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When Insulin Must Wait: The Role of Renal Replacement Therapy in Refractory Hypokalemia During Severe Diabetic Ketoacidosis

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Data Collection B
Statistical Analysis C
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Patient: Male, 37-year-old
Final Diagnosis: Diabetic ketoacidosis (DKA) • refractory hypokalemia • severe sepsis
Symptoms: Abdominal pain • agitation • altered mental status • Kussmaul respirations • shortness of breath
Clinical Procedure: —
Specialty: Critical Care Medicine • Endocrinology and Metabolic • Nephrology

Objective: Unusual clinical course

Background: Diabetic ketoacidosis (DKA) is a common life-threatening metabolic emergency characterized by hyperglycemia, high-anion-gap metabolic acidosis, and ketosis. Severe hypokalemia before insulin therapy is uncommon but may complicate management and delay treatment.

Case Report: A 37-year-old man presented to the emergency department with a 2-day history of generalized abdominal pain and progressive shortness of breath. He was drowsy and tachypneic, with Kussmaul respirations. Laboratory evaluation revealed severe DKA, acute kidney injury, and severe hypokalemia. Insulin therapy was initially withheld according to the DKA protocol. Intravenous potassium supplementation failed to achieve sustained correction, resulting in repeated interruptions of insulin therapy. Due to refractory hypokalemia, an emergency 2-hour session of intermittent hemodialysis was performed, followed by continuous renal replacement therapy (CRRT). During CRRT, serum potassium increased, serum creatinine decreased, metabolic acidosis improved, and the anion gap closed. Insulin therapy was resumed without further interruption. However, the clinical course was complicated by severe septic shock, disseminated intravascular coagulation, and catastrophic intracerebral hemorrhage, ultimately resulting in death.

Conclusions: Severe hypokalemia at presentation in DKA poses a critical therapeutic challenge because insulin therapy must be withheld until the electrolyte abnormality is corrected. In rare cases of refractory hypokalemia, CRRT may serve as an adjunctive strategy to facilitate metabolic stabilization and safe DKA management. This case adds to the limited literature and highlights the need for further studies to clarify the role and effectiveness of this approach. Biochemical resolution of DKA may not correspond to clinical recovery, and critically ill patients warrant continuous monitoring.

Keywords: Hypokalemia • Diabetic Ketoacidosis • Renal Replacement Therapy • Continuous Renal Replacement Therapy
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Introduction

Diabetic ketoacidosis (DKA) is a common endocrine emergency managed with insulin, fluid resuscitation, and electrolyte correction [1]. Severe hypokalemia is a recognized complication; however, refractory hypokalemia that delays insulin initiation and requires renal replacement therapy is uncommon [2,3].

We report a case of severe DKA complicated by refractory hypokalemia and hypophosphatemia that delayed insulin initiation, highlighting the role of renal replacement therapy as a bridging strategy to correct electrolyte abnormalities, facilitate safe insulin administration, and achieve metabolic stabilization. Despite biochemical resolution, the patient's course was ultimately complicated by critical illness and catastrophic intracerebral hemorrhage.

Although renal replacement therapy for refractory hypokalemia in DKA has been reported [3-5], this case is distinguished by the sequential use of emergency intermittent hemodialysis followed by continuous renal replacement therapy with a 4 mEq/L potassium bath in a patient with concurrent acute kidney injury and extreme acidemia (pH 6.93). It also highlights divergence between biochemical resolution of DKA and an ultimately fatal clinical outcome.

Case Report

A 37-year-old man presented to the emergency department with a 2-day history of generalized abdominal pain and progressive shortness of breath. His brother provided the history, which comprised worsening mental status and inability to ambulate. The patient had been evaluated at an outside clinic 2 days earlier, where he received intravenous fluids and was discharged without improvement. His history suggested that he had previously been healthy and was not taking any medications. He had a history of heavy alcohol use, consuming approximately one 750-mL bottle of tequila daily; he reportedly had stopped drinking 4 months before presentation.

Examination revealed that the patient was drowsy and agitated. Signs of volume depletion and Kussmaul respirations were noted. His temperature was 33.7 °C [passive external rewarming was applied], blood pressure was 106/53 mm Hg, heart rate was 80 beats/min, respiratory rate was 26 breaths/min, and SpO_2 was 100% on room air. His body mass index was 21.3 kg/m^2. Lung auscultation and cardiac examination findings were normal, the abdomen was soft and nontender, and skin examination showed scattered boils without surrounding cellulitis.

