently converted into acetyl coenzyme A (acetyl-CoA). While a portion of acetyl-CoA enters the Krebs cycle, the excess is diverted to the hepatic production of ketone bodies. Although ketones can serve as an alternative energy source, they accumulate rapidly in the absence of sufficient insulin, contributing to metabolic acidosis. Additionally, ongoing protein catabolism and glycogenolysis further increase circulating glucose levels, exacerbating hyperglycemia and worsening the clinical picture of DKA.[20,21]

According to the Indonesian Society of Endocrinology (PERKENI).[18] consensus, diagnostic criteria for diabetic ketoacidosis (DKA) include marked hyperglycemia with blood glucose levels ranging from 300 to 600 mg/dL, accompanied by signs and symptoms of acidosis and strong positive plasma ketones. In addition, plasma osmolality is typically elevated (300–320 mOsm/kg), and an increased anion gap.[18] Tjokroprawiro A., *et al.*, further classify the diagnostic criteria for DKA into three domains:

(i) Clinical findings – polyuria, polydipsia, nausea and/or vomiting, Kussmaul respiration (deep and rapid breathing), generalized weakness, dehydration, hypotension progressing to shock, and altered mental status ranging from confusion to coma;
(ii) Laboratory blood findings – hyperglycemia > 300 mg/dL (often exceeding 500 mg/dL), bicarbonate < 20 mEq/L, pH < 7.35, and positive ketonemia;
(iii) Urine analysis – the presence of glucosuria and ketonuria.[17]

These criteria emphasize the importance of recognizing both biochemical abnormalities and clinical manifestations in establishing a diagnosis of DKA. The diagnostic criteria and severity classification of diabetic ketoacidosis are presented in **Table 1**.

Urinalysis in this patient revealed bacteria in the urine (130.30/µL) along with an elevated leukocyte count, suggesting a urinary tract infection (UTI). Bacterial infections, particularly UTIs and pneumonia, are among the most common precipitating factors for diabetic ketoacidosis (DKA), accounting for approximately 50% of cases.[17] Early diagnosis of infection in the context of DKA can be challenging, as many clinical and laboratory features overlap. Symptoms such as fever, leukocytosis, increased neutrophil percentage, elevated C-reactive protein (CRP), and procalcitonin (PCT) levels are common in both infection and DKA, potentially masking the underlying etiology. Infection triggers the release of stress hormones, including adrenaline and cortisol, which exert anti-insulin effects and promote insulin resistance. This hormonal response exacerbates hyperglycemia and ketogenesis, significantly increasing the risk of developing or worsening DKA.[22]

Renal function tests showed elevated serum creatinine and reduced eGFR, indicating renal impairment. Acute kidney injury (AKI) occurs in approximately 43% to 64% of episodes of diabetic ketoacidosis (DKA).[23] In this context, AKI is primarily due to hypovolemia caused




Table 1. Diagnosis criteria of diabetic ketoacidosis.[17]

	Plasma glucose > 250 mg/dL		
	Mild	Moderate	Severe
Arterial pH	7.25–7.30	7.00–< 7.24	< 7.00
Bicarbonate serum, mEq/L	15–18	10–< 15	< 10
Urine ketones	Positive	Positive	Positive
Serum ketones	Positive	Positive	Positive
Effective Serum Osmolality	Variable (may be increased)	Variable (may be increased)	Frequently increased
Anion gap	> 10	> 12	> 12
Mental status	Alert	Drowsy or lethargic	Stupor/coma

by osmotic polyuria induced by glucose and, in some cases, vomiting. AKI associated with DKA is typically mild, resolves rapidly with appropriate treatment, and is caused by decreased renal perfusion resulting from intravascular volume depletion. Several studies in adults with diabetes.[17,23] have identified a link between AKI and the progression to chronic kidney disease (CKD). According to the Indonesian Society of Nephrology (PERNEFRI) 2023 guidelines, the diagnostic criteria for AKI include **(Table 2)**.[23,24]

Pre-renal acute kidney injury (AKI) is the most common cause of kidney dysfunction in DKA patients.[5] This condition arises from renal hypoperfusion due to reduced intravascular volume.

Management of hyperkalemia in DKA patients depends on the severity, symptoms, underlying cause, progression, and response to conservative therapy. In the case of this patient, the condition of AKI improved with the treatment of DKA, particularly with fluid administration.

