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SEVERE DIABETIC KETOACIDOSIS AS THE INITIAL PRESENTATION OF A CHILD WITH METABOLIC SYNDROME: DIAGNOSTIC AND MANAGEMENT CHALLENGES

Pamela Felycia Ardhinata[1]*, Samuel Raditya Wibawa[2]
[1]General Physician, Panti Wilasa Dr. Cipto Hospital, Jl. Dokter Cipto No.50, Bugangan, Semarang Timur, Semarang, Jawa Tengah 50123, Indonesia
[2]Pediatrician, Panti Wilasa Dr. Cipto Hospital, Jl. Dokter Cipto No.50, Bugangan, Semarang Timur, Semarang, Jawa Tengah 50123, Indonesia
*pamelafelyciaa@gmail.com

ABSTRACT
Diabetic ketoacidosis (DKA) is a potentially life-threatening condition that can happen as a complication of diabetes mellitus (DM) or as one of the first manifestations of type 2 DM. DKA treatment involves the use of fluids to treat the patient's dehydration and the administration of intravenous insulin to stop the production of ketones in the body. The two-bag system allows for the adjustment of the rate of administration of glucose and insulin separately. To describe the management of severe diabetic ketoacidosis using high-dose insulin and a two-bag system in a child with suspected ketosis-prone type 2 diabetes mellitus. This case report describes a 12-year-old boy who presented to the Emergency Department of Panti Wilasa Dr. Cipto Hospital, Semarang, with severe diabetic ketoacidosis and suspected ketosis-prone type 2 diabetes mellitus. Clinical and laboratory data, treatment, and outcomes were retrospectively collected from medical records and descriptively reviewed. A 12-year-old boy presented with symptoms of vomiting, dehydration and Kussmaul breathing. Physical examination revealed obesity (BMI 34.16 kg/m^2), abdominal obesity, hypertension and acanthosis nigricans. Blood tests revealed blood glucose levels of 459 mg/dL, HbA1c levels of 10.3%, ketonuria, a pH level of 6.87 and HCO_3^- levels of 1.1 mmol/L, showing a diagnosis of diabetic ketoacidosis with suspected ketosis-prone Type 2 diabetes mellitus. The patient was treated with fluid replacement, continuous intravenous insulin at doses up to 5 IU/hour, antibiotics and a two-bag system. The patient made a full recovery without any complications and was started on basal-bolus insulin therapy. In this case, a child who was severely obese and had marked insulin resistance required a relatively high dose of insulin to manage the severe DKA. However, by carefully monitoring the patient's blood glucose, electrolytes, fluid balance and neurological status, the high-dose insulin therapy along with a two-bag system was successfully implemented without any complications such as cerebral oedema.

Keywords: diabetic ketoacidosis; high dose insulin; metabolic syndrome; two-bag system; type 2 diabetes mellitus

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INTRODUCTION

Diabetic ketoacidosis (DKA) in children can also occur in type 2 diabetes mellitus, especially when insulin levels are unable to meet the body's metabolic needs, such as during metabolic stress that increases insulin requirements (Wolfsdorf et al., 2022; Copeland et al., 2013). DKA is characterized by hyperglycemia, ketosis, and metabolic acidosis that occur due to increased production of ketone bodies as a result of increased lipolysis, which becomes an alternative energy source when glucose cannot be used effectively as the main energy source by cells (Agarwal, 2019; Wolfsdorf et al., 2022; American Diabetes Association, 2001). This condition is an acute, life-threatening metabolic complication (Abulebda et al., 2021; Wolfsdorf et al., 2022) and remains a major cause of morbidity and hospitalization in pediatric patients, especially in cases with previously undetected diabetes (Wolfsdorf et al., 2022; Nadeau et al., 2016).

