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Diabetic ketoacidosis in an 11-year-old girl with type 1 diabetes mellitus: A case study

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Abstract

Background: Diabetic ketoacidosis (DKA) is a life-threatening acute complication of type 1 diabetes mellitus (T1DM) in children that requires prompt recognition and management.

Purpose: To describe the clinical presentation, management, clinical course, and outcome of severe DKA in an 11-year-old girl with T1DM.

Method: A descriptive case study was conducted based on a retrospective review of the patient's medical records. Data included medical history, physical examination, laboratory findings, treatment, clinical progression, and outcome during hospitalization.

Results: An 11-year-old girl with a two-year history of T1DM and three previous episodes of DKA presented with weakness, shortness of breath, nausea, vomiting, abdominal pain, altered consciousness, Kussmaul breathing, and dehydration. Blood glucose was 336 mg/dL with ketonuria +3 at the community health center. Upon hospital admission, blood glucose was GDS 510 mg/dL, ketonuria was +4, and arterial blood gas analysis showed severe metabolic acidosis (pH 6.67; HCO_3^- 2.0 mmol/L). The patient received intravenous fluid therapy, continuous insulin infusion, and intensive monitoring. Consciousness improved on the second day, and metabolic acidosis resolved on the third day (pH 7.40; HCO_3^- 22.2 mmol/L). The patient was discharged in stable condition after six days.

Conclusion: Early recognition, prompt referral, intensive monitoring, and appropriate management resulted in favorable clinical recovery.

Keywords: Children; Diabetic ketoacidosis; Metabolic Acidosis; Type 1 diabetes mellitus.

INTRODUCTION

Type 1 diabetes mellitus (DM) is a chronic metabolic disease caused by autoimmune destruction of pancreatic β -cells, resulting in absolute insulin deficiency. Insulin deficiency impairs to metabolism of carbohydrates, fats, and proteins, necessitating lifelong insulin therapy to maintain glycemic control and prevent acute and chronic complications (Glaser et al., 2022). Unlike type 2 diabetes, which is associated with insulin resistance, type 1 diabetes is more common in children and adolescents. It is one of the chronic diseases with an ever-increasing health burden in many countries. The rising incidence of type 1 diabetes is influenced by the interaction between genetic factors, the environment, viral infections, and lifestyle changes a phenomenon

that continues to be studied (International Diabetes Federation, 2021).

Globally, the number of children and adolescents living with type 1 diabetes is increasing every year. The International Diabetes Federation (IDF) estimates that more than 1.2 million children and adolescents under the age of 20 live with type 1 diabetes, with thousands of new cases diagnosed each year (International Diabetes Federation, 2021). In Indonesia, the incidence of type 1 diabetes in children also shows a steadily rising trend. According to the National Registry of the Indonesian Pediatric Society (IDAI), the prevalence of type 1 diabetes in children rose from 0.0038 per 100,000 children in 2000 to 0.028 per 100,000 in 2010. Then it increased

approximately 70-fold to reach 2 per 100,000 children in 2023 (Indonesian Pediatricians Association, 2022). This increase indicates that type 1 diabetes has become a health issue requiring special attention, particularly in terms of early detection, disease management, and prevention of complications.

One of the most common acute complications in children with type 1 diabetes is diabetic ketoacidosis (DKA). DKA is a metabolic emergency characterized by the triad of hyperglycemia, ketosis, and metabolic acidosis resulting from insulin deficiency accompanied by elevated levels of counterregulatory hormones, such as glucagon, catecholamines, cortisol, and growth hormone. Insulin deficiency leads to increased gluconeogenesis, glycogenolysis, and lipolysis, resulting in the accumulation of ketone bodies that cause metabolic acidosis. This condition is accompanied by osmotic diuresis, which causes massive fluid and electrolyte loss, putting patients at risk of severe dehydration, hypovolemic shock, impaired tissue perfusion, cerebral edema, cardiac arrhythmias, and even death if not treated promptly (Wolfsdorf et al., 2018).