The primary goal of managing hyperglycemia in DKA patients is to restore normal physiological functions, correct underlying causes, and address metabolic disturbances, including acid-base and electrolyte imbalances. Blood glucose levels should be reduced to a target range of 200–250 mg/dL initially. Insulin therapy is essential to prevent lipolysis and reduce ketogenesis, while careful monitoring of blood glucose is necessary to avoid hypoglycemia. Successful management of DKA is indicated by several clinical parameters, such as blood glucose < 200 mg/dL, bicarbonate levels > 18 mEq/L, and the resolution of ketonemia. Before initiating therapy, it is crucial to assess the patient's airway and breathing. In cases where the patient presents with altered consciousness or coma, endotracheal intubation and mechanical ventilation may be required. Oxygen should be administered via a non-rebreathing mask if indicated. A nasogastric tube should be inserted, and its open end should be removed if the patient appears drowsy or nauseous. Airway, breathing, and consciousness levels must be closely monitored during DKA management.[17] The management of diabetic ketoacidosis (DKA) is guided by the recommendations of the Indonesian Society of Endocrinology (Perkumpulan Endokrinologi Indonesia, PERKENI)[18] **(Figure 1).**

Management of hyperglycemia involves three primary components: fluid therapy, insulin therapy, and correction of electrolyte imbalances. In this context, circulation becomes the second priority following the establishment of adequate oxygenation. Patients with diabetic ketoacidosis (DKA) often present with severe dehydration, which can progress to hypovolemic shock if not promptly addressed. Early and aggressive fluid resuscitation plays a critical role in reducing hyperglycemia, hyperosmolality, and counter-regulatory hormone levels particularly within the first few hours of treatment. This, in turn, reduces insulin resistance. Therefore, insulin therapy is most effective when preceded by appropriate fluid and electrolyte replacement. The primary objective of rapid fluid resuscitation is to restore intravascular volume and improve renal perfusion. Isotonic crystalloids, such as normal saline (0.9% NaCl), are typically the first-line fluids used. In cases of hypovolemic shock, colloid solutions may be considered. Current evidence does not indicate superiority of lactated Ringer's solution over sodium-based solutions. For initial fluid resuscitation, either 0.9% NaCl is recommended, or 0.45% NaCl if serum sodium levels exceed 145 mEq/L.[17,23] To minimize the risk of hyperchloremia associated with the use of normal saline, lactated Ringer's solution may be considered as an alternative. An initial fluid regimen may include the administration of 0.9% NaCl at a rate of 15–20 mL/kg body weight/hour, or approximately 1–1.5 liters during the first hour.[16] In the described case, the rehydration protocol consisted of 1,000 mL of 0.9% NaCl over the first 30 minutes, followed by 500 mL over the next 30 minutes,

Table 2. Diagnosis criteria of acute kidney injury.[24]

Stage	Creatinine Serum	Urine Output
1	Serum creatinine 1.5–1.9 times the baseline or an increase ≥ 0.3 mg/dL	Urine output < 0.5 mL/kg body weight per hour for 6–12 hours
2	Serum creatinine 2.0–2.9 times the baseline	Urine output < 0.5 mL/kg body weight per hour for ≥ 12 hours
3	Serum creatinine 3.0 times the baseline, or an increase in serum creatinine ≥ 4.0 mg/dL, or initiation of renal replacement therapy, or in patients < 18 years, a decrease in eGFR < 35 mL/min/1.73 m²	Urine output < 0.3 mL/kg body weight per hour for ≥ 24 hours, or anuria for ≥ 12 hours



