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A 16-Year-Old Female with Diabetic Ketoacidosis, Diabetes Mellitus, Sepsis, and Constipation: A Case Report

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Abstract

Diabetic ketoacidosis (DKA) is a serious and potentially fatal complication of hyperglycemia, with a reported mortality rate of 2–5% depending on age. It commonly occurs in patients with uncontrolled insulin-treated diabetes mellitus (DM), especially when accompanied by severe underlying illness. Infection and insulin withdrawal are the two main triggers of DKA, with bacterial infections accounting for approximately 50% of cases, including urinary tract infections, pneumonia, and gastrointestinal conditions such as constipation. Constipation is more prevalent in diabetic patients with colonic neuropathy and may further complicate clinical outcomes. This case report presents a 16-year-old female with suspected new-onset type 1 DM who was admitted with DKA complicated by severe constipation and sepsis. The case highlights the complex interaction between poor glycemic control, infection, and systemic inflammatory responses in DKA. Diabetes mellitus can alter immune responses, infection patterns, and sepsis outcomes, contributing to increased morbidity and mortality. Diagnosing infection in patients with DKA remains challenging because both conditions share overlapping clinical and laboratory features, including fever, leukocytosis, and systemic inflammation. Early identification of bacterial infection is essential to ensure prompt and appropriate treatment while avoiding unnecessary antibiotic use that may contribute to antimicrobial resistance. This case report emphasizes the importance of early diagnosis of concurrent infection in patients with DKA to enable prompt management and improve clinical outcomes.

1. Introduction

T1DM (Type 1 diabetes mellitus) is an immune-mediated destruction of insulin-producing pancreatic β-cells, leading to insulin deficiency. Individuals with type 1 diabetes require lifelong exogenous insulin replacement. Childhood-onset T1DM tends to present with more severe clinical features, including symptomatic severe hyperglycemia or diabetic ketoacidosis (DKA). DKA is a very serious condition for people with type 1 or 2 diabetes [1].

DKA is a common complication of diabetes that is associated with patient morbidity and mortality. Although there have been many advances in the development of monitoring methods, disease awareness, and hypoglycemic drugs over the past ten years, there has been a consistent increase in mortality rates due to DKA as well as hospitalizations of patients. The mortality rate due to DKA has been reported to be 2–5% in recent studies. Although the annual incidence of DKA is estimated to be between 4.6 and 8 episodes per 1,000 diabetic patients admitted in population-based

studies in the United States, this is not yet known in developing countries. According to the Centers for Disease Control and Prevention (CDC) – National Diabetes Surveillance Program, the number of patients discharged from hospitals with a primary diagnosis of DKA increased from approximately 80,000 in 1988 to approximately 140,000 in 2009. However, the mortality rate due to DKA has significantly decreased over the last 20 years, from 7.96% to 0.67% as reported, but in developing countries it remains high [2].

DKA mainly occurs in patients with type 1 DM. However, patients with type 2 DM can also experience DKA in conditions of severe stress or disease with metabolic decompensation. The most common triggers of DKA are discontinuation of insulin therapy and infection. A study conducted in China reported that DKA is generally caused by infection (40.1%), followed by discontinuation of hypoglycemic drugs (16.8%) and idiopathic causes (36.9%). Infection (the most common cause of DKA) is directly associated with the risk of death.

Bacterial infections that commonly lead to DKA and account for about 50% of all DKA patients include constipation, urinary tract infections, and pneumonia. Bacterial infections in DKA increase the length of hospital stay and mortality rates. These infections must be detected early and treated with appropriate antibiotics to improve outcomes in individuals with DKA. Diagnosis of infection is usually based on symptoms and supporting test results. Some supporting test results take up to 48 hours to become available. Early diagnosis of DKA can be challenging because its specific symptoms resemble those of infection, such as fever, elevated leukocyte count, and elevated neutrophil percentage. This can lead to excessive antibiotic use and the development of antibiotic resistance. Therefore, time and effort are needed to establish an effective and rapid diagnosis for early detection of infection to prevent progression to life-threatening conditions [2, 3].