Clinical manifestations of DKA in children include polyuria, polydipsia, weight loss, nausea, vomiting, abdominal pain, decreased consciousness, Kussmaul breathing, and a characteristic breath odor due to ketosis. The diagnosis is established based on a combination of hyperglycemia (blood glucose >200 mg/dL), metabolic acidosis (venous pH <7.30 or bicarbonate <15 mmol/L), and the presence of ketonemia or ketonuria (International Society for Pediatric and Adolescent Diabetes, 2022). The severity of DKA is classified as mild, moderate, or severe based on pH and serum bicarbonate levels. Severe DKA is characterized by a pH <7.10 or a bicarbonate level <5 mmol/L and is often accompanied by impaired consciousness, thus requiring intensive care in a critical care unit (Laffel et al., 2018).

Diabetic ketoacidosis remains a leading cause of hospitalization, morbidity, and mortality in children with type 1 diabetes. Various studies indicate that approximately 25–40% of children with type 1 diabetes are first diagnosed after experiencing DKA; in some countries with limited access to healthcare, this proportion can exceed 60% (Wolfsdorf et al., 2018). Consistent with another research, a history of previous DKA increases the risk of recurrent DKA

episodes, particularly in adolescents entering puberty. Hormonal changes during puberty increase physiological insulin resistance, thereby raising insulin requirements. If this condition is not balanced by adherence to insulin therapy and good glycemic control, the risk of developing DKA will increase significantly (Gregory et al., 2022).

In addition to age and hormonal changes, several studies have identified various other risk factors contributing to DKA episodes, including delayed diagnosis of type 1 diabetes, poor adherence to insulin therapy, poor glycemic control, infections, physiological stress, a history of previous DKA episodes, low family education levels, low socioeconomic status, and limited access to healthcare facilities (Kostopoulou et al., 2023). Another study reported that a significant proportion of recurrent DKA cases is associated with nonadherence to insulin and a lack of understanding among patients and their families regarding sick-day management. These findings indicate that successful management of type 1 diabetes depends not only on medical therapy but also on ongoing education, family support, and self-monitoring of blood glucose (Ehrmann et al., 2020).

DKA management must be carried out promptly, systematically, and in accordance with guidelines to restore metabolic balance and prevent complications. Management principles include fluid resuscitation, continuous intravenous insulin administration, correction of electrolyte imbalances particularly potassium monitoring of blood glucose levels, blood gas analysis, assessment of fluid balance, and identification and management of precipitating factors. Therapeutic success is greatly influenced by the timeliness of diagnosis, close clinical monitoring, and multidisciplinary collaboration throughout the course of care (Glaser et al., 2022).

RESEARCH METHOD

This study used a descriptive case study design to describe the clinical presentation, diagnostic process, management, clinical progression, and outcome of a pediatric patient with severe DKA associated with T1DM.

The patient was an 11-year-old girl with a two-year history of T1DM who initially received treatment at a community health center and was subsequently referred to a hospital. The patient was managed in the

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emergency department, intensive care unit (ICU), and inpatient ward.

Data were obtained retrospectively from the patient's medical records. The data included patient characteristics, medical history, chief complaints, physical examination findings, vital signs, laboratory findings, arterial blood gas analysis, treatment administered, clinical progression, insulin therapy, fluid management, and outcome until discharge.

The data were analyzed descriptively by presenting the clinical course chronologically from the initial presentation at the community health center to hospital discharge. The clinical findings and management were subsequently compared with the International Society for Pediatric and Adolescent Diabetes (ISPAD) Clinical Practice Consensus Guidelines and relevant scientific literature.

Patient confidentiality was maintained throughout the case report. Personal identifiers were removed, and the patient's identity was not disclosed.