Based on the criteria set by the International Society for Pediatric and Adolescent Diabetes (ISPAD), the diagnosis of DKA is established when there is hyperglycemia with a blood glucose level >200 mg/dL (11 mmol/L), metabolic acidosis characterized by venous pH <7.3 or serum

bicarbonate (HCO_3^-) level <15 mmol/L, and ketonemia indicated by a blood beta-hydroxybutyrate level >3 mmol/L or moderate to large amounts of ketonuria (Wolfsdorf et al., 2022). Based on the severity of metabolic acidosis, DKA is classified into three categories, namely mild degree characterized by venous pH 7.2–7.3 and HCO_3^- <15 mmol/L, moderate degree characterized by venous pH 7.1–7.2 and HCO_3^- <10 mmol/L, and severe degree characterized by venous pH <7.1 and HCO_3^- <5 mmol/L (Wolfsdorf et al., 2022; Indonesian Pediatric Association, 2018). The more severe the degree of metabolic acidosis that occurs, the higher the risk of life-threatening complications, such as severe dehydration, electrolyte disturbances, cerebral edema, decreased consciousness, and even death (Agarwal, 2019; Wolfsdorf et al., 2022). Cerebral edema is responsible for 60–90% of deaths from DKA, with mortality due to cerebral edema reported at approximately 21–24% (Agarwal, 2019).

Globally, the Asian region is known as an important center of the increasing trend of early-onset type 2 diabetes, with many cases occurring in developing countries, which is associated with obesity and severe insulin resistance (International Diabetes Federation, 2021; Reinehr, 2013). Children and adolescents with obesity and severe insulin resistance are at increased risk of developing type 2 diabetes due to progressive metabolic dysfunction (Nadeau et al., 2016; Weiss et al., 2013). A retrospective study showed that approximately 13% of patients aged 9–18 years who presented with DKA were known to have type 2 diabetes (Sapru et al., 2005). In Indonesia, the Indonesian Pediatric Association has reported an increasing burden of diabetes in children and emphasized the importance of recognizing diabetic ketoacidosis and other acute metabolic complications at initial diagnosis (Indonesian Pediatric Association, 2018, 2023). Studies have shown that approximately 5–6% to 25% of children with type 2 diabetes mellitus may present with DKA, a clinical phenotype that is often referred to as ketosis-prone type 2 diabetes (Copeland et al., 2013; Wang et al., 2025).

In children with obesity and severe insulin resistance, pancreatic β-cell dysfunction may result in relative insulin deficiency. During physiological stress, increased insulin requirements may exceed the available insulin secretory capacity, promoting ketogenesis and potentially leading to DKA (Nadeau et al., 2016; Copeland et al., 2013; Wang et al., 2025). The combination of metabolic syndrome, type 2 diabetes, and DKA in children is still relatively rarely reported in daily clinical practice. Furthermore, the clinical presentation of DKA in children is often nonspecific, which can mimic other conditions and increase the risk of delayed diagnosis and management (Wolfsdorf et al., 2022). Therefore, clinical vigilance is essential in recognizing this condition early.

This case report aims to provide a comprehensive description of the clinical presentation, diagnostic evaluation, and management of severe diabetic ketoacidosis (DKA) in a child with suspected ketosis-prone type 2 diabetes mellitus (KPD-T2DM). Particular emphasis is placed on the use of high-dose insulin therapy and a two-bag fluid system, including the rationale for treatment adjustments, monitoring of clinical and biochemical parameters, response to therapy, potential complications, and short-term clinical outcomes. This case also highlights the clinical challenges and considerations involved in managing severe DKA in pediatric patients with a suspected atypical diabetes phenotype.

METHOD

This case report involved one 12-year-old boy who presented to the Emergency Department of Panti Wilasa Dr. Cipto Hospital, Semarang, with severe diabetic ketoacidosis and suspected ketosis-prone type 2 diabetes mellitus with features of metabolic syndrome. We collected data retrospectively from the patient's medical records, including demographic characteristics, clinical symptoms, physical examination findings, laboratory investigations, treatment, and clinical outcomes. The data were reviewed and analyzed descriptively to assess the diagnostic challenges and management of severe diabetic ketoacidosis, particularly the use of high-dose intravenous

insulin and the two-bag system. We maintained patient confidentiality throughout the preparation of this case report and obtained informed consent for publication from the patient's parent or legal guardian. This study received approval from the hospital, as documented in the official approval letter (Letter No.0794.1/RSPWDC/LP/KEPK/VI/2026).

RESULT

A 12-year-old boy presented to the emergency department with complaints of recurrent vomiting more than 10 times per day since 1 week before hospital admission. The vomiting occurred after every meal, with a liquid content, without blood or bile. Complaints were accompanied by nausea, decreased appetite and drinking, and decreased frequency and volume of urination. The patient also complained of heartburn that radiated to the middle of the chest and felt like being pressed. There was no history of fever. The patient had undergone an independent laboratory test before with an HbA1c result of 10.3%, indicating chronic hyperglycemia. Family history revealed type 2 diabetes mellitus in a parent.