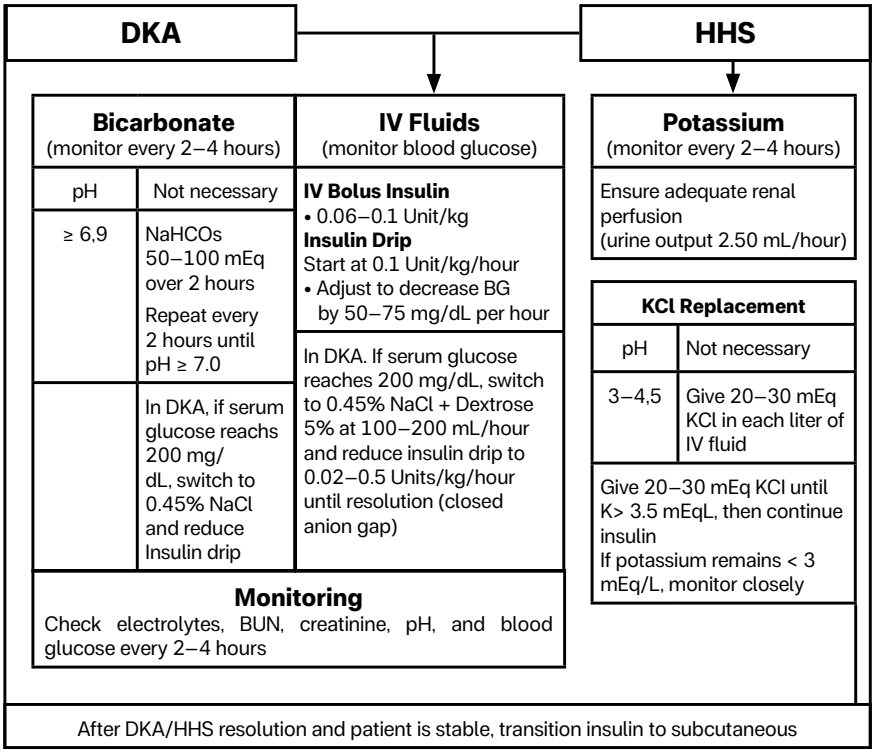



Figure 1. Management of hyperglycemic crisis.[18]
Abbreviations: DKA: Diabetic ketoacidosis; IV: Intravena; HHS: Hyperosmolar hyperglycemic state; BUN: Blood urea nitrogen.

1,000 mL over one hour, 500 mL over the subsequent hour, and finally 250 mL over the next hour. Once the rehydration phase was completed, maintenance fluid therapy was continued with 0.9% NaCl at a rate of 20 drops per minute. Insulin therapy should begin promptly once adequate rehydration is achieved, as this improves insulin efficacy and reduces the risk of hemodynamic instability.[17]

To further correct hyperglycemia, intravenous insulin administration is required, typically initiated one to two hours after the commencement of fluid resuscitation. An initial intravenous bolus of insulin at a dose of 0.1 units/kg body weight is recommended, followed by a continuous infusion at 0.1 units/kg/hour using an insulin pump. Diabetic ketoacidosis (DKA) is considered resolved when the blood glucose level stabilizes below 200 mg/dL, arterial pH exceeds 7.3, and serum bicarbonate reaches ≥ 18 mEq/L. Once these targets are achieved and the patient is able to tolerate oral fluids, a transition to subcutaneous insulin can be initiated. This includes intermediate- or long-acting insulin in combination with short- or rapid-acting insulin formulations.

In the case, intravenous insulin infusion was initiated in the second hour at 4 units/hour. Two hours after admission the blood glucose level was 400 mg/dL. Hourly blood glucose monitoring was performed; three hours after admission, the patient's blood glucose level decreased to 302 mg/dL, the insulin infusion rate was further reduced to 2 units/hour. This infusion rate was maintained as long as the blood glucose levels continued to decline at the targeted rate of 50–75 mg/dL per hour, in accordance with current treatment guidelines.[18] As part of the patient's diabetes management, a diabetic diet providing 1,900 kcal/day was prescribed, alongside subcutaneous administration of insulin glargine at a dose of 28 units every 24 hours. Due to a suspected urinary tract infection, the patient was also treated with cefixime at a dose of 200 mg every 12 hours for 7 days.
Dehydration and osmotic diuresis in diabetic ketoacidosis (DKA) can lead to significant alterations in both intracellular and serum electrolyte balance. Potassium, the primary intracellular cation, plays a vital role in maintaining the electrochemical gradient across cellular membranes. A reduction in intracellular potassium levels may result from several factors, including insulin deficiency, intracellular dehydration, acidosis, and hydrogen ion shifts. Additionally, gastrointestinal losses such as nausea and vomiting may further exacerbate potassium depletion.