RESEARCH RESULTS

An 11-year-old female patient presented with complaints of weakness that had begun one day before her family brought her to the emergency department of the community health center; the weakness was accompanied by progressively worsening shortness of breath, nausea and vomiting (three episodes), and abdominal pain. The patient was diagnosed with type 1 diabetes mellitus (type 1 DM) two years ago and has been on routine insulin therapy. She has experienced three episodes of diabetic ketoacidosis (DKA) in the past two years. Additionally, over the past two months, the patient has been experiencing regular monthly menstrual periods.

During the physical examination in the Community Health Center's emergency room, the following findings were noted: apathetic state of consciousness (E4V3M5), blood pressure 100/70 mmHg, heart rate 110 beats per minute, temperature 36.8°C, respiratory rate 28 breaths per minute, Kussmaul breathing pattern, and SpO₂ 98% on room air. Physical examination revealed slow skin turgor, dry mucous membranes, and symmetrical vesicular breath sounds bilaterally. Laboratory results from the community health center showed a GDS of 336 mg/dL and ketonuria +3. The patient received initial treatment consisting of rehydration with 500 mL of

Ringer's lactate (RL) administered over 20 minutes, repeated twice, along with ranitidine 25 mg (IV) and ondansetron 2 mg (IV). The patient was then immediately referred to a hospital approximately 2 hours away.

Upon arrival at the hospital's emergency department, the patient was in a stuporous state (E2V1M2), with a blood pressure of 90/60 mmHg, a heart rate of 134 beats per minute, a respiratory rate of 34 breaths per minute, and an SpO₂ of 95% on room air with Kussmaul breathing; the patient's urine output was 0.5 mL/kg/hour.

Laboratory tests revealed an Hb level of 14.1 g/dL, Hct 39.2%, white blood cell count 13,880/uL, platelet count 322,000/uL, GDS 510 g/dL, sodium 131.8 mmol, potassium 3.58 mmol, and chloride 108.8 mmol. Complete urinalysis: protein +3, glucose +3, ketones +4, red blood cells 7–11/lpb, white blood cells 2–4/lpb. Arterial blood gas (ABG) results: pH 6.67, pCO₂ 18.7 mmHg, pO₂ 93.6 mmHg, cHCO₃⁻ 2.0 mmol/L, cBE < -2 mmol/L, ctCO₂ 2.6 mmol/L.

The patient was diagnosed with decreased consciousness due to diabetic ketoacidosis, type 1 diabetes mellitus, and severe metabolic acidosis. Supporting evidence for this diagnosis includes decreased consciousness, Kussmaul's breathing pattern, a history of type 1 diabetes mellitus on insulin, elevated blood glucose levels, ketonuria, and decreased serum pH and bicarbonate levels.

The patient was treated in the hospital's emergency department with a nasal cannula delivering 2 liters of oxygen per minute, rehydration with 0.9% normal saline at a rate of 103 ml/hour for 48 hours, an insulin infusion at 0.1/kg body weight per hour (flow rate 2.7 cc/hour), a sodium bicarbonate infusion of 25 cc in 50 cc of sterile distilled water (flow rate 54 cc/hour), repeated a total of 4 times, ranitidine 2 × 30 mg (IV), ondansetron 3 × 2 mg (IV), and paracetamol 3 × 300 mg (IV). Blood glucose levels were monitored hourly, along with urine output and fluid balance.

The patient was then transferred to the Intensive Care Unit (ICU). On the first day, the patient remained in a stupor (E2V1M2), with a blood pressure of 104/60 mmHg, a heart rate of 100 beats per minute, an SpO₂ of 98% on a 2-liter-per-minute nasal cannula, and a temperature of 36.7°C. Treatment administered: fluid resuscitation with 0.9% normal saline at 103 ml/hour for 48 hours (starting at the 24-hour mark); the

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patient's blood glucose level stabilized at 227 mg/dL, so the insulin infusion rate was reduced to 0.05/kg body weight/hour. A repeat ABG test was performed, yielding the following results: pH 7.15, pCO₂ 16.8 mmHg, pO₂ 130.6 mmHg, cHCO₃⁻ 5.7 mmol/L, cBE -20.8 mmol/L, ctCO₂ 6.2 mmol/L.

