



Supraventricular Tachycardia in an Adolescent with Diabetic Ketoacidosis, Severe Hyperkalemia, and Pneumonia: A Case Report from a District Hospital in Indonesia

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ABSTRACT

Introduction: Diabetic ketoacidosis (DKA) is a major cause of morbidity and mortality in children with type 1 diabetes mellitus (T1DM). Although electrolyte disturbances are common, life-threatening arrhythmias such as supraventricular tachycardia (SVT) are rarely reported, especially in resource-limited settings. This case illustrates the successful management of SVT triggered by severe hyperkalemia during DKA.

Case Illustration: A 14-year-old girl with known T1DM presented with unconsciousness, Kussmaul breathing, and a three-day history of fever, cough, and vomiting. Initial assessment revealed Glasgow Coma Scale 9/10, heart rate 158 bpm, and blood pressure 113/64 mmHg. Laboratory findings showed blood glucose 425 mg/dL, pH 6.92, bicarbonate 3.4 mmol/L, and potassium 8.2 mmol/L. Serial electrocardiograms (ECG) showed peaked T-waves followed by narrow-complex SVT. Due to unavailability of adenosine, a vagal maneuver using an ice pack on

the face was performed, along with intravenous calcium gluconate and insulin drip. The rhythm converted to sinus tachycardia within minutes. The patient also had bronchopneumonia and parotitis as DKA precipitants. Recovery was uneventful with no arrhythmia recurrence.

Discussion: Hyperkalemia in DKA results from insulin deficiency, acidosis, and transcellular potassium shift. It can cause peaked T-waves and re-entrant arrhythmias including SVT. Vagal maneuvers are first-line for stable pediatric SVT. Calcium gluconate stabilizes cardiac membranes, while insulin and rehydration correct hyperkalemia.

Conclusion: In DKA with hyperkalemia, early ECG monitoring is essential to detect arrhythmias. Vagal maneuvers can be effective even without adenosine. Stepwise management is life-saving.

Keywords: Diabetic ketoacidosis, supraventricular tachycardia, hyperkalemia, adolescent, vagal maneuver.

INTRODUCTION

The incidence of type 1 diabetes mellitus (T1DM) continues to increase worldwide. In 2013, more than 500,000 cases of T1DM were reported in children under 15 years of age internationally. The Indonesian Pediatric Society has registered over 1,000 T1DM cases, with 1–10% complicated by diabetic ketoacidosis (DKA) (1,2). DKA remains the leading cause of morbidity and mortality in children with T1DM, with 15–70% of new-onset T1DM presenting in DKA (3,4). DKA is defined by the triad of hyperglycemia, ketosis, and metabolic acidosis (4,5). Its complications include electrolyte disturbances, acute kidney injury, cerebral edema, respiratory distress, and rarely, cardiac arrhythmias (6,7).

Although electrolyte abnormalities such as hyperkalemia are common in DKA, significant cardiac arrhythmias are uncommon and rarely reported. Most existing reports focus on ventricular tachycardia or asystole, while supraventricular tachycardia (SVT) secondary to hyperkalemia in pediatric DKA is exceptionally rare (8). Furthermore, the management of SVT in resource-limited settings where adenosine may be unavailable has not been well described. This case report aims to fill this research gap by providing a detailed chronological account of SVT development, serial ECG changes, and successful management using only a simple vagal maneuver with an ice pack, calcium gluconate, and insulin, without adenosine. The novelty of this report lies in its comprehensive documentation of ECG progression from peaked T-waves to SVT and back to sinus rhythm, as well as the identification of concurrent infections (bronchopneumonia and parotitis) as DKA triggers. We hypothesize that severe hyperkalemia (8.2 mmol/L) in the setting of DKA-induced acidosis and insulin deficiency directly triggered re-entrant SVT, and that prompt stabilization of cardiac membranes with calcium combined with vagal maneuver can terminate SVT even in the absence of adenosine. The objectives of this report are to describe this rare case, present serial ECG findings, discuss the pathophysiological link between hyperkalemia and SVT, and propose a practical management approach for resource-limited settings. The benefits include increasing clinical awareness and providing a reference for pediatricians and emergency physicians.