Initial laboratory testing revealed severe hyperglycemia (serum glucose, 500 mg/dL; point-of-care glucose, 422-464 mg/dL), hyponatremia (128 mmol/L), and severe hypokalemia (2.6 mmol/L), with greatly decreased serum bicarbonate (< 10 mmol/L) and an elevated anion gap (> 16). Serum β-hydroxybutyrate was strongly elevated at over 9 mmol/L, confirming clinically significant ketosis. Venous blood gas analysis showed severe metabolic acidosis, with a pH of 6.93, bicarbonate of 2.8 mmol/L, and compensatory hypocapnia (pCO_2, 13.7 mm Hg). Additionally, renal function tests indicated acute kidney injury, with a blood urea nitrogen level of 50 mg/dL, creatinine level of 2.47 mg/dL, and estimated glomerular filtration rate of 33.6 mL/min/1.73 m^2. Serum osmolality was elevated at 309 mOsm/kg. Lactate was mildly elevated at 1.81 mmol/L, indicating no substantial contribution from lactic acidosis. Serum magnesium and phosphorus levels were within normal limits on admission (2.3 and 3.2 mg/dL, respectively). Hemoglobin A1c was considerably elevated at over 14.0%, consistent with poor chronic glycemic control. Notably, serum lipase was greatly elevated at 2887 U/L, and the triglyceride level was 175 mg/dL; however, abdominal imaging revealed no acute abnormalities or evidence of pancreatitis.

Complete blood count revealed a white blood cell count of 10.0×10^9/L with neutrophil predominance, hemoglobin of 13.8 g/dL, and thrombocytosis (platelet count, 560×10^9/L); there was no clear evidence of infection. Procalcitonin was low at 0.28 ng/mL. Urinalysis demonstrated glucosuria and ketonuria (3+ each), without evidence of urinary tract infection. Blood and urine cultures obtained on admission showed no growth. Collectively, these findings were consistent with severe DKA complicated by acute kidney injury and severe hypokalemia. The initial laboratory findings are summarized in **Table 1**.

The patient received 4 L of intravenous fluids in the emergency department on Day 0, along with 2 intravenous sodium bicarbonate boluses and potassium replacement. Despite initial resuscitation and electrolyte replacement, he remained drowsy and became combative during central venous catheter placement. Given the persistent altered mental status, severe metabolic acidosis, and hypokalemia (potassium, 2.6 mmol/L), the patient was transferred to the intensive care unit, where he required endotracheal intubation for airway protection.

DKA management was initiated according to protocol; however, insulin therapy was initially withheld due to severe hypokalemia. Empiric broad-spectrum antimicrobial therapy, including vancomycin and piperacillin-tazobactam, was initiated, along with vasopressor support, because of concern for evolving sepsis of unclear origin. Although scattered skin boils were noted on presentation, there was no surrounding cellulitis, abscess formation, or evidence of invasive soft-tissue infection, making them an unlikely source of sepsis.

By the following day (Day 1), the hypokalemia had proven refractory, with serum potassium levels persistently ranging from

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Table 1. Initial laboratory findings.

Laboratory test	Result	Reference range
Chemistry		
Serum glucose	500 mg/dL	70-110 mg/dL
Point-of-care glucose	422-464 mg/dL	70-110 mg/dL
Hemoglobin A1c	> 14.0%	< 5.7%
Sodium	128 mmol/L	135-145 mmol/L
Potassium	2.6 mmol/L	3.5-5.1 mmol/L
Chloride	102 mmol/L	98-107 mmol/L
Bicarbonate (CO_2)	< 10 mmol/L	22-29 mmol/L
Anion gap	> 16	8-16
Blood urea nitrogen	50 mg/dL	7-20 mg/dL
Creatinine	2.47 mg/dL	0.6-1.3 mg/dL
Estimated glomerular filtration rate	33.6 mL/min/1.73 m^2	> 60 mL/min/1.73 m^2
Serum osmolality	309 mOsm/kg	275-295 mOsm/kg
β-Hydroxybutyrate	> 9 mmol/L	< 0.4 mmol/L
Lactate	1.81 mmol/L	0.5-2.2 mmol/L
Magnesium	2.3 mg/dL	1.5-2.5 mg/dL
Phosphorus	3.2 mg/dL	2.1-4.3 mg/dL
Lipase	2887 U/L	13-60 U/L
Triglycerides	175 mg/dL	< 150 mg/dL
Venous blood gas		
pH	6.93	7.32-7.43
pCO_2	13.7 mm Hg	38-50 mm Hg
Bicarbonate	2.8 mmol/L	22-26 mmol/L
Base excess	-28.2 mmol/L	-2 to +2 mmol/L
Complete blood count		
White blood cell count	10.0 × 10^9/L	4.0-11.0 × 10^9/L
Hemoglobin	13.8 g/dL	13.5-17.5 g/dL
Hematocrit	40.3%	38.8%-50.0%
Platelet count	560 × 10^9/L	150-400 × 10^9/L
Urinalysis		
Glucose	3+	Negative
Ketones	3+	Negative
Leukocyte esterase	Negative	Negative
Nitrites	Negative	Negative

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Table 2. Trends in selected laboratory values.