On the second day of ICU care, the patient's level of consciousness improved (*compos mentis*, E4V5M6), and vital signs were within normal limits. The patient's blood glucose levels ranged from 160 to 380 mg/dL. After 48 hours of fluid resuscitation, maintenance fluid therapy was continued with D51/4NS 500 mL + KCl 25 mEq every 12 hours and 0.9% NS 500 mL every 12 hours, along with an insulin drip of 0.01–0.05 units/kg body weight per hour. On the third day of treatment, the patient underwent a repeat ABG test, yielding the following results: pH 7.40, pCO₂ 36.2 mmHg, pO₂ 34.7 mmHg, and cHCO₃⁻ 22.2 mmol/L.

Next, the insulin drip was discontinued and replaced with subcutaneous injections of 1×10 units of basal insulin and 3×8 units of short-acting insulin. The patient was then transferred to a general ward and remained hospitalized for 6 days. Afterward, the patient attended routine follow-up appointments at the hospital's outpatient clinic and received an adjustment to the new insulin therapy regimen: 8, 10, and 9 units of short-acting insulin, and 12 units of basal insulin.

DISCUSSION

Diabetic ketoacidosis (DKA) is the most common acute complication and the leading cause of morbidity and mortality in children with type 1 diabetes mellitus (DM). Globally, the incidence of DKA remains quite high, both as an initial manifestation of type 1 DM and as a recurrent complication throughout the course of the disease. The 2022 guidelines from the International Society for Pediatric and Adolescent Diabetes (ISPAD) report that approximately 25-40% of children with type 1 DM are first diagnosed while in a state of DKA; in some countries, this figure may even exceed 60% due to delayed diagnosis and limited access to healthcare services. Furthermore, recurrent DKA remains a major problem among patients with a history of DKA, poor glycemic control, and increased insulin requirements during puberty (Glaser et al., 2022).

In this case, several risk factors were identified that can trigger DKA, namely adolescence or puberty and a history of previous DKA. According to ISPAD, recurrent DKA in patients with type 1 diabetes on regular insulin therapy is most often caused by patient nonadherence to insulin use, whether intentional or unintentional (Glaser et al., 2022; EL-Mohandes et al., 2017; Al Zahrani & Al Shaikh, 2019). However, during the current DKA episode, the patient was found to be continuing to receive routine insulin injections, so that this factor can be ruled out. Furthermore, it was noted that the patient had menstruated within the past 2 months.

This significantly affects hormonal and physiological changes in the body, namely a surge in the secretion of growth hormone (GH), sex hormones (estrogen and testosterone), and other anti-insulin hormones (Glaser et al., 2022; Chowdhury, 2015). Physiologically, increased GH levels cause peripheral tissues to become less sensitive to insulin; therefore, adolescents require higher insulin doses than children. This is consistent with another research conducted which shows that the increased incidence of DKA in children and adolescents is influenced not only by non-adherence to therapy but also by delays in adjusting insulin therapy to match changes in patients' metabolic needs (Castellanos et al., 2020).

According to the 2024 National Clinical Practice Guidelines (PNPK) for Diabetes Mellitus in Children, DKA is diagnosed based on the presence of: (1) hyperglycemia, blood glucose level >200 mg/dL, (2) acidosis, pH <7.3 and/or HCO₃⁻ <18 mEq/L, and (3) ketonemia (>3 mmol/L) and/or moderate to severe ketonuria. When assessing the severity of the acidosis, it can be classified as: (1) mild DKA, pH <7.3 and/or HCO₃⁻ <18 mEq/L, (2) moderate DKA, pH <7.2 and/or HCO₃⁻ <10 mEq/L, (3) severe DKA, pH <7.1 and/or HCO₃⁻ <5 mEq/L (Ministry of Health of the Republic of Indonesia, 2025).

