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Back to Life: A Remarkable Recovery from Profound Diabetic Ketoacidosis with Coma and Multisystem Organ Dysfunction

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ABSTRACT: Diabetic ketoacidosis (DKA) is one of the most serious, acute, and life-threatening hyperglycemic emergencies in individuals with type 1 diabetes (T1D). Severe DKA may be accompanied by profound metabolic acidemia and altered mental status, and critically ill patients may develop acute kidney injury, hemodynamic instability, and other systemic complications requiring intensive care. Early recognition and prompt multidisciplinary management are important to reduce the risks of complications without affecting recovery from hyperglycemic crisis. We report a 45-year-old woman with poorly controlled T1D and history of recurrent admissions for DKA who presented to the Emergency Department of Al-Sobh Hospital, Jeddah, Saudi Arabia, after multiple episodes of vomiting followed by loss of consciousness. On admission, she was comatose with a Glasgow Coma Scale score of 3/15, severe dehydration, tachypnea, tachycardia, and profound hypotension (77/42 mmHg). Her initial venous blood gas showed a pH of 6.75 and a bicarbonate concentration of 5 mmol/L, hyperglycemia (>600 mg/dL), with acute kidney injury with serum creatinine of 2.3 mg/dL, and elevated inflammatory markers. Early aggressive resuscitation included intravenous fluids, insulin therapy, electrolyte correction, vasopressors, and intravenous sodium bicarbonate. Acidosis gradually resolved, vasopressors successfully discontinued, renal function recovered, the patient was extubated after 5 days of mechanical ventilation and was discharged with stable hemodynamics and good neurological status.

This case highlights the need for individualized and multidisciplinary critical care management in profound DKA complicated by coma, hemodynamic collapse, acute kidney injury, respiratory failure, and major electrolyte disturbances, and illustrated the selective use of sodium bicarbonate as an adjunct in the setting of profound acidemia and hemodynamic instability.

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INTRODUCTION

Diabetic ketoacidosis (DKA) is an acute complication of diabetes mellitus characterized by hyperglycemia, high anion gap metabolic acidosis, and accumulation of ketone bodies **(American Diabetes Association Professional Practice Committee, 2024)**. From a pathophysiological standpoint, insulin deficiency leads to increased lipolysis and hepatic production of ketone bodies, with consequent accumulation of organic acids and development of metabolic acidosis **(Wolfsdorf et al., 2018)**. Respiratory compensation, through hyperventilation, represents a fundamental survival mechanism, allowing a reduction in pCO_2 and a partial rebalancing of pH **(Wolfsdorf et al., 2018)**.

In addition to metabolic derangements, DKA is associated with significant fluid and electrolyte disturbances, including dehydration, sodium imbalance, and total body potassium depletion, even in the presence of normal or elevated serum potassium levels at presentation. These alterations require careful and dynamic correction, as inappropriate or overly rapid management may result in serious complications **(Elendu et al., 2023)**. Clinical practice is often influenced by the numerical severity of acidemia, with a tendency to pursue rapid correction of pH values through aggressive interventions. However, this approach may overlook the underlying physiological compensatory mechanisms, particularly respiratory compensation, which plays a crucial role in maintaining acid-base balance **(Jung et al., 2026)**.

This case describes a critically ill patient with severe DKA with coma, profound hypotension, acute kidney injury, respiratory failure, marked electrolyte disturbances, and subsequent multisystem complications. The patient demonstrated progressive neurological, hemodynamic, renal, and respiratory recovery following comprehensive multimodal critical care, including individualized intravenous sodium bicarbonate administration during profound acidemia. This case highlights the complexity of managing exceptionally severe DKA and the need for individualized critical care strategies when profound acidemia is accompanied by hemodynamic and multisystem dysfunction.

Case Presentation

A 45-year-old woman with type 1 diabetes mellitus, epilepsy, chronic immobility with residual left-sided weakness, chronic Foley catheterization, and recurrent DKA admissions (approximately five annually) was brought by ambulance to the Emergency Department of Al-Sobh Hospital, after being found unresponsive at home.

According to her family, she had multiple episodes of vomiting followed by loss of consciousness one day before admission. During transport, her random blood glucose was 800 mg/dL, and intravenous normal saline was initiated.

- **Initial Assessment and Clinical Findings**

On arrival, the patient was comatose, severely dehydrated, tachypneic, tachycardic (HR 105 bpm), and hypotensive (BP 77/42 mmHg), but afebrile. Oxygen saturation was 98% on room air. Neurological examination showed a GCS score of 3/15, flaccidity, and bilaterally reactive pupils.

