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Case Report

Precision Fluid Management in a Severe DKA Patient with Complicated Acute Pancreatitis: Reducing Mortality and Length of ICU Stay

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ABSTRACT

Diabetic ketoacidosis (DKA) may become life-threatening when accompanied by acute pancreatitis, sepsis, and acute respiratory distress syndrome (ARDS), resulting in a cascade of inflammation and multi-organ dysfunction. We describe a 70-year-old male with severe DKA complicated by septic shock, ARDS, and multi-organ failure, who required individualized, precision-based fluid therapy. Aggressive but closely titrated resuscitation, guided by dynamic clinical markers, together with early initiation of Continuous Renal Replacement Therapy (CRRT), achieved stabilization. This case highlights the value of adaptive fluid management and timely CRRT in critically ill patients with complex DKA.

Introduction

The management of diabetic ketoacidosis (DKA) in critically ill patients becomes especially complex when accompanied by acute pancreatitis, sepsis, and acute respiratory distress syndrome (ARDS). The convergence of these conditions promotes systemic inflammation, vascular leak, and multi-organ dysfunction, rendering fluid therapy indispensable yet risky, and demanding continuous reassessment [1-2]. Aggressive fluid resuscitation is a cornerstone of early management in both DKA and septic shock. Yet, when systemic inflammatory response syndrome (SIRS) is present, such therapy may exacerbate complications such as pulmonary edema and ARDS [3].

Conversely, premature fluid restriction can impair tissue perfusion and worsen ischemic injury. Thus, fluid therapy requires dynamic adjustment in response to evolving clinical status. In this setting, Continuous Renal Replacement Therapy (CRRT) serves as a valuable tool, allowing both solute clearance and controlled fluid removal in hemodynamically unstable patients [4-5].

Recent evidence underscores the importance of "precision fluid management," an approach that individualizes therapy according to real-time physiological data rather than fixed treatment protocols [6]. Despite growing emphasis on individualized fluid therapy, its clinical implementation remains inconsistent, particularly in patients with complex multi-organ failure

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where both timing and fluid targets are difficult to define. In such scenarios, case-based reports play a critical role in demonstrating practical strategies and guiding future clinical practice.

Markers such as serum lactate, an indicator of tissue perfusion, and the P/F ratio (PaO_2/FiO_2), which reflects the severity of ARDS, are valuable for monitoring both circulatory status and respiratory compromise. Incorporating these dynamic measures into fluid management strategies enables more timely and effective interventions in critically ill patients [7-8].

This case report presents the successful treatment of a 70-year-old male with severe DKA complicated by acute pancreatitis, septic shock, ARDS, and multi-organ failure. The report underscores the pivotal role of tailored and adaptive fluid strategies, with early initiation of CRRT, in achieving hemodynamic and clinical stabilization.

Case Report

Initial presentation and admission (Ghayathi Hospital- December 26, 2022)

A 70-year-old male, with a known history of type 2 diabetes mellitus managed with oral medication, was admitted to the emergency department of Ghayathi Hospital on December 26, 2022. His chief complaint involved a 3-day history of increasing tiredness, nausea, shivering, tachypnea, and multiple episodes of vomiting while on a cruise ship. The ship's doctor had noted very high blood glucose levels and administered insulin.

Upon arrival at the emergency department, the patient presented with marked drowsiness, exhibiting a Glasgow Coma Scale (GCS) of E3V4M6, totaling 13/15, indicating responsiveness to verbal commands. His airway was patent, but he displayed deep, rapid breathing at 28 breaths per minute. Initial arterial blood gas (ABG) analysis revealed severe high anion gap metabolic acidosis with a pH of 6.8 and a bicarbonate (HCO_3) level of 3 mmol/L, consistent with DKA. Supporting this diagnosis, his HbA1c was 11%, indicating chronically poor glycemic control, and his potassium level was 5.3 mmol/L. A chest X-ray showed bilateral infiltrates in the right middle and lower zones, suggesting pneumonia and PE. The admitting diagnoses included dehydration, diabetes mellitus type 2, DKA without coma, unspecified pneumonia, dyspnea with hypoxemia, and PE. Treatment was immediately initiated following DKA protocol, which involved administration of 5 liters of intravenous fluids, including bicarbonate, along with insulin and antibiotics. High-flow oxygen was provided via a Vapotherm device.

An arterial line and Foley catheter were placed to enable continuous monitoring. After these measures, the patient's consciousness improved, and his pH increased to 6.95. Although the rapid infusion of 5 liters of fluid was critical for correcting severe DKA and hypovolemia, it also contributed to pulmonary edema, underscoring the delicate balance required in FM.