Dehydration in DKA is usually caused by osmotic diuresis and electrolyte imbalances resulting from hyperglycemia and hyperketonemia. Ketonemia increases blood lactate levels, leading to nausea and vomiting, and to Kussmaul breathing as a compensatory mechanism. In addition, hyperglycemia increases diuresis, thereby further exacerbating dehydration in DKA (Kim, 2024).

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Upon arrival at the community health center's emergency department, the patient was beginning to experience a decline in consciousness (apathy), accompanied by Kussmaul breathing, slow skin turgor, dry mucous membranes, tachycardia, a blood glucose level of 336 mg/dL, and ketonuria +3. Based on the patient's clinical presentation, along with simple supportive tests conducted at the community health center and a brief medical history, an emergency diagnosis was established and managed promptly in accordance with the Pediatric Advanced Life Support (PALS) guidelines, by securing the airway and ensuring adequate breathing-as indicated by the patient's stable SpO₂ of 98% on room air-and administering rapid rehydration fluids totaling 500 mL over 20 minutes, repeated twice, to address circulatory compromise. The patient was then referred to the hospital's emergency department via ambulance for further management (Glaser et al., 2022)

During the long trip, the patient's condition progressively worsened, as evidenced by a decline in consciousness upon arrival at the hospital's emergency department (somnolence, E2V1M2), Kussmaul breathing, and tachycardia. Laboratory results showed a GDS of 510 mg/dL, ketonuria +4, pH 6.67, and cHCO₃- 2.0 mmol/L. These findings met the diagnostic criteria for DKA and indicated severe DKA (Indonesian Pediatric Society. 2017; Kim, 2024).

The principles of DKA management, as previously explained, include initial care in accordance with PALS guidelines-namely, securing the airway and ensuring adequate breathing by administering oxygen via a nasal cannula at 2 liters per minute upon arrival at the hospital. This is followed by fluid therapy to treat dehydration and restore circulatory function, insulin administration to halt excessive ketone production, correction of electrolyte imbalances, and addressing the underlying cause of DKA (Indonesian Pediatricians Association, 2022).

The fluid therapy used must be isotonic, such as 0.9% NaCl or Ringer's Lactate (RL) at a dose of 20 mL/kg body weight, administered over 20–30 minutes. If no clinical improvement is observed, the treatment may be repeated once. If the patient's condition improves but has not yet stabilized, administer another fluid bolus of 10 mL/kg body weight over 1–2 hours. Insulin therapy is administered 30 minutes to 1 hour after fluid rehydration is

completed. If hypokalemia is present, it should be corrected concurrently with initial fluid resuscitation, before insulin administration. Subsequently, fluid rehydration is administered over 48 hours, consisting of the remaining fluid deficit (after bolus administration) plus the maintenance fluid requirement for 48 hours (Glaser et al., 2022; Kim, 2024).

The occurrence of cerebral edema can result in a 60–90% mortality rate in patients with DKA. Cerebral edema can occur before treatment is administered or even develop 12 hours or more after treatment.4 Therefore, patients with DKA need to be closely monitored. The clinical criteria used to diagnose cerebral edema in patients with DKA are either 1 diagnostic criterion plus 2 major criteria or 1 major criterion plus 2 minor criteria (Kim, 2024).

Diagnostic criteria: abnormal motor and verbal responses to pain; decorticate or decerebrate posture; cranial nerve palsy; abnormal breathing patterns (tachypnea, Cheyne-Stokes respiration, apnea). Major criteria: fluctuations in level of consciousness; a consistent decrease in heart rate not related to improvement in intravascular volume or sleep; age-inappropriate incontinence. Minor criteria: vomiting; headache; lethargy or difficulty waking; diastolic blood pressure >90 mmHg; age <5 years (Glaser et al., 2022).