CASE ILLUSTRATION

Patient Identity and Initial Presentation

A 14-year-old Indonesian girl with a known diagnosis of type 1 diabetes mellitus (diagnosed approximately one year prior) presented to the emergency department of RSUD Puri Husada Tembilahan, a district hospital in Indragiri Hilir, on 27 July 2025 at 09:30 Western Indonesian Time. She was brought by her parents with the chief complaint of decreased consciousness that had begun the previous night. Her medical record number was 376859.

History of Present Illness

Three days before admission, the patient developed a fever, non-productive cough, and sore throat. One day prior to admission, she experienced intermittent vomiting (more than five episodes, vomiting only fluid) and swelling on the left side of her face near the ear, suggestive of parotitis. On the night of admission, she developed progressive shortness of breath, which was not influenced by weather or position. At home, she had been receiving regular insulin therapy consisting of Novorapid 3 times daily at 13 units per dose and Levemir once daily at 20 units per dose, which is not injected routinely.

Past Medical and Family History

The patient had no prior history of heart disease or arrhythmias. Her last outpatient pediatric control visit was on 17 September 2024. She had no known drug allergies. The family history was negative for sudden cardiac death or hereditary arrhythmias.

Physical Examination on Admission

On admission, the patient appeared acutely ill and was unconscious. Her Glasgow Coma Scale score was 9 out of 10 (E2, V3, M4), indicating response only to painful stimuli. Her vital signs showed a heart rate of 158 beats per minute (regular and tachycardic), blood pressure of 113/64 mmHg, respiratory rate of 36 breaths per minute with Kussmaul breathing pattern, and oxygen saturation of 98% while receiving 2 liters per minute of oxygen via nasal cannula. Her axillary temperature was 38.5°C. Head and neck examination revealed swelling of the right parotid region without meningeal signs. Chest examination showed symmetrical expansion with **ronchi**.

Cardiovascular examination revealed tachycardia without murmurs, and peripheral pulses were palpable. Abdominal examination was soft and non-tender with no hepatosplenomegaly. Extremities were cool with a capillary refill time greater than three seconds.

Initial Laboratory Findings and Diagnostic Studies

Table 1 summarizes the initial laboratory findings on admission (27 July 2025, approximately 10:00). These results confirmed severe DKA, life-threatening hyperkalemia, acute kidney injury, and the presence of ketonuria.

Table 1. Initial Laboratory Findings on Admission (27 July 2025)

Parameter	Result	Unit	Reference Range
Blood glucose	425	mg/dL	70–140
pH (ABG)	6.92	-	7.35–7.45
pCO ₂ (ABG)	16.5	mmHg	35–45
HCO ₃ (ABG)	3.4	mmol/L	22–26
Serum potassium	8.2	mmol/L	3.6–5.8
Serum sodium	124	mmol/L	135–145
Serum chloride	98	mmol/L	98–108
Urea	89	mg/dL	10–50
Creatinine	1.3	mg/dL	<0.9
Hemoglobin	13.6	g/dL	10.0–16.0
Leukocytes	7.3	×10 ³ /uL	5–10
Platelets	195	×10 ³ /uL	150–450
Urine glucose	+++	-	Negative
Urine ketones	+++	-	Negative
Urine leukocytes	11–12	/HPF	<5
Urine yeast cells	+++	-	Negative

Serial Electrocardiogram Findings

Serial electrocardiograms were performed on the day of admission. The first ECG at 11:52 showed sinus tachycardia at 154 beats per minute with tall, peaked T-waves in the precordial leads, consistent with severe hyperkalemia. There was also an abnormal ST segment. The second ECG at 12:32 showed persistent sinus tachycardia at 152 beats per minute with unchanged peaked T-waves. The third ECG at 13:12 demonstrated a change in rhythm to supraventricular tachycardia with narrow QRS complexes (80 ms) and an irregular rhythm; no visible P waves were observed, consistent with SVT, likely atrioventricular re-entrant tachycardia or atrial tachycardia. A fourth ECG performed immediately after interventions at approximately 13:30 showed conversion to sinus rhythm at 140 beats per minute with marked diminution of the peaked T-waves and no further arrhythmia.