Time point	Potassium (mmol/L)	Phosphorus (mg/dL)	Anion gap (mmol/L)	pH	HCO_3^- (mmol/L)	pCO_2 (mm Hg)
Day 0 (ED)	2.6	3.2	> 16	6.93 (VBG)	2.8	13.7
Day 1 (early)	2.2-3.3	< 1.0	13-15	6.99-7.13 (ABG)	5.1-9.4	21-29
Day 1 (late)	2.5-2.8	< 1.0-1.3	> 14-18	7.11-7.20 (ABG)	8.3-13.8	26-35
Day 2 (early)	4.1-5.3	3.0-3.1	Closed	7.29-7.35 (ABG)	13.8-17.3	28-32
Day 2 (late)	3.7-4.3	3.1-3.5	Normal	7.35-7.36 (ABG)	17.9-19.7	31-36

Abbreviations: ABG, arterial blood gas; ED, emergency department; VBG, venous blood gas.

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2.2 to 2.6 mmol/L; it was further complicated by the development of hypophosphatemia. This change required aggressive electrolyte replacement, encompassing repeated intravenous potassium boluses and continuous infusions totaling more than 300 mEq within the first 48 hours, including potassium acetate. Phosphate was replaced with sodium phosphate and multiple doses of potassium phosphate. Magnesium and calcium were also replaced.

A reduced-dose insulin infusion (0.05 units/kg/h) was cautiously initiated after the serum potassium level increased to 3.3 mmol/L. However, it was repeatedly interrupted because the potassium level again declined to 2.2 mmol/L despite aggressive ongoing replacement. In the setting of refractory hypokalemia, acute kidney injury, and persistent metabolic derangements, an emergency 2-hour session of intermittent hemodialysis was initiated at 17: 30 on Day 1, when the serum potassium level was 2.5 mmol/L. After hemodialysis and ongoing potassium replacement, the serum potassium level was 2.8 mmol/L at 20: 30 on Day 1. At 21: 00, an additional 200 mEq of intravenous potassium acetate in 500 mL of 5% dextrose (in water) was administered, infused at 40 mEq/hour over 5 hours. Because insulin therapy could not be safely continued in the setting of profound hypokalemia, continuous renal replacement therapy (CRRT) with a 4 mEq/L potassium bath was initiated around 00: 00 on Day 2; intravenous potassium replacement was maintained. The serum potassium immediately before CRRT initiation was 2.8 mmol/L. The insulin infusion was resumed at a reduced dose of 0.05 units/kg/h at approximately 02: 00 on Day 2, with close metabolic monitoring. During CRRT, the serum potassium level increased from 2.8 to 4.1 mmol/L, serum creatinine decreased from 2.47 to 1.77 mg/dL, pH improved from 7.11 to 7.35, bicarbonate increased from 8.3 to 17.9 mmol/L, and the anion gap closed. After resolution of the anion-gap metabolic acidosis, the patient was transitioned from intravenous to subcutaneous insulin and experienced no further hypokalemia. **Table 2** summarizes trends in selected laboratory values.

Despite the closure of the anion gap on Day 2, the patient's clinical course was complicated by progressive septic shock and multiorgan failure. He developed severe acute respiratory distress syndrome requiring maximal ventilatory support and escalating vasopressor therapy. Inflammatory markers substantially increased: procalcitonin rose from 0.28 ng/mL on admission to more than 200 ng/mL, and C-reactive protein remained persistently elevated. The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) antigen test was negative on admission. However, a respiratory pathogen panel obtained concurrently later showed positivity for SARS-CoV-2; this result became available 1 week after admission. Chest computed tomography demonstrated extensive bilateral ground-glass opacities, consolidations with cavitary lesions, bronchiectasis, and tree-in-bud nodularity, as well as a small right pleural effusion, findings consistent with severe multifocal infectious pneumonia and bronchiolitis. His course was further complicated by disseminated intravascular coagulation; worsening thrombocytopenia was noted and schistocytes were evident on peripheral blood smear assessment. Neurologic deterioration prompted neuroimaging, which revealed multifocal intracerebral hemorrhages with cerebral edema and subsequent herniation. Despite aggressive supportive care, including broad-spectrum antimicrobial therapy and ventilatory and hemodynamic support, the patient died of catastrophic intracerebral hemorrhage on hospital day 18.