- **Laboratory findings:**

Venous blood gas analysis showed severe acidosis (pH 6.75, HCO_3^- 5 mmol/L). Laboratory tests revealed marked hyperglycemia , hyperkalemia (6 mmol/L), elevated creatinine (2.3 mg/dL), hypernatremia (155 mmol/L), and elevated inflammatory markers (CRP 10 mg/dL; procalcitonin 19 ng/mL). Pancreatic enzymes were also elevated (amylase 933 U/L; lipase 695 U/L). **(Table 1)**

Table 1: Initial Laboratory Findings

Parameter	Result
Blood glucose	"HI" reading (>600 mg/dL)
Potassium	6 mmol/ L
Sodium	155 mmol/ L
C-reactive protein (CRP)	10 mg/dL

Serum amylase	933 U/L
Serum lipase	695 U/L
Creatinine	2.3 mg/dL

Severe diabetic ketoacidosis with coma, hypovolemic and septic shock, acute kidney injury, hyperosmolar hypernatremia, and acute respiratory failure.

Management:

Central venous access was obtained through the right internal jugular vein, and aggressive resuscitation was initiated in the emergency department and ICU. Management included intravenous fluids, insulin infusion according to the DKA protocol, electrolyte correction, vasopressors (norepinephrine, adrenaline, and vasopressin), endotracheal intubation, and mechanical ventilation. Broad-spectrum antibiotics were started, and sepsis workup with microbiological cultures was performed.

Sodium bicarbonate was administered cautiously as intermittent intravenous doses for severe acidemia (arterial pH =6.75), aiming to raise pH above 6.9–7.0 and improve tissue perfusion and hemodynamic stability rather than achieve complete normalization **(Eledrisi and Elzouki, 2020)**.

Because of profound acidosis and refractory shock, bicarbonate was administered early during resuscitation, with frequent monitoring of blood gases, electrolytes, potassium, and anion gap.

Serial blood gas analyses demonstrated progressive correction of the initial severe acidemia during the ICU course. **(Table 2)**

Table 2: Serial blood gases

Time point	**pH**	**PCO_2**	**HCO_3^-**	**Lactate**
Admission	6.75	**16**	5	**5**
Day 2 ICU	7.17	23	10.5	4.5
Day 3	7.42	41	26	**2**
Day 5	7.46	38	26	1.8

The patient required mechanical ventilation for 5 days and was successfully weaned from vasopressors after 6 days.

ICU Course:

During the ICU stay, the patient developed oliguric acute kidney injury and recurrent hypokalemia, hypophosphatemia, and hypernatremia, requiring close monitoring and correction. **(Table 3)**

Table 3: Initial and Follow up Laboratory Findings

Parameter	**Admission**	**Follow-up/Outcome**
Serum creatinine (mg/dL)	2.3	0.7
Serum sodium (mmol/L)	155	134
Procalcitonin (ng/mL)	19	0.2

- **Renal function and serum electrolytes:**

Renal function improved progressively, with creatinine decreasing from 2.3 to 0.7 mg/dL, while serum sodium gradually decreased from 155 to 134 mmol/L. (**Table 4**)

- **Respiratory complications:**

Respiratory status improved, allowing transition to CPAP on day 3 and extubation after 5 days. Mild post-extubation stridor resolved with dexamethasone and adrenaline nebulization. (**Table 4**)

Table 4: Complications During ICU Stay

Complication	Clinical Course
Recurrent hypokalemia	Required frequent potassium replacement
Hypophosphatemia	Corrected with phosphate supplementation
Persistent hypernatremia	Gradually corrected with fluid management
Acute kidney injury with oliguria	Improved with resuscitation and supportive care
Recurrent severe hypoglycemia	Required D25 infusion, D50 boluses, and insulin adjustment
Elevated pancreatic enzymes with ileus-like symptoms	Managed conservatively with rectal decompression and gradual refeeding
Post-extubation mild stridor	Improved with dexamethasone and adrenaline nebulization

- **Gastrointestinal and metabolic complications:**

The patient developed abdominal distension, bilious vomiting, and elevated pancreatic enzymes, suggestive of ileus. Rectal decompression was performed, and oral feeding was resumed gradually.

After resolution of DKA, she developed recurrent severe hypoglycemia requiring dextrose infusion and boluses, insulin adjustment, and nutritional consultation.

- **Infection:**

Broad-spectrum antibiotics were initiated. Procalcitonin decreased from 19 to 0.2 ng/mL, while cultures and viral screening were negative. Vancomycin was discontinued because of elevated serum levels.

- **Hematological Course**

Hemoglobin decreased to 7.3 g/dL and packed RBCs transfusion was administered. Occult blood testing was negative.

- **Outcome and follow-up**

The patient demonstrated progressive neurological and hemodynamic improvement during the ICU stay. Vasopressor support was discontinued, renal function returned to normal, metabolic acidemia resolved, and oral feeding was gradually tolerated.
The patient was discharged 7 days after admission in a hemodynamically stable condition, with a GCS score of 15/15 and oxygen saturation of 98% on room air. Glycemic control was stable on basal and sliding-scale insulin. Follow-up was arranged with internal medicine, psychiatry, and clinical nutrition.

DISCUSSION

This case demonstrates an exceptionally severe presentation of DKA characterized by profound metabolic acidemia, coma, severe hypotension, acute kidney injury, respiratory failure, major electrolyte disturbances, and a prolonged requirement for critical care support. The initial pH of 6.75 and bicarbonate of 5 mmol/L fulfill the biochemical criteria for severe DKA described in the 2024 consensus report, while the GCS of 3/15 and requirement for intensive care further demonstrate the severity of the presentation.