On arrival, the patient was fully conscious (composmentis) with the impression of being seriously ill. The patient appeared weak, dehydrated, and experiencing respiratory distress. The patient's weight was known to be 93 kg with a height of 165 cm (BMI 34.16 kg/m²), which is included in the obesity category, and the patient's waist circumference was measured at 98 cm, which is based on the Taylor waist circumference curve for a 12-year-old boy; this value is above the 80th percentile and thus meets the criteria for abdominal obesity. Vital signs showed blood pressure of 157/86 mmHg (grade 1 hypertension), pulse rate of 128 beats/minute (tachycardia), respiratory rate of 30 breaths/minute (tachypnea), and oxygen saturation of 97% without oxygen support.

Physical examination showed signs of dehydration in the form of sunken eyes and slow return of skin turgor. Hyperpigmentation was found in the neck region, acanthosis nigricans, which leads to insulin resistance. On chest examination, the breathing pattern appears rapid and deep (Kussmaul) with basic vesicular lung sounds without any additional sounds. Abdominal examination revealed tenderness in the epigastric region and left hypochondrium without signs of muscular defense. The extremities were warm without edema, with diaphoresis. After urinary catheterization, urine output was 1400 cc in 10 hours (±2 mL/kgBW/hour).

Table 1.
Blood Test

Hemoglobin	17.6 g/dL (H)
Hematocrit	50%
Leukocytes	19.1 x10^3/mcL (H)
Erythrocytes	6.3 M/mcL (H)
Platelets	259 K/mcL
Diff Count	
Eosinophils %	0.00 % (L)
Basophils %	0.50 %
Neutrophils %	85.40 % (H)
Lymphocytes %	9.20 % (L)
Monocytes %	4.90 %
Neutrophil Lymphocyte Ratio (NLR)	9.28
Sodium	135.1 mmol/L
Potassium	5.15 mmol/L (H)
Chloride	102.2 mmol/L
Blood Glucose	459 mg/dL (H)

Laboratory tests revealed severe metabolic disturbances. Hematology revealed a hemoglobin of 17.6 g/dL, hematocrit of 50%, and erythrocyte count of 6.3 million/mcL, suggesting hemoconcentration due to dehydration. A leukocytosis of 19.1 ×10^3/mcL with a predominance of neutrophils (85.4%) and an NLR of 9.28 indicated a systemic inflammatory response, with infection as a possible precipitating factor. Blood chemistry revealed severe hyperglycemia with a random

blood glucose of 459 mg/dL. Sodium of 135 mmol/L was within the low normal range, with the possibility of pseudohyponatremia due to hyperglycemia. Potassium of 5.15 mmol/L appeared elevated, but likely represented pseudohyperkalemia due to a shift of potassium from intracellular to extracellular fluid in acidosis, with actual decreased total body potassium reserves.

Table 2.
Blood Gas Analysis

Temperature	36.8^0C
FiO2	32%
pH	6.87 (L)
pCO2	<10 mmHg (L)
pO2	102.0 mmHg
BE	<-30 (L)
TCO2	1.0 mmol/L (L)
HCO_3^-	1.1 mmol/L (L)
ST HCO_3^-	6.4 mmol/L (L)
SO2	94 %
Lactate	1.26 mmol/L

Arterial blood gas analysis showed severe metabolic acidosis with a pH of 6.87, HCO_3^- of 1.1 mmol/L, a base excess of <-30, and a pCO_2 of <10 mmHg, indicating maximal respiratory compensation. A pO_2 of 102 mmHg and an oxygen saturation of 94% indicated adequate oxygenation. A lactate level of 1.26 mmol/L was within normal limits, suggesting that the acidosis was primarily due to ketone body accumulation, not lactic acidosis.