Discussion

This case illustrates the challenges of managing severe DKA complicated by refractory hypokalemia and hypophosphatemia, which delayed insulin initiation and required renal replacement therapy. Although metabolic parameters improved upon closure of the anion gap, the patient's clinical course was complicated by critical illness and catastrophic intracerebral hemorrhage.

Hypokalemia, defined as a serum potassium level of less than 3.6 mmol/L, is common during the treatment of DKA. However, severe hypokalemia at presentation before insulin therapy is

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exceedingly uncommon [6]. Although insulin deficiency, hyperosmolality, and acidemia typically maintain normal or elevated serum potassium concentrations by promoting a shift of potassium from the intracellular to the extracellular compartment, patients with DKA have pronounced total body potassium depletion due to osmotic diuresis. Therefore, severe hypokalemia at presentation reflects profound potassium depletion and severe metabolic derangement [7].

Several factors likely contributed to the severe hypokalemia in the present case. DKA itself is associated with substantial urinary potassium losses due to osmotic diuresis, often compounded by reduced oral intake and possible gastrointestinal losses [8]. Chronic heavy alcohol use may have further exacerbated potassium depletion through poor nutritional intake and possible intracellular magnesium deficiency, which can impair effective potassium replacement. Although the serum magnesium level was normal on admission, the potential for intracellular depletion cannot be excluded. Subsequent development of hypophosphatemia further indicated substantial electrolyte depletion, warranting concurrent replacement of magnesium, phosphorus, and potassium.

Few case reports have described refractory hypokalemia in DKA requiring renal replacement therapy, including CRRT modalities such as continuous venovenous hemodialysis [3,4]. In our patient, potassium levels remained unstable despite repeated boluses and continuous infusions totaling over 300 mEq during the first 48 hours. Given that insulin promotes an intracellular shift of potassium, repeated interruptions of insulin therapy were necessary to prevent severe hypokalemia and associated arrhythmias. Persistent severe hypokalemia and acute kidney injury prompted initiation of emergency intermittent hemodialysis. Because the serum potassium level remained low afterward, CRRT with a 4 mEq/L potassium bath was initiated to facilitate correction of the hypokalemia.

CRRT was used as part of a combined rescue strategy associated with gradual metabolic stabilization. During this period, insulin therapy was resumed without further interruption, and the anion gap subsequently closed. Although CRRT in DKA can be challenging because bicarbonate-containing dialysate and insulin clearance during hemofiltration may alter glucose and electrolyte homeostasis, these effects can be managed with careful insulin titration, a lower initial insulin infusion rate, and close metabolic monitoring [3].

In our patient, aggressive potassium replacement (> 300 mEq within the first 48 hours) and intermittent hemodialysis failed to achieve sustained correction of hypokalemia; reduced-dose insulin therapy required repeated interruption. CRRT with a 4 mEq/L potassium bath served as a component of a combined rescue strategy associated with subsequent correction of refractory hypokalemia, while also providing renal support for acute kidney injury and severe metabolic acidosis. During this period, insulin therapy was resumed without further interruption, and the anion gap closed. Consistent with previous reports, these findings suggest that CRRT may be regarded as an individualized adjunct when standard measures are unsuccessful [5,9]. However, the effects of CRRT cannot be fully distinguished from those of the other interventions used in this single case.

Despite closure of the anion gap, biochemical improvement did not result in clinical recovery. The patient subsequently developed severe septic shock in the setting of COVID-19-associated multifocal pneumonia and acute respiratory distress syndrome, disseminated intravascular coagulation, and catastrophic intracerebral hemorrhage, ultimately resulting in death.

Conclusions

Refractory hypokalemia can delay insulin therapy in severe DKA. In selected cases, renal replacement therapy may serve as an adjunctive strategy to correct electrolyte abnormalities, facilitate safe insulin administration, and achieve metabolic stabilization. However, available evidence remains limited, highlighting the need for further studies to better define the role and appropriate use of this approach in critically ill patients. Biochemical resolution of DKA does not guarantee a favorable clinical outcome, underscoring the importance of continuous monitoring.

Institution Where the Work Was Performed

Department of Internal Medicine, New York Medical College at Saint Michael's Medical Center, Newark, NJ, USA.

Patient Consent

Written informed consent was obtained from the patient's next of kin for publication of this case report.

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