The American Diabetes Association defines diagnostic criteria: hyperglycemia (i.e., plasma glucose > 250 mg/dL) and high anion gap metabolic acidosis (i.e., arterial pH < 7.30, serum bicarbonate < 18 mEq/L, and an anion gap > 10) with positive serum or urine ketones (ADA) [4].

The symptoms of DKA are nonspecific, with many diseases that mimic the presentation with additional underlying severe illness, such as constipation with sepsis or another severe infection. Constipation is more common in those with diabetes and colonic neuropathy. The etiology of constipation in diabetes is multifactorial. Autonomic neuropathy is thought to be important; constipation is more common in those with diabetes and autonomic impairment.

In the next stage, untreated constipation can lead to bacterial colonization and cause severe infections. Infection is the most common predisposing factor (range 32-60%) for the condition of DKA, hyperglycemic crises, and hyperosmolar hyperglycemic state. This condition can trigger unregulated body responses, leading to life-threatening organ dysfunction or sepsis. The form of sepsis characterized by circulatory and cellular metabolic abnormalities is called septic shock, and its occurrence can increase mortality risk [4].

This case report describes an adolescent with uncontrolled T1DM who presented with severe DKA complicated by sepsis. The report highlights the diagnostic challenges, precipitating factors, clinical course, and management of DKA associated with severe infection, with the aim of emphasizing the importance of early recognition and prompt treatment to improve patient outcomes.

2. Cases

We present the case of a 16-year-old female with complaints of abdominal pain, constipation for 2 weeks, and fever. But lasting 6 hours before going to the hospital, she had progressive weakness and also had tachypnea with shortness of breath. In her medical history, she had unrecognized polyuria, polydipsia, polyphagia, and weight loss. There was no family history of diabetes mellitus, but she is obese with a BMI of 30 and was just diagnosed with type 1 diabetes three months before, but she never used her insulin.

She presented to the hospital with a Glasgow Coma Scale score that dropped from 13 to 3 in only 30 minutes. She had tachypnea with Kussmaul respirations, sinus tachycardia, blood pressure (BP) 99/52 mmHg, body temperature 39.8°C, and cold extremities (capillary refill time > 2 seconds). Clinical examination revealed severe dehydration, respiratory distress with a respiratory rate of 40/min. The abdomen is bloated and distended with meteorism. Fever can be a clinical marker of DKA in the setting of infection. Some studies report higher body temperatures in patients with DKA accompanied by infection; body temperatures in patients without bacterial infection have ranged from 32.9°C to 38.7°C since admission. Thermoregulatory function may be impaired in patients with diabetes [5, 6].

Arterial blood gas analysis results are presented in Table 1, demonstrating severe metabolic acidosis with a pH of 6.974. Blood test results shown in Table 2 revealed hyperglycemia (543 mg/dL), leukocytosis (28,100/mm^3),

Table 1. Arterial blood gas analysis.

Test	Result
pH	6.974
pCO2 (mmHg)	15.1
HCO3 (mmol/L)	52
BE (mmol/L)	-28.3

Table 2. Blood test results.

Test	Result
Hb	15.0 g/dL
Leukocyte	28.000/mm3
Hematocrit	47.50%
Thrombocyte	387.000/mm3
Erythrocyte	-28.3
Eosinophil	0
Neutrophil	83.2
Lymphocyte	4.8