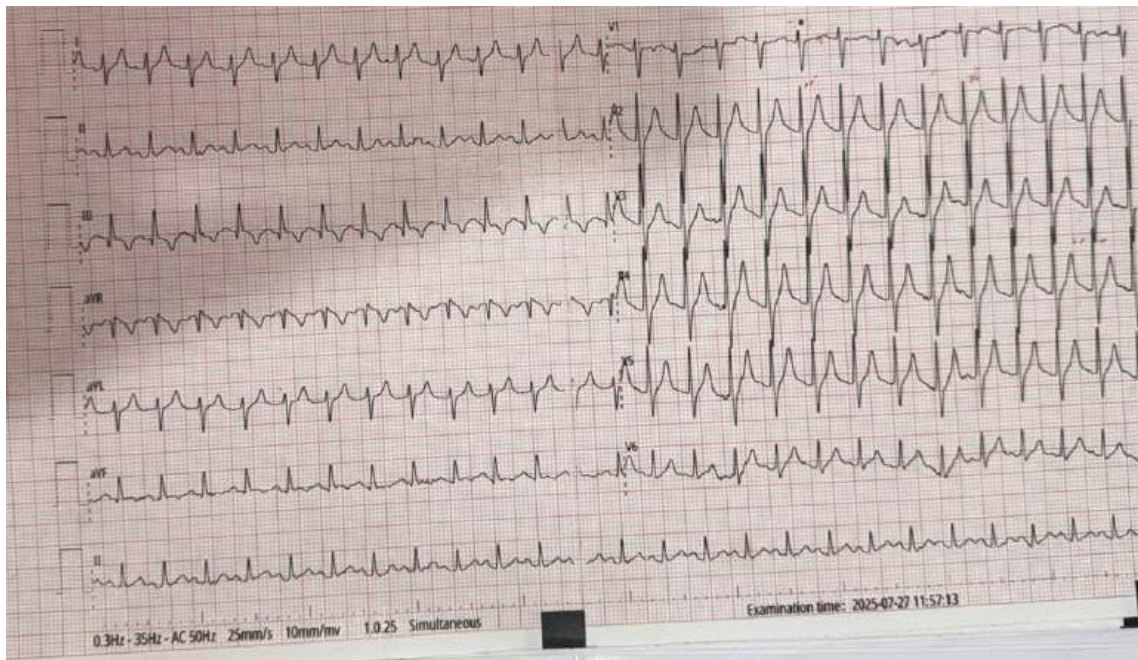


Figure 1. First ECG

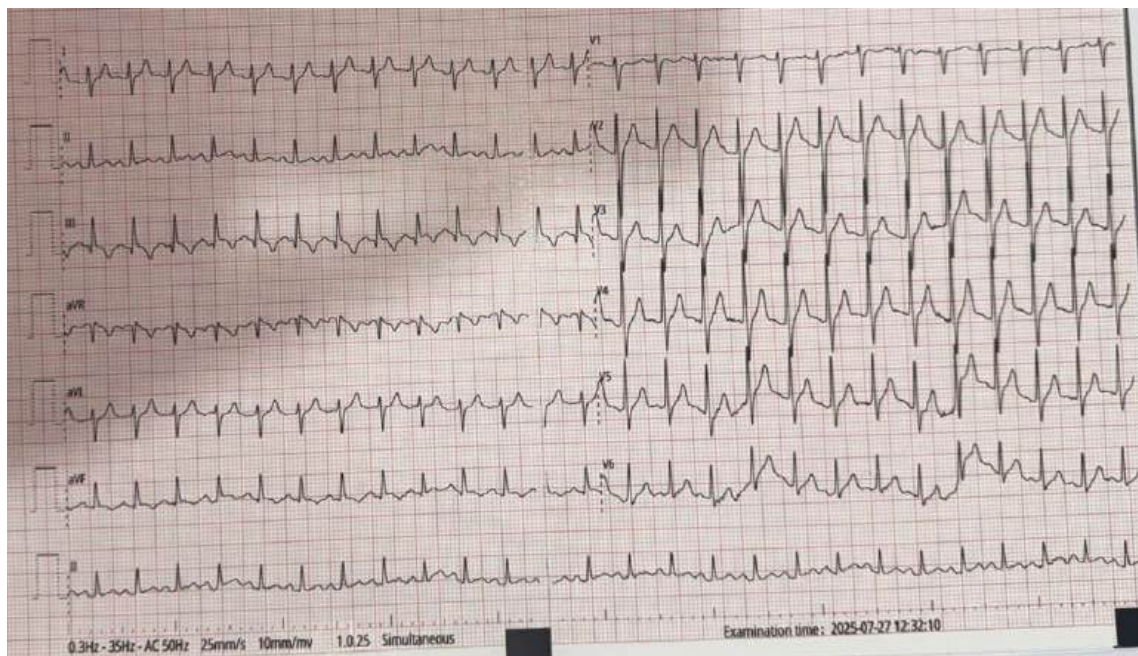


Figure 2. Second ECG

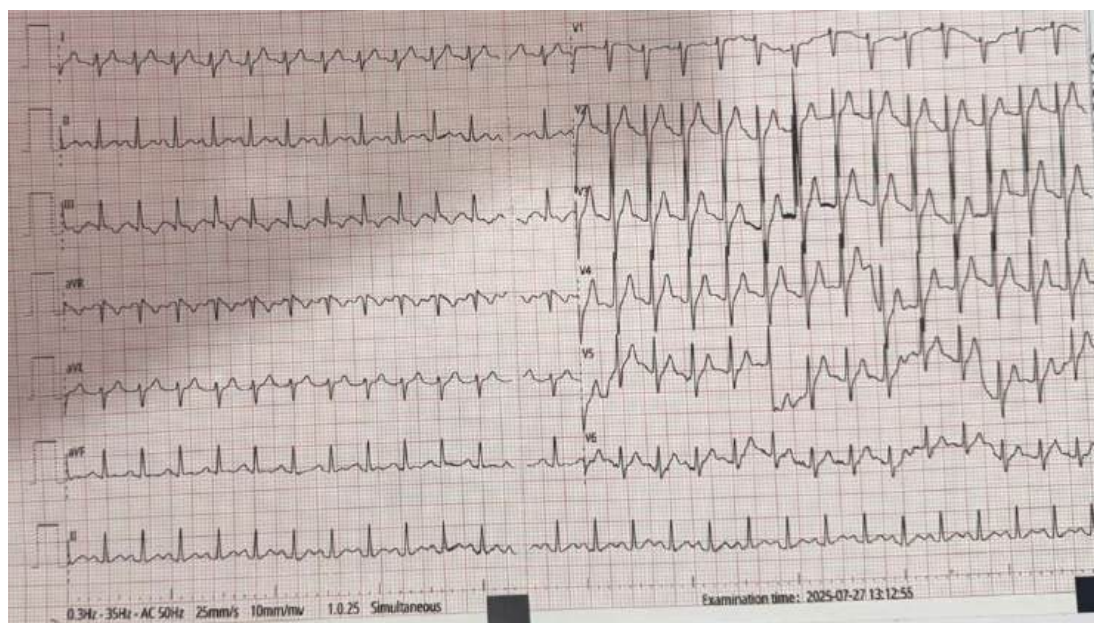


Figure 3. Third ECG

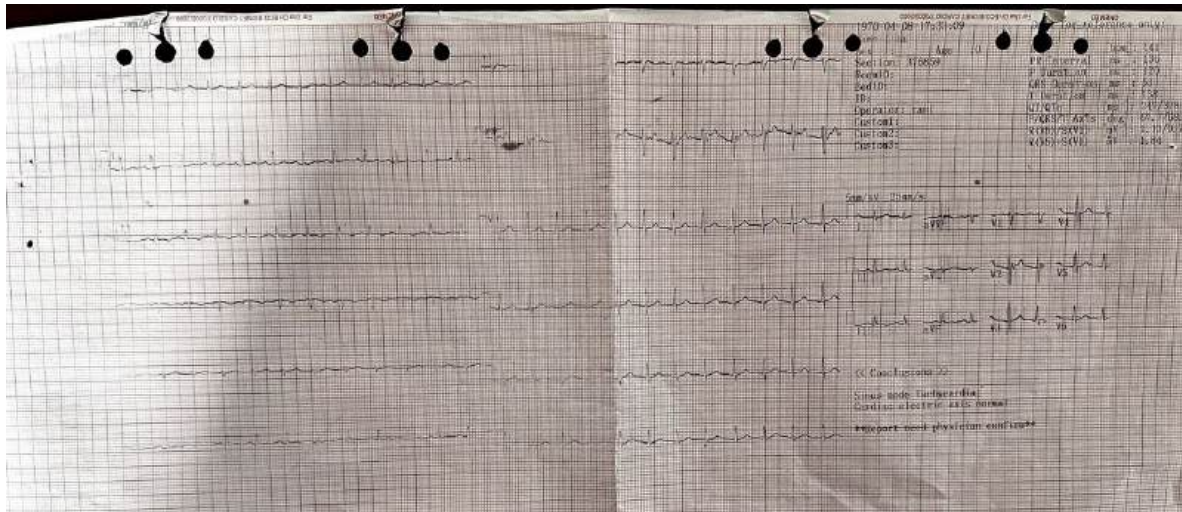


Figure 4. Fourth ECG

Chest X-Ray Findings

A chest X-ray performed on 27 July 2025 revealed infiltrates in the left lower lung field with no cardiomegaly and normal sinuses and diaphragm. The impression was bronchopneumonia.

Interventions and Treatment by Day

Day 1 (27 July 2025) – Emergency Management and Interventions

Upon admission, the patient was immediately given oxygen at 2 liters per minute via nasal cannula. Intravenous access was established. Because the hospital did not have adenosine available, a vagal maneuver was performed by applying an ice pack to the patient's face over the trigeminal nerve distribution for 15 to 20 seconds; this maneuver was repeated once. Within two minutes, the ECG showed conversion from SVT to sinus rhythm. Intravenous calcium gluconate 10% was administered as a slow injection at a dose of 1 mL/kg (14 mL total) to stabilize the cardiac membrane against hyperkalemia. An insulin drip was started at 0.1 units per kilogram