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Severe Diabetic Ketoacidosis with Delayed Resolution in Type 2 Diabetes Mellitus: A Case Report from a Resource-Limited Setting

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Abstract

Background: Diabetic ketoacidosis (DKA) is a life-threatening complication of diabetes, with infections and poor treatment adherence as common precipitants. Prolonged resolution is uncommon and suggests severe disease or ongoing precipitating factors.
Case Presentation: A 34-year-old male with poorly controlled type 2 diabetes mellitus (HbA1c 14%) presented with fever, polyuria, vomiting, and biochemical features of severe DKA (random glucose 24.4 mmol/L, bicarbonate 6.9 mmol/L, ketonuria 3+). Initial precipitants included poor medication adherence and malaria. On day 4, he developed urinary symptoms with urinalysis suggestive of infection, though urine culture was sterile.
Management and Outcome: Suspected urinary tract infection was treated with empirical antibiotics. Insulin therapy and electrolyte correction were continued. DKA resolved on day 9 (bicarbonate 23 mmol/L), and he was discharged on day 13.
Conclusion: Multiple or evolving precipitants, including suspected infection, may contribute to delayed recovery in severe DKA.

Keywords: *Diabetic ketoacidosis, type 2 diabetes, precipitants, malaria, infection*

Background

Diabetic ketoacidosis (DKA) is an acute, life-threatening complication of diabetes mellitus, characterized by hyperglycaemia, ketonaemia, and

metabolic acidosis (Umpierrez et al., 2024). Although it occurs more commonly in type 1 diabetes, it is increasingly recognized in individuals with type 2 diabetes mellitus (T2DM), accounting for approximately 10–30% of adult DKA cases (Alali et al., 2025; Dhatariya et al., 2025; Zhong et al., 2018). The incidence of DKA is rising worldwide, likely reflecting the increasing global prevalence of diabetes mellitus (IDF, 2025; Puttanna & Padinjakara, 2014). The number of people living with diabetes has increased from 537 million in 2021 to 589 million in 2025, underscoring the growing burden of acute metabolic complications such as DKA (IDF, 2021; IDF, 2025).

In addition to rising incidence, the most common precipitants of DKA include inadequate insulin therapy and infections (Umpierrez et al., 2024). Respiratory tract infections and urinary tract infections are among the most frequently reported infectious precipitants (Dragila et al., 2023; Shahid et al., 2020). Other important precipitants include myocardial infarction, cerebrovascular events, pulmonary embolism, and pancreatitis.

Comorbidities often prolong hospital stay and complicate recovery in patients with DKA (Alwahbi et al., 2022). The presence of multiple or evolving precipitants may contribute to delayed resolution of metabolic derangements, particularly in severe cases. This case underscores the need for early recognition and prompt management of both precipitants and comorbidities to improve outcomes in severe DKA.

Case Presentation

A 34-year-old male presented to the emergency department with a 2-day history of fever, polyuria, polydipsia, and persistent vomiting. The vomiting was non-bloody and associated with generalized abdominal discomfort. He had been diagnosed with type 2 diabetes mellitus four years prior and had poor glycaemic control due to non-adherence to medications (vildagliptin/metformin 50/1000mg twice daily, glimepiride 2mg once daily, SC insulin glargine 18units nocte).

He consumed tobacco in the form of shisha (hookah) but he had no history of alcohol consumption. He was not known to be hypertensive and had no family history of diabetes. His last clinic visit was one year prior, when his HbA1c was 9.8%. His job required frequent travel, and glucose monitoring was supervised by a relative who was a nurse. He had commenced parenteral antimalarial (intramuscular A-B arteether) and ceftriaxone two days prior to presentation without significant improvement in symptoms hence he presented to the emergency unit.

On presentation, he had a low-grade fever (37.5°C) and he was moderately dehydrated. His pulse rate was 112 beats/min, blood pressure 122/66 mmHg, and respiratory rate of 24 breaths/min. He was fully conscious (GCS 15) and his BMI was 27.4 kg/m2. The remainder of the physical examination was unremarkable.