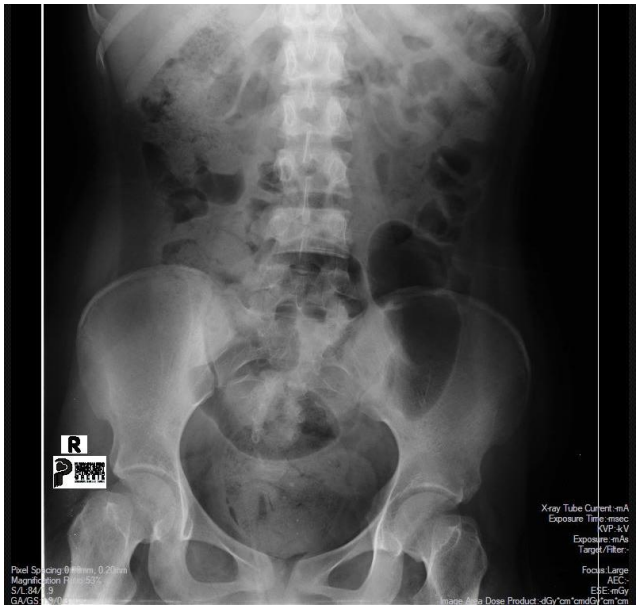

Figure 1. BOF showed increased intestinal gas and intestinal obstruction with prominent fecal material.

ketonuria (3+), proteinuria (2+), and hematuria (2+). Leukocyte Count in DKA with Infection. DKA increases systemic inflammation and oxidative stress. Acidosis also causes downregulation of platelet function and increases neutrophil activity. In addition, there is activation of T-lymphocyte cells. The presence of infection also creates an inflammatory environment with cytokine production and oxidative stress. Several studies have found changes in white blood cell counts in DKA. A study by Karavanaki observed that the blood leukocyte count was higher in DKA patients upon arrival at the hospital, namely 15.2 × 10^2/mm3. Other studies noted that leukocyte levels were higher in DKA patients, and that those with infection had significantly higher blood leukocyte levels (16,910/mm3) than DKA patients without infection (10,310/mm3). Several studies by Huang et al. found that the leukocyte count threshold for DKA with infection was >10 × 10^9/L [3].

Among white blood cell fractions, attention in recent years has been focused on NLR. There is a correlation between inflammation-related diseases such as diabetes mellitus, neoplasms, obesity, hypertension, and atherosclerosis. NLR reflects the function of adaptive and innate immune responses. Several studies have found an increased NLR in patients with DKA. In a study, the median NLR in patients without ketoacidosis was 1.11 (0.80–1.80). In mild DKA cases, the value was 1.58 (1.17–1.93). In moderate DKA cases, the value was 3.71 (1.98–4.85). In severe cases, the median NLR was 5.77 (4.04–9.63). NLR may be a potential predictor of the severity of systemic inflammation in DKA. In DKA, an increased NLR indicates an increase in neutrophil count and a decrease in lymphocyte count. This decrease in lymphocyte count may be due to DNA damage in peripheral lymphocytes induced by reactive oxygen species generated by acute hyperglycemia. Similarly, sepsis causes a significant increase in NLR by stimulating lymphocyte apoptosis and increasing neutrophil counts.

BOF showed in Figure 1 that increased intestinal gas and intestinal obstruction with prominent fecal material. Clinicians typically reserve X-ray or computed tomography imaging for patients who fail to improve after initial therapy or who exhibit signs that raise concern for complications. Abdominal radiography or CT scanning can help confirm excessive stool buildup in the intestines, colonic dilation, or possible ileus. Acute colonic pseudo-obstruction is identified as a diagnosis of exclusion, with imaging often demonstrating significant colonic enlargement, especially in the cecum and right colon. Healthcare providers are increasingly using bedside point-of-care ultrasonography to evaluate gastrointestinal function. This technique may detect decreased or absent peristalsis, enlarged bowel loops (> 5 cm), thickened mucosal layers, and reduced perfusion of the intestinal wall—all findings that suggest fecal stasis related to constipation.

Based on the patient's clinical presentation, physical examination, and supporting investigations, we suspect that the patient is experiencing DKA accompanied by sepsis, with a rapid assessment supported by the qSOFA score (The qSOFA (quick SOFA) score is a bedside prompt used outside the ICU to rapidly identify patients with suspected infection at high risk for poor outcomes, including mortality. A score indicates high risk. It uses three criteria (1 point each): respiratory rate of 22/min, altered mental status (GCS < 15), and systolic blood pressure of 100 mmHg. Based on the patient's history, we suspect that the source of infection is constipation. As is

well known, poorly controlled diabetes mellitus increases the risk of constipation.

