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DIABETIC KETOACIDOSIS MIMICKING RESPIRATORY DISTRESS IN AN OBESE ADOLESCENT PATIENT: A CASE REPORT

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ABSTRACT

Introduction: Diabetic ketoacidosis (DKA) is a serious acute complication of diabetes mellitus characterized by hyperglycemia, ketosis, and metabolic acidosis requiring prompt management. In children and adolescents, DKA often represents the initial manifestation of diabetes and may mimic respiratory disorders, potentially leading to delayed diagnosis and increased morbidity.

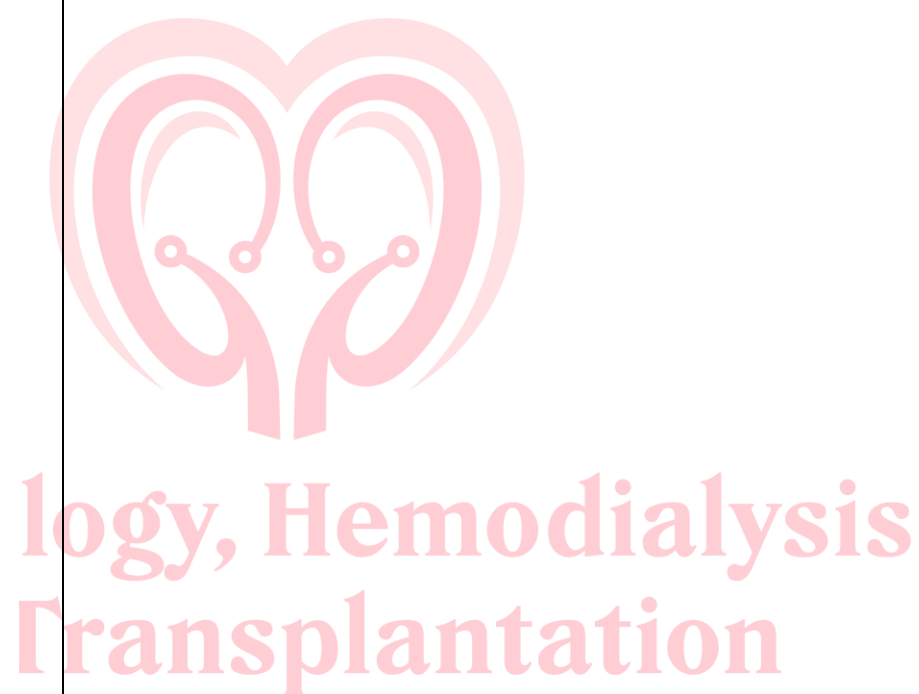
Case Illustration: We report a case of a 13-year-old obese boy who presented to the emergency department with progressive shortness of breath for one week, accompanied by polyuria, polydipsia, nausea, fatigue, and significant weight loss of 5 kg. Physical examination revealed Kussmaul respiration and acanthosis nigricans on the neck. Laboratory findings

demonstrated severe hyperglycemia (random blood glucose 381 mg/dL), marked ketonuria (+4), and severe metabolic acidosis with arterial pH 7.06 and bicarbonate 2 mEq/L, confirming severe DKA. The patient received comprehensive management according to ISPAD guidelines, including oxygen therapy, intravenous rehydration with calculated fluid replacement based on ideal body weight, and continuous intravenous insulin aspart infusion at 0.08 units/kg/hour. Blood glucose monitoring guided fluid adjustments with dextrose-containing solutions as glucose levels declined.

Discussion: The patient's respiratory manifestations, particularly Kussmaul respiration, represented physiological compensation for severe metabolic acidosis rather than primary pulmonary pathology. Obesity and acanthosis nigricans suggested underlying insulin resistance, potentially indicating type 2 diabetes mellitus, though definitive classification required further evaluation. Management followed ISPAD guidelines with gradual fluid correction over 48 hours to minimize cerebral edema risk, continuous insulin infusion until DKA resolution, and transition to subcutaneous basal-bolus insulin regimen. Clinical improvement was achieved by day four with pH increasing to 7.32 and bicarbonate to 18 mEq/L.

Conclusion: This case highlights the importance of recognizing DKA in adolescents presenting with respiratory symptoms, particularly in obese patients with insulin resistance markers. Prompt diagnosis and adherence to evidence-based protocols are essential for optimal outcomes and prevention of life-threatening complications.