per hour according to DKA management guidelines. Simultaneously, a resuscitation bolus of 20 mL per kilogram per hour of normal saline was given, and maintenance fluids were calculated for

rehydration over a 48-hour period. The patient was then transferred to the pediatric intensive care unit with an altered mental status.

Day 2 (28 July 2025) – Stabilization and Comprehensive Plan

On day 2, the patient remained somnolent with a Glasgow Coma Scale of 10 (responding to voice). A repeat arterial blood gas showed improvement with a pH of 7.29, although acidosis persisted. Serum potassium decreased to 5.8 mmol/L, which was still elevated but no longer in the critical range. The attending pediatric specialist established a comprehensive diagnosis of bronchopneumonia, parotitis, diabetic ketoacidosis, severe hyperkalemia, and a history of supraventricular tachycardia. Intravenous fluids of 0.9% normal saline were continued at 60 mL per hour. The continuous insulin drip was maintained, and broad-spectrum antibiotics (ceftriaxone 1 gram every 12 hours and gentamicin 80 mg every 12 hours) were initiated for pneumonia. Paracetamol was given for fever, and ranitidine was added for gastric protection. A transition plan was made to switch to subcutaneous insulin (Levemir and Novorapid) once the patient stabilized.

Day 3 (29 July 2025) – Identification of Additional Infections

On day 3, the patient's consciousness remained fluctuating with a Glasgow Coma Scale of 10, and she continued to have a fever of 38.0°C. Laboratory evaluation revealed worsening renal function with urea of 89 mg/dL and creatinine of 1.3 mg/dL, indicating acute kidney injury, most likely due to severe dehydration. A complete urinalysis still showed glucose (+++) and ketones (+++), and additionally identified a urinary tract infection (leukocytes 11–12 per high-power field) and candiduria (yeast cells +++). The laboratory diagnosis was expanded to include sepsis. Intravenous antibiotics were continued, and hydration was aggressively maintained.

Day 4 (30 July 2025) – Persistent Fever

On day 4, the patient experienced a high fever of up to 39.1°C. However, her level of consciousness began to show progressive improvement. Blood glucose monitoring continued hourly. Because of persistent fever despite ceftriaxone and gentamicin, the antibiotic regimen was escalated to meropenem due to concern for possible septicemia.