In this case, the patient presented at a relatively late stage, with comorbidities that had not been properly addressed, and was still in denial regarding a diagnosis of diabetes mellitus made three months earlier at a young age, which led to not using insulin therapy.

The treatment we administered followed the guidelines of the American Diabetes Association. Initial intravenous fluid resuscitation aimed to restore effective circulating intravascular volume, using isotonic saline or balanced crystalloid solutions at a rate of 500–1,000 mL/hour during the first 2–4 hours. Treatment protocols recommend an initial bolus of short-acting insulin (0.1 units/kg, given intravenously or intramuscularly), followed by a continuous infusion at 0.1 units/kg/hour. Vital signs were continuously monitored. As antibiotic therapy, we administered ceftriaxone (1 g every 12 hours).

During six days of intensive care unit (ICU) treatment, the patient's condition progressively deteriorated and did not respond to therapy according to established guidelines, ultimately resulting in the patient's death.

3. Discussions

3.1. Mechanism of Constipation in DM

The effects of myopathy caused by Diabetes mellitus, colon neuropathy, and mucosal absorption can alter the colon's sensory and motor responses. Diabetes mellitus alters the intestinal flora and disrupts the release of neurotransmitters and local hormones. Constipation in DM patients is associated with intracellular signaling pathways in enterocytes, increased oxidative stress, and impaired contraction and relaxation responses in circular muscle strips of the colon [7]. Microbial and inflammatory changes associated with DM can trigger and release inflammatory cytokines [e.g., interleukin (IL)-1α, interferon (IFN)-γ, IL-4, IL-18] that target the intestinal epithelial compartment and enteric neurons, and promote local inflammation. It has also been suggested that the autoimmune response in T1D (type 1 diabetes) may involve the intestine and the neurons, leading to cytokine production, inflammatory responses, and tissue degeneration [8, 9].

A decrease in intestinal contractility has various causes. A reduction in interstitial connective tissue cells (ICCs) in the proximal colon can impair contractility; cholinergic receptors and neuronal nitric oxide synthase play important roles in colon dysfunction associated with diabetes mellitus [10].

3.2. Intestinal Bacterial Changes in Diabetes Mellitus Patients with Constipation

Certain imbalances in the gut microbiota contribute to insulin resistance and are associated with the development of diabetes. Diabetes alters the gut flora, disrupts the release of local neurotransmitters and hormones, and affects the permeability and function of the intestinal barrier, as well as gastrointestinal motility. Indeed, differences have been identified in the blood microbiome and gut bacteria in patients with DM, suggesting that bacteria may enter the bloodstream from the gut. Reduced insulin sensitivity and elevated levels of branched-chain amino acids (BCAAs) in the blood of people with insulin resistance. Importantly, elevated blood BCAA levels are associated with specific changes in body composition and function.

Type 1 diabetes was associated with transfer to the phylum Bacteroidetes. At the genus level, the proportion of lactic acid production increased, and bifidobacteria and lactobacilli were found late in the progression of T1D. T1D increases lactate levels in the cecum and decreases butyrate levels in the cecum compared to the healthy control group. It has been suggested that an imbalance in SCFA-producing bacteria affects intestinal permeability, leading to T1D. Brown et al. describe the importance of butyrate in intestinal permeability in T1D. Short-chain fatty acids (SCFAs), particularly butyric acid, and the bacteria that produce them are critical in the management of constipation and diabetes.

To investigate whether improving gut dysbiosis through lifestyle interventions or probiotic management can reduce circulating inflammatory marker levels and bacterial translocation rates and improve glycemic control.