During insulin therapy for DKA, correction of acidosis and fluid resuscitation can cause a further decline in serum potassium levels.[18] Insulin plays a critical role in the management of hyperkalemia by promoting intracellular potassium shift. It stimulates Na^+/H^+ antiport activity at the cell membrane, facilitating sodium influx, which in turn activates the Na^+/K^+-ATPase pump. This electrogenic mechanism drives potassium into the intracellular compartment. Therefore, it is essential to assess serum potassium levels prior to initiating insulin therapy. Serum potassium concentration serves as a useful indicator of the overall potassium deficit. General recommendations suggest that potassium supplementation should begin when serum levels are below 5.0–5.5 mEq/L and the patient has adequate renal function, evidenced by the ability to produce urine.[18,23] Potassium is typically administered in the second liter of 0.9% sodium chloride infusion. In cases of severe hypokalemia (< 3.3 mEq/L), insulin administration should be delayed until serum potassium is corrected to ≥ 3.3 mEq/L to avoid life-threatening arrhythmias.[25]

In this case, following fluid resuscitation and insulin therapy, the patient's serum potassium level declined, necessitating potassium replacement; 25 mEq KCl diluted in 500 mL of 0.9% NaCl was administered via intravenous infusion.

Timely and appropriate management significantly improves patient outcomes.[26] The prognosis of patients with DKA can often be determined within the first 72 hours of treatment. Persistent hyperglycemia in DKA may lead to a range of complications, including cerebrovascular accidents (stroke), myocardial infarction, and acute kidney injury.[27]

Throughout the patient's hospitalization, clinical symptoms, vital signs, and blood glucose levels were monitored. Urine output was assessed every six hours to evaluate




renal function and identify early indications for renal replacement therapy. By the third day of treatment, laboratory parameters including serum creatinine, blood urea nitrogen (BUN), and estimated glomerular filtration rate (eGFR) showed improvement, indicating recovery of renal function.

Following hospitalization, the continuation and adjustment of insulin therapy should be carefully considered as part of long-term glycemic management. The initiation of antihyperglycemic agents whether oral medications or insulin should begin at low doses, with gradual titration based on the patient's glycemic response. Combination therapy involving two oral antihyperglycemic agents is often necessary and should include drugs with complementary mechanisms of action. If glycemic targets are not achieved with dual oral therapy, the addition of insulin to the treatment regimen may be warranted. In circumstances where insulin use is clinically contraindicated or not feasible, a triple oral therapy regimen may serve as an alternative strategy. When insulin is combined with oral antihyperglycemic agents, therapy typically begins with basal insulin, which may be either intermediate-acting or long-acting. Intermediate-acting insulin is preferably administered at bedtime, while long-acting insulin may be given in the evening, at bedtime, or in the morning, depending on patient preference and lifestyle. This basal-supported approach has been shown to be effective in achieving glycemic control with relatively low insulin doses. The initial recommended dose for basal insulin in such a regimen ranges from 0.1 to 0.2 units/kg body weight. Subsequent adjustments are guided by fasting blood glucose levels measured on the following day, allowing for individualized titration based on therapeutic response.[17]

In addition to pharmacological interventions, non-pharmacological management plays a crucial role in comprehensive diabetes care. Given the patient's suboptimal lifestyle practices, structured education on lifestyle modification was provided. Recommendations included increasing physical activity, adopting a balanced dietary pattern comprising 45%–65% of total energy intake from high-fiber carbohydrates, 20%–25% from fats, 10%–15% from proteins, and sodium intake limited to <1,500 mg/day and avoiding processed and high-sugar foods and beverages. Furthermore, patients were advised to maintain regular sleep hygiene and implement stress-reduction strategies.[28]

CONCLUSION

A case of diabetic ketoacidosis (DKA) in a 32-year-old male patient with type 2 diabetes mellitus, complicated by stage 1 acute kidney injury (AKI) of prerenal origin, suspected urinary tract infection (UTI), and hypokalemia due to intracellular potassium shift, has been reported. This case highlights the importance of early identification, prevention, and appropriate management of acute metabolic complications in diabetes mellitus. Timely intervention is essential to preserve organ function, prevent further complications, and ultimately improve the quality of life in patients living with diabetes.

DECLARATIONS

Consent for Publication

Written informed consent to publish the patient's clinical information was obtained. All information was presented anonymously to protect the patient's identity.

Funding

This case report received no specific grant or financial support from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

PDAW contributed to the case report's conception and design, data collection, literature review, and manuscript drafting. NPTD and IBAN contributed to the clinical management, interpretation of clinical data, and critical revision of the manuscript. All authors reviewed and approved the final version of the manuscript.

Conflict of Interest

The authors declare no financial or non-financial conflicts of interest related to this case report.

Declaration on the Use of Artificial Intelligence (AI)

Artificial intelligence (AI) tools were used to assist with language editing and manuscript preparation. The authors independently reviewed, verified, and approved all manuscript content and remain fully responsible for its accuracy, interpretation, and final content.

Acknowledgments

Not applicable.

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