Day 5 (31 July 2025) – Significant Clinical Improvement

On day 5, a remarkable clinical improvement occurred. The patient's level of consciousness returned to full alertness with a Glasgow Coma Scale of 15 (compos mentis). Fever began to subside, although it remained fluctuating. Hemodynamic status was stable, and no further arrhythmias were detected on serial ECG monitoring.

Day 6 (1 August 2025) – Full Recovery and Discharge Planning

On day 6, the patient was fully alert and afebrile. Blood glucose levels were within normal ranges with continued insulin therapy. A repeat complete blood count, urea, creatinine, and electrolytes were all normalized. Serial ECG showed no recurrence of SVT or any other arrhythmia. The patient was evaluated as stable for discharge. Comprehensive education was provided to the family, including instructions for regular pediatric follow-up, medication adherence, self-monitoring of blood glucose, infection prevention, sick-day rules, and dietary management.

Serial Laboratory Follow-Up (Day 1 to Day 5)

Table 2 provides a day-by-day comparison of key laboratory parameters from admission through day 5, demonstrating the progressive correction of acidosis, hyperkalemia, and renal function.

Table 2. Serial Laboratory Follow-Up from Day 1 to Day 5

Parameter	Day 1 (27 Jul)	Day 2 (28 Jul)	Day 3 (29 Jul)	Day 4 (30 Jul)	Day 5 (31 Jul)	Reference Range
Blood glucose (mg/dL)	425	310	245	180	120	70–140
pH (ABG)	6.92	7.29	7.32	7.36	7.40	7.35–7.45
HCO ₃ (mmol/L)	3.4	12.5	16.2	19.8	23.0	22–26
Potassium (mmol/L)	8.2	5.8	4.5	4.0	3.8	3.6–5.8
Sodium (mmol/L)	124	130	135	138	140	135–145
Urea (mg/dL)	89	85	70	45	30	10–50
Creatinine	1.3	1.2	1.0	0.8	0.7	<0.9

(mg/dL)						
Leukocytes ($\times 10^3/\mu\text{L}$)	7.3	9.5	12.0	10.2	8.1	5–10
Urine ketones	+++	++	+	–	–	Negative

DISCUSSION

This case report describes a rare presentation of supraventricular tachycardia in an adolescent with severe diabetic ketoacidosis complicated by life-threatening hyperkalemia (8.2 mmol/L), acute kidney injury, bronchopneumonia, and parotitis. The successful management using only a vagal maneuver with an ice pack, intravenous calcium gluconate, and insulin—without adenosine—highlights important lessons for resource-limited settings.

The pathophysiology of hyperkalemia in DKA is multifactorial. Insulin deficiency reduces the activity of the Na^+/K^+ ATPase pump, impairing intracellular potassium shift (9). Additionally, the hyperosmolar state and metabolic acidosis promote the movement of potassium from the intracellular to the extracellular compartment in exchange for hydrogen ions (10). In this patient, the presenting serum potassium of 8.2 mmol/L was life-threatening. Although total body potassium is often depleted in DKA due to urinary losses, the transcellular shift leads to severe hyperkalemia at presentation, particularly when acute kidney injury from dehydration reduces renal potassium excretion (11). This patient's elevated urea and creatinine confirmed acute kidney injury, further exacerbating hyperkalemia.

The electrocardiographic manifestations of hyperkalemia follow a predictable progression as potassium levels rise. Early changes include tall, peaked T-waves with a narrow base, typically seen first in the precordial leads (12). As hyperkalemia worsens, the PR interval prolongs, QRS duration widens, P waves flatten and disappear, and eventually a sine wave pattern or ventricular fibrillation occurs. However, supraventricular tachycardia is an uncommon presentation. The proposed mechanism for SVT in hyperkalemia involves the slowing of intra-atrial and atrioventricular nodal conduction, creating dispersion of refractoriness and facilitating re-entry circuits (13). In this

patient, the transition from peaked T-waves to narrow-complex SVT on serial ECG supports hyperkalemia as the direct trigger. The absence of prior cardiac history makes underlying accessory pathways or structural heart disease unlikely.