Table 2.
Urinalysis Test

Macroscopic	
Color	Yellow
Turbidity	A bit cloudy
Weight Type	1.025
pH	5.0
Ketone	Positive
Microscopic	
Erythrocytes	4.6 /mcL
Leukocytes	2.5 /mcL
Epithelium	8.4 /mcL
Cylinder	6.18 /mcL
Bacteria	132.2 /mcL
Protein in urine	2+
Urine Glucose	3+

Urinalysis revealed 3+ glucosuria and positive ketonuria, confirming hyperglycemia and ketosis. 2+ proteinuria was likely transient due to the acute condition and dehydration. A urine specific gravity of 1.025 indicated increased urine concentration. Bacteria were found in the urine (132.2/mcL), suggesting a possible urinary tract infection as a precipitating factor. Other supporting examinations showed a normal ECG and chest X-ray.

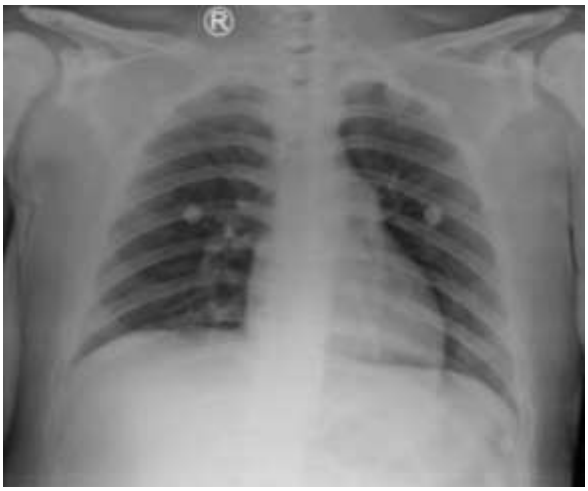

Figure 1. Thorax X-ray (heart size within normal limits and without pulmonary infiltrates)

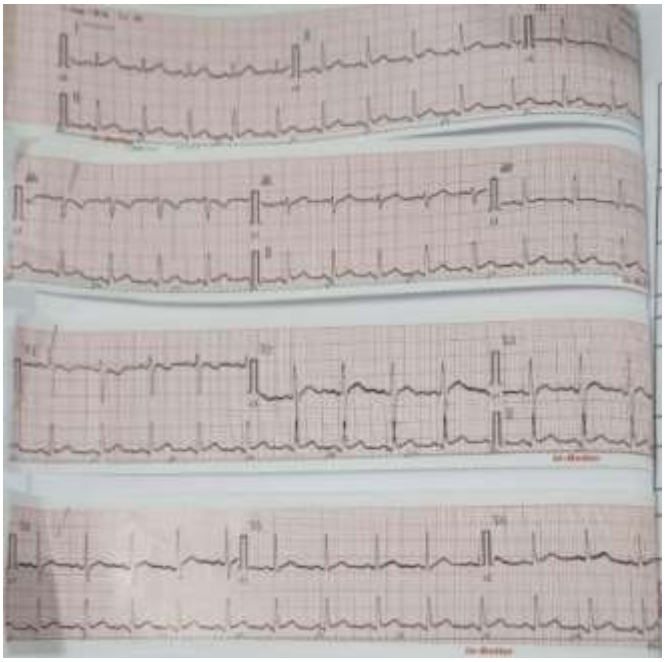

Figure 2. ECG (sinus tachycardia without T wave abnormalities)

In the initial phase of resuscitation in the ER, the patient received rapid-acting insulin intravenously as an initial bolus, accompanied by fluid resuscitation using 0.9% NaCl (initial bolus 500 mL followed by infusion of 200 mL/hour). The patient was then admitted to the ICU monitoring room and given continuous intravenous insulin therapy using a drip rapid-acting insulin with an initial dose of 2 IU/hour if blood glucose >300 mg/dL. The antibiotic ceftriaxone was administered considering the possibility of infection as a precipitating factor. The patient also received antihypertensive therapy in the form of candesartan and amlodipine due to comorbid hypertension in the context of metabolic syndrome.

During intravenous insulin therapy, patients are closely monitored using serial blood glucose checks every 1–2 hours with a target glucose reduction of no more than 90 mg/dL per hour to prevent complications of cerebral edema. The patient's blood glucose levels remained high, ranging from 480–432 mg/dL, so the insulin drip dose was increased to 3 IU/hour. Subsequent evaluations showed blood glucose levels still in the range of 304–394 mg/dL, with ketoacidosis not fully resolved. Therefore, the insulin dose was again increased to 4 IU/hour. Because persistent hyperglycemia persisted with blood glucose levels of 315–407 mg/dL, the insulin drip was then increased to 5 IU/hour.