Laboratory evaluation revealed severe DKA, with a random blood glucose of 24.4 mmol/L, bicarbonate of 6.9 mmol/L, and ketonuria (3+). His anion gap was 32.7 mmol/L and the calculated serum osmolality was 279 mOsm/kg. There was leucocytosis with neutrophilia, as well as a positive qualitative C-reactive protein (CRP) result (Table 1). A blood film for malaria was positive for *Plasmodium falciparum*, although parasite density was not quantified. Serum ketone quantification, blood gas analysis, and serum lactate measurements were not available due to resource limitations.

Table 1. Significant findings of investigations done on admission

Chemistry		**Haematology**	
Sodium	124mmol/L	Total WBC	16,300/uL
Potassium	4.6mmol/L	Granulocytes	79%
Bicarbonate	6.9mmol/L	Hb	15g/dL
Blood glucose	24.4mmol/L	**Microbiology**	
HbA1c	14%	Blood film	1+ for P. falciparum
Total Cholesterol	6.3mmol/L	Urine culture	No growth
LDL-C	4.9mmol/L	Blood culture	No growth
Proteinuria	2+		
Glycosuria	2+		
Ketonuria	3+		
Haematuria	Negative		
Qualitative CRP	Positive		

He was diagnosed with severe DKA precipitated by poor medication adherence and malaria, with a suspected bacterial infection. He was managed with intravenous normal saline at a rate of 1 L/hour for the first 2 hours, followed by 250 mL/hour, while urine output and hydration status were closely monitored. Insulin therapy was commenced after 1 hour of rehydration as a continuous intravenous infusion at 0.1 units/kg/hour. Potassium chloride (20 mmol) was added to each litre of normal saline administered, as his serum potassium level was within the normal range.

He was also commenced on intravenous artesunate and broad-spectrum antibiotics while awaiting culture results. Supportive measures, including antiemetics and antipyretics, were instituted. Twelve hours after initiation of treatment, his blood glucose level reduced to 11.2 mmol/L, and the fluid regimen was transitioned to glucose-potassium-insulin (GKI) infusion according to protocol. His vomiting and abdominal pain improved, and he was transitioned to a basal-bolus insulin regimen on the third day of admission (16units of subcutaneous regular insulin 30 minutes before meals and 24 units of insulin glargine at bedtime).

However, on the fourth day of admission, he developed haematuria, recurrence of persistent vomiting, generalized abdominal pain, with associated urinary urgency. There was no bleeding from any other site, and coagulation studies were normal. He had no history suggestive of renal colic or urinary tract calculi. Repeat urinalysis showed blood 3+, ketones 3+, protein 3+, and leucocytes+, although urine culture yielded no bacterial growth. This negative culture result may have been influenced by prior antibiotic exposure before sample collection. His blood glucose levels were between 12 and 16 mmol/L. Repeat laboratory investigations revealed haemoglobin of 11.5 g/dL, bicarbonate of 6 mmol/L, potassium of 3.5 mmol/L, mild elevation in serum urea (7 mmol/L), and normal serum creatinine levels. Abdominal ultrasonography showed essentially normal findings.

A suspected urinary tract infection was considered, and antibiotic therapy was changed to intravenous levofloxacin based on local antimicrobial sensitivity patterns. Intravenous fluids and continuous insulin infusion were recommenced until blood glucose levels were below 14 mmol/L, after which GKI infusion was resumed (with 32 units of soluble insulin and 10 mmol of intravenous potassium chloride added to 500 mL of dextrose saline to run over 4 hours).

Despite ongoing management, glycaemic control remained unstable, with marked fluctuations in blood glucose levels (Figure 1), necessitating frequent insulin dose adjustments. By the eighth day of admission, the bicarbonate level had improved to 17 mmol/L and ketonuria had reduced to 1+. He continued to improve clinically, and by the ninth day of admission he met the resolution criteria for DKA, with a bicarbonate level of 23 mmol/L, blood glucose level of 10.8 mmol/L, and ketonuria of 1+. At this point, a basal-bolus insulin regimen was commenced with a 2-hour overlap of GKI infusion. He was discharged after 13 days of hospitalization on premixed 70/30 insulin twice daily and oral

vildagliptin/metformin 50/1000 mg twice daily. At follow-up one week later, he had good glycaemic control and was in good clinical condition.