Keywords: diabetic ketoacidosis, adolescent, Kussmaul respiration, insulin resistance, obesity



INTRODUCTION

Diabetic ketoacidosis (DKA) is one of the most serious acute complications of diabetes mellitus and represents a medical emergency that requires prompt management. The incidence of DKA is estimated to be approximately 4–8% among patients with type 1 diabetes mellitus (T1DM). In children and adolescents, DKA frequently represents the initial manifestation of diabetes, with approximately 15–70% to 80% of children younger than 15 years presenting with DKA at the time of their first diagnosis of T1DM.¹

DKA occurs as a result of absolute or relative insulin deficiency, leading to hyperglycemia, ketosis, metabolic acidosis, and dehydration. This condition is more commonly associated with type 1 diabetes mellitus; however, it may also occur in type 2 diabetes mellitus, particularly in adolescents with obesity or metabolic stress such as infection or delayed diagnosis.² Clinical manifestations of DKA vary widely, ranging from polyuria, polydipsia, and weight loss to respiratory abnormalities characterized by rapid and deep breathing (Kussmaul respiration).³

In some cases, the respiratory manifestations of DKA may resemble respiratory distress or pulmonary disease, potentially leading to delayed diagnosis if not recognized promptly.⁴ Therefore, early recognition of the clinical features of DKA is crucial to prevent more severe complications. This case report presents an adolescent with diabetic ketoacidosis who presented with shortness of breath mimicking respiratory distress and discusses the clinical manifestations and management based on current therapeutic guidelines.

CASE REPORT

A 13-year-old boy presented to the emergency department with a chief complaint of shortness of breath for one week prior to admission, which had worsened several hours before hospitalization. The dyspnea was aggravated by physical activity. The patient had previously received nebulization therapy at another healthcare facility without improvement. The symptoms were accompanied by intermittent fever for one week. The patient also reported excessive thirst, frequent urination particularly at night, nausea without vomiting, easy fatigability, and an approximate weight loss of 5 kg during this period. Bowel movements were normal. There was no

family history of diabetes mellitus. The patient had a history of asthma, with the last exacerbation occurring one month prior to admission.

Anthropometric examination revealed a body weight of 68 kg and height of 153 cm, which were categorized as obese according to the Waterlow classification. The patient was fully conscious with a Glasgow Coma Scale (GCS) score of 15. Vital signs showed blood pressure of 160/87 mmHg, heart rate of 127 beats per minute, body temperature of 37.1°C, and oxygen saturation of 98% on room air.

Physical examination revealed rapid and deep breathing consistent with Kussmaul respiration. Acanthosis nigricans was observed on the neck. The oral mucosa appeared dry. Lung auscultation revealed vesicular breath sounds without crackles or wheezing. Abdominal examination revealed tenderness in the epigastric region. Skin turgor was within normal limits, and no edema was observed in the extremities.

Laboratory investigations showed a random blood glucose level of 381 mg/dL. Urinalysis demonstrated ketonuria (+4), glucosuria (+3), and proteinuria (+2). Arterial blood gas analysis revealed severe metabolic acidosis with pH 7.06 (reference range 7.35–7.45), bicarbonate (HCO_3^-) level 2 mEq/L (reference range 22–26 mEq/L), pCO_2 7 mmHg (reference range 35–45 mmHg), and base excess of -29 mmol/L (reference range -2 to +2 mmol/L). Complete blood count showed slight leukocytes $11.51 \times 10^3/\mu\text{L}$ (reference range $4-10 \times 10^3/\mu\text{L}$), hemoglobin 16.3 g/dL (reference range 13–17 g/dL), hematocrit 47.2% (reference range 40–50%), and platelets $295 \times 10^3/\mu\text{L}$ (reference range $150-450 \times 10^3/\mu\text{L}$). Renal function tests showed urea 14 mg/dL (reference range 10–40 mg/dL) and creatinine 0.9 mg/dL (reference range 0.6–1.2 mg/dL). Liver function tests revealed AST (SGOT) 14 U/L (reference range 10–40 U/L) and ALT (SGPT) 11 U/L (reference range 7–56 U/L). Electrolyte analysis showed sodium 130 mEq/L (reference range 135–145 mEq/L), potassium 3.8 mEq/L (reference range 3.5–5.0 mEq/L), and chloride 100 mEq/L (reference range 98–106 mEq/L). HbA1c level was 10% (reference range 4–5.6%).