Because the patient presented with a pH of 6.75, intravenous sodium bicarbonate was administered as an adjunct during initial resuscitation. Although routine bicarbonate therapy is not recommended in DKA, the 2024 consensus allows its consideration in severe acidemia with pH <7.0. Importantly, bicarbonate was used in this case as an individualized adjunct to fluids, insulin, hemodynamic support, and ventilation rather than as the primary treatment. 2026 meta-analysis of eight studies involving 646 patients found no significant improvement in acidosis resolution, pH, or potassium outcomes with bicarbonate therapy, while evidence in extreme acidemia remains limited **(Omayer et al., 2026)**.

The treatment of this patient was directed toward correction of the underlying metabolic disturbance while simultaneously supporting the affected organ systems. Initial resuscitation included intravenous fluid administration to restore effective circulating volume and improve tissue and renal perfusion, together with intravenous insulin according to the DKA protocol to suppress ongoing lipolysis and ketone production and facilitate glucose utilization **(Evans, 2019)**. Electrolyte abnormalities were monitored closely and corrected dynamically **(Bulloch et al., 2024)**.

Sodium bicarbonate was administered intravenously during the initial management of the profound acidemia as an adjunct to the primary treatment **(von Groote et al., 2026)**. In addition, norepinephrine, adrenaline, and vasopressin were used to support arterial pressure and maintain systemic perfusion during the period of severe hypotension. Because of coma, gasping respiration, and respiratory compromise, the patient underwent endotracheal intubation and mechanical ventilation. Broad-spectrum antimicrobial therapy was also initiated because infection was considered as a possible contributing or precipitating factor **(Umpierrez et al., 2024)**. This multimodal approach is consistent with current DKA management principles, in which correction of hypovolemia, suppression of ketogenesis with insulin, replacement of electrolytes, and treatment of potential precipitating conditions are performed concurrently rather than as isolated interventions **(Barski et al., 2023)**.

A recent case report of severe DKA with altered consciousness, acute kidney injury, hyperkalemia, and hemodynamic instability similarly described the combined use of fluid resuscitation, insulin, intravenous bicarbonate, and mechanical ventilation, emphasizing the need to individualize supportive therapy in patients with profound metabolic and respiratory compromise **(Vidanapathirana, 2024)**.

The evolution of the electrolyte abnormalities further illustrates the dynamic nature of DKA treatment. Despite an initial potassium concentration of 6 mmol/L, the patient subsequently developed recurrent hypokalemia, reaching 2.7 mmol/L and requiring repeated replacement. This apparent transition is consistent with the known depletion of total-body potassium caused by osmotic diuresis, followed by intracellular potassium redistribution after insulin administration and correction of acidosis. Hypophosphatemia also developed during recovery. These findings are consistent with contemporary DKA recommendations emphasizing frequent electrolyte monitoring throughout treatment rather than relying on admission values alone **(Vidanapathirana, 2024)**.

The patient developed recurrent severe hypoglycemia after DKA resolution, requiring dextrose therapy, insulin adjustment, and nutritional consultation. Consistent with this observation, **Omayer et al.** found no significant difference in hypoglycemia risk between bicarbonate-treated patients and controls **(Omayer et al., 2026)**.

The case demonstrates that profound DKA with coma, hemodynamic collapse, acute kidney injury, and respiratory failure can be reversible with comprehensive intensive care. The main clinical lesson is that patients with extreme acidemia require individualized management while insulin therapy, appropriate fluid resuscitation, electrolyte correction, hemodynamic support, and ventilatory management when indicated remain the foundation of treatment.

CONCLUSION:

Severe DKA with profound acidemia may cause shock, respiratory failure, acute kidney injury, and coma. In critically ill patients with pH <6.9 and shock, insulin therapy, appropriate fluid resuscitation, electrolyte correction, hemodynamic support, and ventilatory management when indicated remain the foundation of treatment.

DISCLOSURE

Conflicts of Interest: The authors declare that they have no actual or potential conflicts of interest, including financial, personal, or professional relationships, that could have influenced the work reported in this manuscript.

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Ethical Approval: Ethical approval was not required for this single case report according to the institutional policy of Al-Sobh Hospital, Jeddah, Saudi Arabia.

Patient Consent: Written informed consent for publication of this case report and the accompanying clinical information was obtained from the patient.

AUTHOR CONTRIBUTIONS:

Amr Abdelhamid Saber Karam conceived the case report, collected and interpreted the clinical data, reviewed the literature, and drafted and revised the manuscript. Asem Naji contributed to the clinical management of the patient, interpretation of the clinical data, and critical revision of the manuscript. Soliman Belal contributed to the critical care management of the patient, collection of clinical data, and critical revision of the manuscript. Ehab Salah Hegazy contributed to the interpretation of the clinical findings, literature review, and critical revision of the manuscript for important intellectual content. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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