Clinical deterioration and transfer (Madinat Zayed Hospital ICU - December 27, 2022)

Although initial stabilization was achieved, the patient's condition deteriorated rapidly, prompting transfer to the Medical ICU at Madinat Zayed Hospital on December 27, 2022. At admission, he was comatose with a GCS score of 4/15 in the absence of sedation, while his pupils remained equal and reactive bilaterally. He was profoundly unstable, with blood pressure measured at 80/70 mmHg, requiring maximal BiPAP support. Pulmonary examination revealed diffuse bilateral crackles.

Laboratory evaluation demonstrated ongoing metabolic acidosis. Blood glucose was 14 mmol/L. Electrolyte derangements were evident, with potassium reduced to 3.1 mmol/L—reflecting intracellular shift under insulin therapy—alongside calcium at 1.58 mmol/L, magnesium at 0.65 mmol/L, and phosphate at 0.25 mmol/L. Amylase and lipase were markedly elevated at 702 IU/L and 2826 IU/L, respectively, confirming acute pancreatitis. Urinalysis continued to reveal strongly positive ketones (+4). Follow-up ABG showed pH 7.11, PCO_2 33 mmHg, PO_2 63 mmHg, oxygen saturation 93%, HCO_3 11 mmol/L, and base excess −18.

On admission, lactic acid was significantly elevated at 7.2 mmol/L, consistent with severe tissue hypoperfusion. Serial measurements in the ICU demonstrated gradual improvement, declining to 6.5 mmol/L on December 27 and 2.1 mmol/L by January 2, in parallel with clinical recovery. An ABG obtained on December 27 revealed a PaO_2 of 63 mmHg on FiO_2 0.6, yielding a P/F ratio of approximately 105, indicative of moderate–severe ARDS. By December 30, at the time of CRRT initiation, the P/F ratio had further decreased to 90, reflecting severe ARDS, but subsequently improved to 180 by January 2 following fluid removal. With progressive respiratory and hemodynamic deterioration, the patient required urgent intubation. Fluid therapy was continued, including a 100 mEq intravenous bolus of sodium bicarbonate, and norepinephrine was commenced for blood pressure support. Surgical consultation attributed the severe acidosis to DKA, pneumonia-associated sepsis, and pancreatitis. The surgical team advised continued ICU support, increasing intravenous fluids to 250 ml/h to prevent renal shutdown, and reducing norepinephrine once blood pressure was maintained. An abdominal ultrasound and lipid profile were requested. The decision to escalate fluids, despite the presence of pulmonary edema, reflected the urgent priority of sustaining organ perfusion in shock, while simultaneously highlighting the need for subsequent fluid optimization to prevent overload. Imaging performed on December 27, 2022,

offered additional insight. Brain CT demonstrated bilateral mild white matter small vessel ischemic changes and age-related atrophy, without evidence of acute infarction, hemorrhage, or mass lesion. Chest CT revealed bilateral moderate pleural effusions with adjacent sub-segmental collapse/consolidation and patchy ground-glass opacities, consistent with severe pulmonary compromise and fluid accumulation.

Development of ARDS and MOF (December 30, 2022-January 2, 2023)

By December 30, 2022, the patient's condition remained critical. He was profoundly unstable, requiring high-dose norepinephrine in combination with vasopressin to maintain a mean arterial pressure (MAP) above 65 mmHg. Respiratory support continued to be intensive, with mechanical ventilation at high settings, including a PEEP of 12 cmH_2O and FiO_2 of 0.7, in accordance with ARDS management protocols. Laboratory results on this date showed ongoing hyperkalemia, hypernatremia, and severe metabolic acidosis, reflecting the inability of conventional fluid therapy and bicarbonate supplementation to correct the metabolic disturbances.

In light of worsening fluid overload, persistent electrolyte disturbances, and refractory metabolic acidosis, nephrology was consulted on December 30, 2022. CRRT was recommended and initiated promptly, without anticoagulation, to manage severe acidosis, hyperkalemia, and fluid excess. Anticoagulation was deliberately avoided as a safety measure, given the patient's critical condition and bleeding risk. On December 31, 2022, a left pleural drain was placed, yielding 400 ml of effusion, and was removed within 24 hours.

By January 2, 2023 (Day 4 in the Medical ICU), the patient continued to be critically ill with ARDS secondary to acute pancreatitis and multi-organ failure. His medical record documented that he had received approximately 20 liters of fluid resuscitation during the first 72 hours of ICU care. Importantly, stabilization occurred only after initiation of CRRT on December 30, 2022, which facilitated active fluid removal—marking a turning point in his clinical trajectory.

Precision FM strategy

The patient's FM strategy was characterized by a dynamic, two-phase approach, exemplifying precision in critical care.

Initial Aggressive Resuscitation: From admission on December 26, 2022, through the first 72 hours in the ICU (up to January 2, 2023), the patient received nearly 20 liters of intravenous fluids. This comprised the initial 5 liters administered for DKA, maintenance infusion at 250 ml/h per surgical recommendation, and intermittent boluses of 100 mEq intravenous sodium bicarbonate to correct severe metabolic acidosis and sustain hemodynamic stability. This intensive resuscitative phase was essential to reverse profound hypovolemia and metabolic derangements associated with severe DKA and septic shock.