Based on the clinical presentation and laboratory findings, the patient was diagnosed with severe diabetic ketoacidosis accompanied by mild dehydration. The patient received oxygen therapy via nasal cannula at a flow rate of 2 L/min. Initial rehydration was performed with intravenous 0.9% sodium chloride during the first hour. Therapy was continued with continuous

insulin infusion using rapid-acting insulin aspart at a rate of 3.5 units per hour (0.05–0.1 units/kg/hour). The patient also received intravenous fluids consisting of D5 ½ normal saline supplemented with potassium chloride (KCl) 10 mEq at a rate of 36 drops per minute. Blood glucose levels were monitored every four hours. The patient was also treated with ceftriaxone 1 g every 12 hours. During hospitalization, the patient received nutritional therapy with an estimated energy requirement of approximately 2300 kcal per day, consisting of breakfast (500 kcal), lunch (600 kcal), dinner (600 kcal), and three snacks including morning snack (150 kcal), afternoon snack (200 kcal), and evening snack (250 kcal).

Four hours after therapy initiation, the blood glucose level decreased to 311 mg/dL. Subsequent management was adjusted according to blood glucose monitoring. When blood glucose levels were between 200–350 mg/dL, the patient received intravenous D5 ½ normal saline with additional KCl 10 mEq along with continuous insulin infusion at 3.5 mL per hour. When blood glucose levels decreased to 150–200 mg/dL, intravenous fluids were changed to 10% dextrose with additional KCl 10 mEq. If blood glucose levels dropped below 150 mg/dL, the insulin infusion rate was reduced to 3 mL per hour, and further reduced to 2 mL per hour if blood glucose levels fell below 100 mg/dL. If blood glucose levels decreased below 60 mg/dL, insulin infusion was temporarily discontinued for one hour and then resumed at a rate of 2 mL per hour. Arterial blood gas and electrolyte levels were monitored every 24 hours.

On the fourth day of hospitalization, the patient's clinical condition improved. The complaint of shortness of breath resolved and vital signs returned to normal limits. Arterial blood gas analysis showed improvement with pH increasing from 7.06 to 7.32 and bicarbonate level increasing from 2 mEq/L to 18 mEq/L.

Insulin therapy was subsequently transitioned from intravenous infusion to subcutaneous administration. The patient received an insulin regimen consisting of insulin aspart 10 units three times daily and insulin detemir 24 units once daily at night. The patient also received education on insulin administration and self-monitoring of blood glucose. On the sixth day of hospitalization, the patient was discharged in stable condition with advice to follow up at the pediatric endocrinology outpatient clinic one week after discharge.

DISCUSSION

Diabetic ketoacidosis (DKA) is a serious acute complication of diabetes mellitus characterized by the triad of hyperglycemia, ketosis, and metabolic acidosis. This condition occurs predominantly in type 1 diabetes mellitus but may also be observed in type 2 diabetes mellitus, particularly in adolescents with obesity or insulin resistance.^{1,2} In pediatric and adolescent populations, DKA frequently represents the initial manifestation of diabetes and occurs in approximately 15–70% of patients at the time of first diagnosis.¹

The diagnosis of DKA is established based on the presence of hyperglycemia (plasma glucose >200 mg/dL), metabolic acidosis (venous pH <7.3 or bicarbonate <15 mEq/L), and ketosis demonstrated by ketonemia or ketonuria.⁴ The severity of DKA is classified as mild, moderate, or severe according to pH and bicarbonate levels. Severe DKA is defined by a pH <7.1 or bicarbonate level <5 mEq/L.⁴ In the present case, the patient had a pH of 7.06 and a bicarbonate level of 2 mEq/L, therefore fulfilling the criteria for severe DKA.