When blood glucose levels fell to <200 mg/dL, the insulin dose was gradually reduced to 2.5 IU/hour, and dextrose fluid therapy was maintained using a two-bag system with 0.9% NaCl and 10% Dextrose to prevent hypoglycemia and still allow continuous insulin administration in the resolution of ketosis. Although the patient's clinical condition improved, persistent hyperglycemia with blood glucose >300 mg/dL was noted on several occasions, requiring insulin drip maintenance at a dose of 3–5 IU/hour during the intensive care phase.

Once the clinical condition is stable, the patient appears more active and oral tolerance improves, a transition from intravenous insulin to a basal-bolus subcutaneous insulin regimen is performed. The patient is given insulin glargine 40 IU per day as basal insulin and insulin aspart 27 IU three times daily before meals. The total initial daily insulin requirement reached 121 IU/day or approximately 1.3 IU/kg/day, indicating significant insulin resistance. To prevent rebound hyperglycemia, the insulin drip was gradually tapered by 2.5 IU/hour after basal insulin administration, then discontinued after the first dose of prandial insulin was administered. Prior to discharge, the insulin regimen was adjusted to insulin aspart 28-28-28 IU and insulin glargine 40 IU, with improved glycemic control and a stable clinical condition for outpatient treatment.

DISCUSSION

This case describes the occurrence of severe DKA in a 12-year-old boy with metabolic syndrome. According to IDAI, metabolic syndrome in children is defined by the presence of abdominal obesity, as assessed by a waist circumference greater than or equal to the 80th percentile according to the Taylor curve (Indonesian Pediatric Association, 2014; Taylor et al., 2000), accompanied by the presence of 2 parameters of metabolic disorders, including hypertension (average systolic and/or diastolic blood pressure value more than the 95th percentile) (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents, 2005), dyslipidemia (HDL ≤ 40 mg/dL or triglycerides ≥ 110 mg/dL), or insulin resistance (fasting blood glucose ≥ 100 mg/dL or diagnosed with DM) (Indonesian Pediatric Association, 2014; Weiss et al., 2013). The criteria for diagnosing diabetes mellitus in children based on IDAI referring to American Diabetes Association (ADA), namely if fasting plasma glucose examination is found to be ≥126 mg/dL, plasma glucose 2 hours after loading ≥200 mg/dL, random plasma glucose ≥200 mg/dL accompanied by classic symptoms of diabetes (polyuria, polydipsia, nocturia and weight loss without clear cause), or HbA1c ≥6.5% (Indonesian Pediatric Association, 2023; American Diabetes Association, 2024).

In this case, it was found that the patient's waist circumference was 98 cm, which is far beyond the P80 limit for 12-year-old boys, thus indicating central fat accumulation or abdominal obesity (Indonesian Pediatric Association, 2014). The patient's blood pressure was found to be 157/86 mmHg, which is based on the criteria of The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents; the value is above the 95th percentile for the patient's age, gender, and height, thus meeting the criteria for hypertension (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents, 2005). The patient's random blood glucose test results were 459 mg/dL, and HbA1c was 10.3%, which met the criteria for a diagnosis of diabetes mellitus according to IDAI (Indonesian Pediatric Association, 2023; American Diabetes Association, 2024). Based on the findings of abdominal obesity, hypertension, and type 2 diabetes mellitus in the patient, it can be concluded that the patient met the criteria for a diagnosis of metabolic syndrome according to the IDAI consensus (Indonesian Pediatric Association, 2014; Indonesian Pediatric Association, 2023).

Excessive caloric intake and sedentary behavior contribute to obesity and progressive insulin resistance. In susceptible individuals, pancreatic β-cell dysfunction eventually becomes insufficient to compensate for increasing insulin demand, leading to relative insulin deficiency. Impaired glucose utilization and increased hepatic gluconeogenesis contribute to hyperglycemia and

progression to type 2 diabetes mellitus. In severe insulin resistance, this relative insulin deficiency may also predispose to ketogenesis during metabolic stress (Indonesian Pediatric Association, 2014).