On the third day of treatment, intravenous insulin is gradually discontinued 4 hours after subcutaneous basal insulin administration. This is intended to allow sufficient time for the basal insulin to take effect, thereby preventing a spike in hyperglycemia that could trigger another episode of DKA. On the last day of treatment, the patient's condition was stable, and he had no complaints, so he was discharged. The patient and his family were educated about the current course of his disease and asked to continue follow-up visits at the hospital's outpatient clinic. The insulin dosage during puberty can be increased to 1.2–2 units/kg body weight/day. This is consistent with the therapy the patient received following a follow-up visit at the hospital outpatient clinic; the initial dosage prescribed for a patient weighing 27 kg was 3 × 8 units of short-acting insulin and 1 × 10 units of basal insulin. After dose adjustments to achieve a target insulin dose of 32.4–54 units per day, the patient received insulin therapy consisting of short-acting insulin at doses of 8 units, 10 units, and 9 units, and basal

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insulin at 12 units (total 39 units per day) (Dhatariya et al., 2020).

These dosages are not standardized, as people with type 1 diabetes who are entering puberty—especially females—experience social dynamics that can also affect their psychological and hormonal states (Mavinkurve et al., 2024 ; Thabet et al., 2024). Furthermore, there is concern that patients with type 1 diabetes whose blood sugar levels are uncontrolled may face long-term risks, including an impact on their adult height. Therefore, patients and their families are advised to attend regular checkups and thoroughly document all activities, food intake, and the patient's daily blood glucose levels (Alsolaimani et al., 2024; Smew et al., 2025).

This case study has important implications for clinical practice. First, primary care providers need to be highly vigilant for early symptoms of DKA so that diagnosis and initial management can be carried out as soon as possible before referral. Second, periodic evaluation of insulin needs in children entering puberty should be part of routine care, as hormonal changes increase insulin resistance and the risk of DKA. Third, ongoing education for patients and families regarding insulin therapy adherence, self-monitoring of blood glucose, dietary management, physical activity, and sick-day management is crucial to preventing DKA recurrence. A multidisciplinary approach involving doctors, nurses, dietitians, patients, and families is expected to improve self-management skills, reduce hospitalization rates due to recurrent DKA, and improve the quality of life for children with type 1 diabetes.

CONCLUSION

DKA in people with type 1 diabetes can recur due to various triggering factors; one of these is when a person with type 1 diabetes enters puberty. Even with regular insulin use, patients can still develop HCA due to the hormonal changes that occur during puberty. Therefore, people with type 1 diabetes must consistently take their medication, visit the nearest healthcare facility for check-ups, and be attentive to changes in their bodies to prevent unwanted complications. Early diagnosis and comprehensive management also play a crucial role in reducing morbidity and mortality rates among DKA patients, particularly in primary care settings with limited resources.

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SUGGESTION

Based on the results of this case study, healthcare professionals are encouraged to increase their vigilance regarding the early signs and symptoms of diabetic ketoacidosis (DKA) in children with type 1 diabetes mellitus (DM), particularly in patients with a history of recurrent DKA who have entered puberty. Early diagnosis, prompt management in accordance with guidelines, and periodic evaluation and dose adjustment of insulin are necessary to anticipate increased insulin requirements due to hormonal changes during puberty. Ongoing education for patients and their families regarding insulin therapy adherence, self-monitoring of blood glucose, implementation of sick-day management, and early detection of DKA symptoms needs to be improved to prevent recurrence, enhance self-management skills, and optimize glycemic control.

For future research, studies with larger sample sizes and analytical or cohort designs are needed to identify factors associated with recurrent DKA in children with type 1 diabetes, particularly the impact of hormonal changes during puberty on insulin requirements and clinical outcomes. The results of such studies are expected to serve as a basis for developing more effective strategies for the prevention and management of DKA.

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