Insulin deficiency in DKA leads to increased hepatic glucose production through gluconeogenesis and glycogenolysis, as well as reduced peripheral glucose utilization. This results in significant hyperglycemia. In addition, insulin deficiency enhances lipolysis, producing free fatty acids that are subsequently converted in the liver into ketone bodies, namely acetoacetate and β -hydroxybutyrate. The accumulation of these ketone bodies leads to metabolic acidosis, which represents the hallmark of DKA.⁵

In response to metabolic acidosis, the body develops respiratory compensation characterized by rapid and deep breathing known as Kussmaul respiration.⁶ This breathing pattern aims to reduce carbon dioxide levels and thereby help correct the acid–base imbalance. In this case, the patient presented with shortness of breath and Kussmaul respiration as a compensatory response to severe metabolic acidosis. These respiratory manifestations may resemble respiratory distress or pulmonary disease, potentially leading to delayed diagnosis if not recognized promptly.⁴

Hyperglycemia exceeding the renal threshold for glucose reabsorption results in glucosuria, which induces osmotic diuresis. This process leads to substantial loss of water and electrolytes, causing dehydration and excessive thirst.^{3,7,8} Fluid loss in DKA may reach approximately 5–10% of body weight, although clinical estimation of dehydration severity is often

inaccurate.⁴ In this case, the patient demonstrated mild clinical dehydration characterized by dry oral mucosa without signs of peripheral hypoperfusion.

The initial management of pediatric DKA focuses on stabilization of airway, breathing, and circulation (ABC), followed by fluid therapy, insulin administration, and correction of electrolyte disturbances. Rehydration is a crucial component of DKA management as it improves tissue perfusion, increases glomerular filtration, and lowers blood glucose levels through enhanced renal glucose excretion.⁴

Assessment of fluid requirements in pediatric DKA is generally based on estimated dehydration severity and maintenance fluid needs. In overweight or obese patients, several guidelines recommend using ideal body weight for fluid calculation to avoid excessive fluid administration that may increase the risk of cerebral edema.⁴ In this case, the patient had a height of 153 cm and an actual body weight of 68 kg. Based on the CDC growth chart, the patient's ideal body weight was estimated at 43 kg. Clinical dehydration was estimated at approximately 3%, and the fluid deficit was therefore calculated as follows:⁹

$$\begin{aligned}\text{Fluid deficit} &= \text{degree of dehydration} \times \text{ideal body weight} \times 1000 \text{ ml} \\ \text{Fluid deficit} &= 3\% \times 43 \text{ kg} \times 1000 \text{ ml} \\ &= 1290 \text{ ml}\end{aligned}$$

This fluid deficit was replaced gradually along with maintenance fluid requirements over 24–48 hours according to the recommendations of the International Society for Pediatric and Adolescent Diabetes (ISPAD) guidelines to prevent rapid changes in osmolality.⁴ Maintenance fluid requirements over 48 hours were calculated using the Holliday–Segar formula, which consists of 200 mL/kg for the first 10 kg, 100 mL/kg for the next 10 kg, and 40 mL/kg for each additional kilogram.¹⁰ Using the patient's ideal body weight of 43 kg, the total maintenance fluid requirement was calculated as 3920 mL over 48 hours (2000 mL for the first 10 kg, 1000 mL for the next 10 kg, and 920 mL for the remaining 23 kg).

According to the Indonesian Pediatric Society (IDAI) consensus, total fluid requirements in patients with DKA are calculated as the sum of the fluid deficit and maintenance fluid requirements.⁹ With a fluid deficit of approximately 1290 mL, the patient's total fluid requirement

was calculated to be approximately 5210 mL over 48 hours, administered at a rate of approximately 36 drops per minute using a macrodrip set. This approach is consistent with the ISPAD recommendations and IDAI consensus, which emphasize gradual correction of fluid deficits over 24–48 hours to improve tissue perfusion while minimizing the risk of neurological complications such as cerebral edema.⁴ Continuous intravenous insulin therapy is an essential component of DKA management. ISPAD guidelines recommend continuous intravenous insulin administration at a dose of 0.05–0.1 units/kg/hour, initiated approximately one hour after fluid therapy has begun.⁴

In this case, rapid-acting insulin aspart was administered at a dose of approximately 3.5 units per hour (≈ 0.08 units/kg/hour) based on ideal body weight, which falls within the recommended ISPAD range for continuous intravenous insulin therapy in DKA (0.05–0.1 units/kg/hour). This therapy aims to suppress ketogenesis, gradually reduce blood glucose levels, and correct metabolic acidosis until DKA resolution is achieved.⁴