The patient presented with classic manifestations of DKA, including repeated vomiting, dehydration, tachypnea, severe hyperglycemia, positive ketonuria, and severe metabolic acidosis with a pH of 6.87. An HbA1c value of 10.3% indicates chronic hyperglycemia that likely predates the diagnosis. In pediatric patients, DKA is often the initial manifestation of diabetes, both type 1 and type 2, particularly in patients with metabolic syndrome (International Society for Pediatric and Adolescent Diabetes, 2022; Sapru et al., 2005).

Diabetic ketoacidosis is an acute complication of diabetes characterized by the triad of hyperglycemia, ketosis, and metabolic acidosis. This condition occurs due to absolute or relative insulin deficiency accompanied by increased levels of counterregulatory hormones such as glucagon, catecholamines, cortisol, and growth hormone (International Society for Pediatric and Adolescent Diabetes, 2022; Kitabchi et al., 2009). Insulin deficiency causes increased gluconeogenesis and glycogenolysis and decreased glucose utilization by peripheral tissues, resulting in hyperglycemia. Hyperglycemia then triggers osmotic diuresis, leading to significant fluid and electrolyte loss, dehydration, and hemoconcentration (International Society for Pediatric and Adolescent Diabetes, 2022).

In addition, insulin deficiency increases lipolysis, producing free fatty acids that are metabolized in the liver into ketone bodies, primarily acetoacetate and β-hydroxybutyrate. The accumulation of ketone bodies causes metabolic acidosis with an increased anion gap. In this case, a pH of 6.87 and an HCO_3^- of 1.1 mmol/L were found, indicating severe metabolic acidosis. To compensate, the body increases alveolar ventilation in the form of Kussmaul breaths to lower pCO_2 (International Society for Pediatric and Adolescent Diabetes, 2022; Sapru et al., 2005).

In metabolic acidosis, potassium also shifts from intracellular to extracellular so that serum potassium levels may appear normal or increased, even though the body's total potassium reserves are actually decreased due to osmotic diuresis (American Diabetes Association Professional Practice Committee, 2024). This is consistent with the patient's potassium level of 5.15 mmol/L. Furthermore, the patient experienced moderate to severe dehydration, characterized by decreased skin turgor, sunken eyes, tachycardia, and hemoconcentration with a hemoglobin of 17.6 g/dL. Leukocytosis with a predominance of neutrophils and bacteriuria suggest a possible infection as a precipitating factor for DKA in this case (International Society for Pediatric and Adolescent Diabetes, 2022).

Based on clinical findings and supporting examinations, the patient fulfilled the diagnostic triad of diabetic ketoacidosis, namely hyperglycemia (GDS 459 mg/dL), ketosis (positive ketonuria), and severe metabolic acidosis (pH <7.0; HCO_3^- <5 mmol/L), so was classified as severe DKA (International Society for Pediatric and Adolescent Diabetes, 2022).

Management of DKA in children focuses on four main pillars, namely fluid resuscitation, insulin therapy, electrolyte correction, and identification and management of triggering factors (International Society for Pediatric and Adolescent Diabetes, 2022). In this case, fluid resuscitation using 0.9% NaCl was performed to address dehydration and improve tissue perfusion. Gradual fluid correction is crucial to prevent complications of cerebral edema, especially in pediatric patients (Indonesian Pediatric Association, 2018; International Society for Pediatric and Adolescent Diabetes, 2022).

After initial stabilization, the patient received continuous intravenous insulin therapy. Insulin administration aims to halt ketogenesis and gradually lower blood glucose levels. In this case, a rapid bolus of 20 units of intravenous insulin was initially used, followed by a continuous insulin drip of 2 IU/hour, gradually increasing to 5 IU/hour due to persistent hyperglycemia. The relatively high insulin requirements are likely related to severe insulin resistance due to obesity and metabolic syndrome (American Diabetes Association Professional Practice Committee, 2024; International Society for Pediatric and Adolescent Diabetes, 2022).

ISPAD recommends a gradual decrease in glucose levels of around 50–90 mg/dL per hour to prevent cerebral edema as the most serious complication in pediatric DKA (Indonesian Pediatric Association, 2018; International Society for Pediatric and Adolescent Diabetes, 2022). In these patients, glucose monitoring is performed every 1–2 hours with a target reduction of no more than 90 mg/dL per hour. Once blood glucose levels reach approximately 250–300 mg/dL, a two-bag fluid system, a combination of 0.9% NaCl and 10% Dextrose, is used to maintain continuous insulin administration to correct ketoacidosis while preventing hypoglycemia (International Society for Pediatric and Adolescent Diabetes, 2022; American Diabetes Association Professional Practice Committee, 2024).