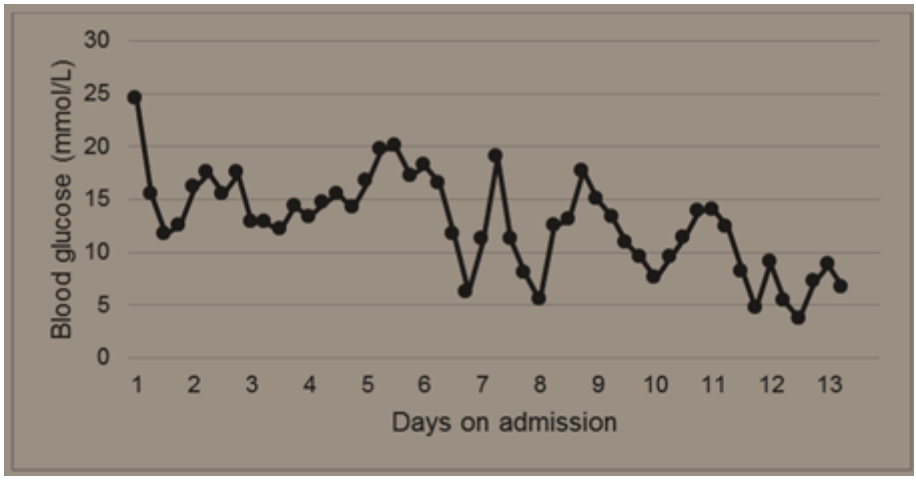

Figure 1. Daily blood glucose trend during admission

Table 2. Timeline of Clinical Events

DAY	CLINICAL EVENT
Day 1	Admission with severe DKA; intravenous fluids, insulin infusion, artesunate, and antibiotics commenced
Day 3	Improvement in symptoms; transitioned to basal-bolus insulin regimen
Day 4	Suspected UTI; IV levofloxacin commenced, insulin infusion recommenced
Day 8	Bicarbonate improved to 17 mmol/L, ketonuria reduced
Day 9	DKA resolved, transitioned back to basal-bolus insulin
Day 13	Discharged home in stable condition

Discussion

Diabetic ketoacidosis results from severe insulin deficiency combined with increased levels of counter-regulatory hormones, including glucagon, cortisol, catecholamines, and growth hormone. These hormonal changes lead to increased hepatic gluconeogenesis, glycogenolysis, and lipolysis, ultimately resulting in hyperglycaemia, ketone body production, and metabolic acidosis (Umpierrez et al., 2024; Dhatariya et al., 2025). The commonest precipitants of DKA include inadequate insulin therapy and infections. Inadequate insulin therapy may result from poor medication adherence, interruption of insulin administration, or suboptimal treatment regimens (Dragila et al., 2023; Newton & Raskin, 2004).

In the index case, two potential precipitants were identified on admission: poor medication adherence and malaria. However, the clinical significance of malaria as a precipitating factor remains uncertain, as the degree of parasitaemia was not quantified and symptoms may have overlapped with those of DKA. In malaria-endemic settings, asymptomatic parasitaemia is not uncommon, and its contribution to acute metabolic decompensation may therefore be difficult to establish with certainty.

A further possible contributing factor was the suspected bacterial infection, supported by neutrophilia and a positive CRP on admission. It is possible that an underlying bacterial infection was present at admission but became clinically apparent only later during hospitalization. However, this remains speculative in the absence of microbiological confirmation, particularly in the context of prior antibiotic exposure. During admission the patient developed haematuria, urinary urgency, pyuria, persistent ketonuria, and worsening metabolic abnormalities, raising suspicion for urinary tract infection. Although urine culture was negative, prior antibiotic use before sample collection may have reduced the likelihood of isolating an organism. This case underscores the importance of vigilant monitoring for infections during DKA management, particularly in resource-limited settings where diagnostic uncertainty may persist despite negative culture results.