After metabolic stabilization was achieved, intravenous insulin therapy was transitioned to subcutaneous insulin using a basal–bolus regimen consisting of insulin aspart 10 units three times daily as prandial insulin and insulin detemir 24 units once daily at night as basal insulin, with a total daily dose of 54 units. This dosage is consistent with ISPAD recommendations suggesting an initial subcutaneous insulin dose of approximately 0.8–1 unit/kg/day in children and adolescents. Based on the patient's actual body weight of 68 kg, the estimated total daily insulin requirement was approximately 54 units (0.8 units/kg/day).⁴

Insulin therapy aims to suppress ketone production, gradually reduce blood glucose levels, and correct metabolic acidosis. During intravenous insulin therapy, blood glucose levels should be monitored serially to ensure a safe rate of glucose reduction, typically around 50–90 mg/dL per hour.⁴

When blood glucose levels decrease to approximately 250–300 mg/dL, intravenous fluids are recommended to include dextrose to prevent hypoglycemia and allow insulin therapy to continue until ketoacidosis resolves.⁴ In this case, intravenous fluids were subsequently changed to D5 ½ normal saline and later to D10% as blood glucose levels decreased further.

Intravenous insulin therapy is continued until DKA resolution is achieved, which is defined by pH >7.30, bicarbonate ≥ 18 mEq/L, or closure of the anion gap.⁴ In this patient, improvement in acid–

base balance was observed on the fourth day of hospitalization with an increase in pH from 7.06 to 7.32 and bicarbonate from 2 mEq/L to 18 mEq/L, after which insulin therapy was transitioned to subcutaneous administration.

After metabolic stabilization, the patient began receiving structured nutritional therapy. Energy requirements in children and adolescents can be estimated using the simple formula $1000 + 100 \times \text{age (years)}$, which is commonly used in pediatric practice as an approximation of daily caloric needs. In this 13-year-old patient, the estimated daily energy requirement was approximately 2300 kcal. Energy intake was distributed into three main meals and three snacks to help maintain stable blood glucose levels and to align with the insulin regimen.

The macronutrient distribution consisted of approximately 65% carbohydrates, 20% protein, and 15% fat. According to the International Society for Pediatric and Adolescent Diabetes (ISPAD) 2022 guidelines, nutritional therapy for children with diabetes should be individualized, taking into account age, nutritional status, physical activity, and metabolic condition.⁴ In this case, although the patient had obesity, energy intake during the early phase of treatment was intended to ensure adequate caloric intake, prevent a catabolic state following the episode of diabetic ketoacidosis, and support metabolic stabilization during recovery. In this patient, obesity and acanthosis nigricans were observed, both of which are markers of insulin resistance and are commonly associated with type 2 diabetes mellitus in children.^{11, 12}

However, overweight and obesity may also be found in some patients with type 1 diabetes mellitus.¹³ Therefore, the type of diabetes in this patient could not be definitively determined because C-peptide and pancreatic autoantibody examinations were not performed during hospitalization. Further metabolic evaluation is required to establish a definitive diagnosis.

Overall, this case report highlights the importance of early recognition of the clinical manifestations of diabetic ketoacidosis that may mimic respiratory disorders. Prompt and appropriate management in accordance with ISPAD guidelines—including fluid therapy, continuous intravenous insulin administration, and correction of metabolic disturbances—plays a crucial role in improving clinical outcomes and preventing severe complications.

CONCLUSION

This case report demonstrates that diabetic ketoacidosis should be considered in the differential diagnosis of adolescents presenting with respiratory symptoms, particularly when accompanied by classic manifestations of hyperglycemia such as polyuria, polydipsia, and weight loss. The presence of obesity and acanthosis nigricans further raises suspicion for underlying insulin resistance, which may predispose to DKA even in type 2 diabetes mellitus. Early recognition of Kussmaul respiration as a compensatory mechanism for metabolic acidosis rather than primary pulmonary pathology is essential to prevent diagnostic delays and ensure appropriate management. This case underscores the importance of adhering to evidence-based protocols, including calculated fluid resuscitation based on ideal body weight in obese patients, continuous intravenous insulin infusion, and gradual metabolic correction, all of which contributed to the favorable clinical outcome in this patient. Comprehensive evaluation including C-peptide and autoantibody testing is recommended to establish the definitive diabetes type and guide long-term management.

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