Correction of electrolytes, particularly potassium, is a crucial aspect of DKA management. Although patients appear hyperkalemic at the start of treatment, total body potassium may remain low, requiring close monitoring during insulin therapy to prevent hypokalemia, which can lead to arrhythmias (American Diabetes Association Professional Practice Committee, 2024). Sodium bicarbonate was also considered early in treatment due to the severe acidosis, but was not administered. This is in line with current recommendations that discourage routine bicarbonate administration in pediatric DKA except in certain conditions such as cardiac arrest or life-threatening hyperkalemia (Indonesian Pediatric Association, 2018; International Society for Pediatric and Adolescent Diabetes, 2022).

Furthermore, the decision not to immediately intubate was appropriate because the patient's spontaneous ventilation was still capable of providing respiratory compensation for metabolic acidosis. Intubation in patients with severe DKA can worsen the condition if post-intubation ventilation is unable to maintain adequate hyperventilation compensation, resulting in CO_2 retention and worsening acidosis. Close neurological monitoring is performed to detect complications such as cerebral edema, which is a major cause of mortality in pediatric DKA (Agarwal, 2019; International Society for Pediatric and Adolescent Diabetes, 2022).

The administration of the antibiotic ceftriaxone to this patient was based on the suspicion of infection as a precipitating factor, supported by findings of leukocytosis and bacteriuria. Identifying and treating precipitating factors is a crucial step in preventing DKA recurrence (International Society for Pediatric and Adolescent Diabetes, 2022).

During treatment, the patient's clinical condition gradually improved with improved consciousness, reduced shortness of breath, and improved oral tolerance. After the ketoacidosis resolved, the patient was switched to a basal-bolus subcutaneous insulin regimen using insulin glargine 40 IU and insulin aspart 27–28 IU three times daily. The total insulin requirement reached approximately 1.3 IU/kgBW/day, which is considered high and suggests significant insulin resistance. The patient's phenotype included obesity, hypertension, and acanthosis nigricans, and a history of consuming high-sugar drinks further supports the possibility of type 2 diabetes mellitus with presentation of diabetic ketoacidosis or ketosis-prone diabetes (Nadeau et al., 2016; Reinehr, 2013; Wang et al., 2025). This case demonstrates that severe DKA in obese children requires intensive monitoring and higher insulin requirements compared to classic DKA in type 1 diabetes mellitus. Prompt treatment, close serial monitoring, and adequate insulin titration provide good clinical outcomes without

severe complications such as cerebral edema, shock, or the need for mechanical ventilation (International Society for Pediatric and Adolescent Diabetes, 2022).

CONCLUSION

DKA can also be an early manifestation in children with characteristics of type 2 DM, particularly in those with obesity, insulin resistance, and metabolic syndrome. The diagnosis of DKA is based on the presence of the triad of hyperglycemia, ketosis, and metabolic acidosis, which in this case is supported by clinical findings in the form of dehydration and Kussmaul breathing and laboratory results showing severe hyperglycemia, ketonuria, and severe metabolic acidosis. Findings of leukocytosis and bacteriuria also suggest the possibility of infection as a precipitating factor for acute metabolic decompensation.

Comprehensive management, including fluid resuscitation, insulin therapy, electrolyte correction, and identification and management of precipitating factors, has been shown to play a crucial role in improving the patient's condition. In this case, a higher intravenous insulin requirement was found compared to the classic management of DKA in children, with gradual insulin drip titration from 2 IU/hour to 5 IU/hour due to persistent hyperglycemia, which is likely related to severe insulin resistance. A two-bag system, serial blood glucose, and close monitoring are crucial to prevent serious complications such as cerebral edema until resolution of ketoacidosis is achieved. This case highlights the importance of clinician awareness of the evolving clinical spectrum of DM in children, including the potential for DKA in type 2 DM, which requires higher insulin requirements due to significant insulin resistance. A systematic approach, individualized insulin dosing, and guideline-based management are essential to reduce morbidity and mortality in this condition.

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