A diagnosis of DKA is made in the presence of blood glucose ≥ 11.1 mmol/L or prior history of diabetes, β-Hydroxybutyrate concentration ≥3.0 mmol/L or urine ketones ≥ 2+, and metabolic acidosis i.e. blood pH <7.3 and/or bicarbonate <18 mmol/L (Umpierrez et al., 2024). All three criteria were met in the index case and the markedly low bicarbonate level of 6.9 mmol/L fulfilled criteria for severe DKA. Serum ketone quantification and blood gas analysis were not available because of resource limitations. However, urine ketones and serum bicarbonate levels were used to guide diagnosis and monitoring.

A key feature of this case is the markedly prolonged time to resolution of DKA. Using standard criteria (bicarbonate >18 mmol/L), resolution occurred on the ninth day of admission, compared to the typical duration of 18–24 hours reported in the literature (Alwahbi et al., 2022; Sabry et al., 2024). This delay is likely multifactorial. The patient presented with severe DKA, evidenced by a markedly low bicarbonate level (6.9 mmol/L), which is known to be associated with slower metabolic recovery (Sabry et al., 2024). In addition, the presence of multiple or evolving precipitants, including suspected urinary tract

infection, may have contributed to persistent metabolic stress (Alwahbi et al., 2022).

The observed oscillations in blood glucose in this case may have also contributed to delayed resolution of the DKA. These fluctuations were likely multifactorial, including infection-related insulin resistance, variability in insulin administration and changes in nutritional intake. Infection is known to exacerbate hyperglycaemia by increasing insulin resistance and impairing glucose utilization (Umpierrez et al., 2024). In addition, dynamic adjustments in insulin therapy during DKA management may have contributed to glycaemic variability.

Although current guidelines allow cautious use of bicarbonate in profound acidosis, it was not administered in this patient (Umpierrez et al., 2024). Recovery was ultimately achieved with standard therapy consisting of fluids, insulin, electrolyte replacement, and treatment of precipitating factors. The administration of bicarbonate in severe DKA remains controversial, given the potential risks including hypokalaemia, cerebral oedema, reduced tissue oxygen delivery, and paradoxical worsening of intracellular acidosis (Patel et al., 2018; Tran et al., 2017). The absence of bicarbonate therapy in this patient underscores the effectiveness of standard management with fluids and insulin, even in severe cases, although recovery may be prolonged.

Another consideration in this case is the possibility of ketosis-prone T2DM (KPD), a subtype of diabetes characterized by presentation with DKA in individuals who lack the typical features of autoimmune type 1 diabetes. KPD is more commonly reported in individuals of African descent and is often associated with reversible β-cell dysfunction and subsequent recovery of insulin secretion (Umpierrez et al., 2024). The index patient had features compatible with T2DM, including overweight status and a prior history of oral hypoglycaemic therapy. Although further evaluation with autoantibody testing and C-peptide levels was not performed due to resource constraints, such classification may have implications for long-term management and prognosis.

The management of severe DKA in resource-limited settings presents several challenges. Important investigations such as serum β-hydroxybutyrate levels, arterial or venous blood gas analysis, and lactate levels may not be readily available. These constraints may affect both diagnostic precision and the monitoring of treatment response, potentially contributing to delayed recognition of complications. In such settings, clinicians may need to rely on clinical assessment, urine ketones,

serum bicarbonate levels, and serial electrolyte monitoring to guide management.

Conclusion

This case describes severe DKA in a patient with poorly controlled T2DM precipitated by poor medication adherence, malaria, and suspected bacterial infection. The case illustrates how infections can significantly prolong recovery in severe DKA, underscoring the importance of early detection and aggressive management of comorbidities. It also highlights important diagnostic and therapeutic challenges encountered in resource-limited settings where advanced investigations may not be readily available. Early recognition and prompt management of both precipitants and complications are essential to improve outcomes in severe DKA.

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