The immediate management of SVT in a hemodynamically stable pediatric patient begins with vagal maneuvers (14). In this case, an ice pack applied to the face stimulates the trigeminal nerve and elicits the diving reflex, which increases vagal tone and slows atrioventricular nodal conduction. This maneuver has a reported success rate of 19–54% for terminating SVT in infants and children (15). Because adenosine was unavailable at this district hospital, the ice pack vagal maneuver was the first-line intervention and successfully converted SVT to sinus rhythm within minutes. This demonstrates that even simple, low-technology interventions can be life-saving when advanced drugs are not accessible.

Concurrently, the patient received intravenous calcium gluconate. Calcium does not lower serum potassium but rapidly stabilizes the cardiac myocyte membrane by raising the threshold potential, thereby reducing the risk of arrhythmias (16). This effect occurs within minutes and is essential for preventing progression to ventricular fibrillation. Insulin administration followed, which directly lowers serum potassium by driving potassium back into cells via Na^+/K^+ ATPase activation (17). The combination of calcium for immediate membrane stabilization and insulin for definitive potassium correction is the standard of care for hyperkalemic emergencies. Rehydration with normal saline also helps correct dehydration, improve renal perfusion, and lower blood glucose, all of which indirectly lower potassium.

The identification of bronchopneumonia and parotitis as DKA precipitants is clinically significant. Infection is the most common trigger for DKA in children with known diabetes, accounting for 30–50% of cases (18). The inflammatory response increases counterregulatory hormones (cortisol, glucagon, epinephrine), leading to accelerated ketogenesis and worsening hyperglycemia (19). In this patient, persistent fever despite initial antibiotics necessitated escalation to meropenem, which ultimately controlled the sepsis. The later findings of urinary tract infection and candiduria are also common in hyperglycemic states due to impaired leukocyte function and glycosuria (20).

The acute kidney injury observed in this patient was most likely prerenal in origin, resulting from severe dehydration due to osmotic diuresis from hyperglycemia. The rapid improvement in urea and creatinine with aggressive rehydration supports this mechanism. However, careful monitoring is required because cerebral edema is the most feared complication of DKA treatment, particularly in children and adolescents (21). This patient did not develop signs of cerebral edema, and her neurological status progressively improved.

Serial laboratory follow-up demonstrated the expected response to treatment. By day 2, pH had improved to 7.29 and potassium had fallen to 5.8 mmol/L. By day 5, all parameters had normalized, including blood glucose, electrolytes, and renal function. The absence of arrhythmia recurrence on serial ECG confirmed that the SVT was a transient complication directly related to the acute metabolic derangement rather than an underlying cardiac condition.

One limitation of this report is the absence of echocardiography to definitively rule out structural heart disease or an accessory pathway. However, the patient's lack of prior symptoms and complete resolution without antiarrhythmic medication make an underlying substrate less likely. Additionally, electrophysiologic studies were not available at this facility.

CONCLUSION

This case demonstrates that severe hyperkalemia in the setting of diabetic ketoacidosis can trigger supraventricular tachycardia in an adolescent without prior cardiac disease. Early recognition through serial electrocardiography is essential, as the progression from peaked T-waves to arrhythmia can be rapid. In resource-limited settings where adenosine may be unavailable, a simple vagal maneuver using an ice pack applied to the face can be effective for terminating supraventricular tachycardia. Combined with intravenous calcium gluconate for immediate cardiac membrane stabilization and insulin for definitive correction of hyperkalemia, this approach is life-saving. The underlying precipitant of diabetic ketoacidosis, in this case bronchopneumonia and parotitis, must be identified and treated aggressively. We recommend that all children with diabetic ketoacidosis undergo early electrocardiographic monitoring and serial electrolyte measurement, and

that clinicians in resource-limited settings be trained in vagal maneuvers as a first-line therapy for stable supraventricular tachycardia.

Suggestion for future practice: District hospitals should develop protocols for the management of hyperkalemic emergencies that include immediate ECG assessment, administration of calcium gluconate, insulin infusion, and vagal maneuver protocols. Transfer to a pediatric intensive care unit should be arranged for children with arrhythmias or altered mental status.

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