From the anamnesis and other examinations, the patient had a history of uncontrolled T1DM and constipation. Laboratory results revealed high blood glucose and positive urine ketones (3+). The American Diabetes Association defines DKA as blood glucose >250 mg/dL; plasma HCO3 <18 mEq/L; plasma pH <7.30; increased anion gap, and the presence of ketone bodies in the blood and urine. This patient was diagnosed with DKA based on the results of a blood gas analysis, which showed severe metabolic acidosis.

The most serious and life-threatening hyperglycemic emergencies are DKA and hyperglycemic hyperosmolar state (HHS). DKA and HHS commonly occur in patients with both type 1 and type 2 DM. DKA is most frequent in young adults with type 1 DM. Infection is a common cause; according to a recent report from a safety-net hospital in Atlanta, 56% of patients did not use insulin

during the first episode of DKA, and 78% had multiple episodes of DKA. Other potential triggering factors include infection (14%) and non-infectious causes (4%). Nevertheless, non-adherence to insulin therapy is the primary triggering factor for DKA in young adult patients with type 1 DM in the urban population of the United States. In diabetic patients, infections are frequently encountered because hyperglycemia causes immune system dysfunction (e.g., impaired neutrophil function, suppression of the antioxidant system, and humoral immunity), micro- and macro-angiopathy, neuropathy, decreased antibacterial activity in urine, and gastrointestinal and urinary dysmotility, as well as a greater number of medical interventions [11, 12].

T1DM is caused by an autoimmune process that destroys insulin-producing pancreatic β-cells. Individuals with type 1 diabetes require lifelong exogenous insulin replacement. The presence of hyperglycemia, metabolic acidosis, and ketosis defines DKA. Fluid resuscitation and maintenance, insulin therapy, electrolyte replacement, and supportive care are the mainstays of DKA management. Management of this case followed the 2024 American Diabetes Association guidelines [13].

Initial intravenous fluid resuscitation aimed to restore effective circulating intravascular volume, using isotonic saline or balanced crystalloid solutions at a rate of 500–1,000 mL/h during the first 2–4 hours. Treatment protocols recommend the initial administration of a short-acting insulin bolus (0.1 units/kg, intravenously or intramuscularly), followed by 0.1 units/kg/hour. Vital signs were monitored throughout. As an antibiotic, we used ceftriaxone (1 g every 12 hours).

Upon arrival, the patient was transferred to the intensive care unit (ICU) and began receiving supportive therapy. This included intravenous fluids to maintain hydration, antibiotics, and pump-assisted NR. Additionally, the patient's condition was closely monitored. Regrettably, despite all efforts, there were no signs of improvement, and the patient's condition continued to deteriorate, ultimately resulting in her demise after approximately 6 days of treatment. DKA remains a life-threatening but preventable complication of diabetes.

4. Conclusions

This case highlights DKA as a severe and life-threatening complication of uncontrolled insulin-dependent diabetes mellitus, particularly when accompanied by infection. The case was diagnostically challenging because the patient initially presented with severe constipation and suspected intestinal obstruction before being diagnosed with DKA complicated by infection, with constipation considered a potential infectious trigger. In patients with diabetes mellitus, constipation is associated with oxidative stress, inflammatory pathways, and impaired colonic motility, all of which may contribute to systemic inflammation. During hospitalization, the patient's condition progressively worsened, including a decline in consciousness, and despite intensive multidisciplinary management, the patient died after approximately six days of treatment. This case underscores the importance of early recognition and prompt management of DKA in the context of suspected infection, as the clinical manifestations and inflammatory biomarkers of DKA often overlap with those of bacterial infection. Biomarkers such as serum lactate, white blood cell count, neutrophil-to-lymphocyte ratio, and inflammatory cytokines may help distinguish DKA with infection from DKA without infection in the emergency setting, enabling more appropriate antibiotic use and reducing the risk of antimicrobial resistance. Early diagnosis and implementation of evidence-based management strategies, including the Surviving Sepsis Campaign hour-1 bundle when sepsis is suspected, are essential to improve patient outcomes. Further research is needed to identify more rapid and accurate molecular markers for detecting infection in patients with DKA.

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