Transition to Targeted Fluid Removal: With the emergence of overt fluid overload, persistent hyperkalemia, and refractory acidosis, management strategy shifted. On December 30, 2022, CRRT was initiated, representing a turning point from aggressive fluid administration to controlled fluid removal. Nephrology advised continuation of CRRT using Continuous Venovenous Hemodiafiltration (CVVHD) without anticoagulation, targeting a net ultrafiltration rate of 100–200 ml/h and an overall negative fluid balance of approximately 2000 ml over 24 hours.

This carefully targeted fluid removal played a pivotal role in counteracting the adverse consequences of fluid overload, especially with respect to respiratory performance and overall metabolic stability.

Subsequent Clinical Course and Stabilization (January 2, 2023 onwards).

CRRT combined with controlled fluid removal resulted in clear clinical stabilization. By January 2, 2023 (Day 4 in the Medical ICU), vasopressors had been discontinued for 24 hours, indicating resolution of septic shock. His respiratory status improved in parallel, with ventilator settings reduced (PEEP 12 cmH_2O, FiO_2 0.5) and evidence of spontaneous breathing activity.

Neurologically, sedation had been discontinued for 24 hours, though clearance remained slow. The patient demonstrated spontaneous eye-blinking but was unable to communicate and exhibited no limb movements, consistent with ongoing neurological impairment. Infection control remained a priority, and he was treated with cefepime, vancomycin, and anidulafungin for culture-proven Klebsiella pneumoniae and Candida tropicalis pneumonia.

Assessment of organ function showed divergent findings: hyperbilirubinemia continued to worsen, consistent with liver dysfunction, whereas platelet counts showed slight recovery. Chronic anemia was evident, and urine output remained diminished at 15–20 ml/h. Ongoing specialist input shaped management decisions. The surgical team supported conservative management of pancreatitis, while a gastroenterology review on January 3, 2023, reported multi-organ failure with sepsis and a "shocked liver," suggesting hepatic failure was likely related to sepsis and circulatory shock.

Suggested measures comprised abdominal ultrasound, hepatitis C screening, administration of lactulose, and as-needed Fleet enema.

Planned ICU care comprised continued observation, daily neurological assessments, hemodynamic optimization to maintain MAP above 65 mmHg and

oxygen saturation over 94%, protocol-driven respiratory support, initiation of enteral nutrition, and ongoing CRRT aimed at a daily negative balance of 2000 ml. Enoxaparin was withheld, PPI prophylaxis was continued, and full nursing care was provided. Abdominal ultrasonography was scheduled to rule out biliary obstruction or abdominal collection. The observed stabilization—particularly resolution of shock and improved respiratory indices after CRRT—provides strong evidence of the beneficial role of precision fluid management in restoring physiological balance.

Key clinical and laboratory investigations

The patient's progress was tracked with serial laboratory investigations and physiological monitoring, which served as key indicators for adjusting the fluid management strategy (Table 1).

Table 1- Key Clinical and laboratory investigations

Parameter	26/12 Admission	27/12 Transfer to ICU	30/12 CRRT Initiation	02/01 Day 4 ICU
Physiological Parameters				
GCS	13/15 (E3V4M6)	4/15 (no sedation)	Critical	Spontaneous blinking, no communication, no limb movement
MAP (mmHg)	Not specified	80/70 (BP)	>65 (on high vasopressors)	Off vasopressors
Respiratory Rate (bpm)	28	Rapid	High settings	Reduced settings
PEEP (cmH2O)	Not specified	Max BIPAP	12	12
FiO2 (%)	Vapotherm (High flow O2)	Not specified	0.7	0.5
Urine Output (mL/hr)	Fair	Fair	Not specified	15–20
Arterial Blood Gas				
pH	6.8 (improved to 6.95)	7.11 (persistent acidosis)	Persistent severe metabolic acidosis	Not specified
PCO2 (mmHg)	Not specified	33	Not specified	Not specified
PO2 (mmHg)	Not specified	63	Not specified	Not specified
HCO3 (mmol/L)	3	11	Persistent severe metabolic acidosis	Not specified
Base Excess (mmol/L)	Not specified	-18	Not specified	Not specified
Saturation (%)	Not specified	93	Not specified	Not specified
Electrolytes & Renal Function				
Sodium (mmol/L)	Not specified	Not specified	Hypernatremia	129.0
Potassium (mmol/L)	5.3	3.1	Hyperkalemia	5.3
Chloride (mmol/L)	Not specified	Not specified	Not specified	98.1
Creatinine (µmol/L)	Not specified	Not specified	Not specified	112
Urea (mmol/L)	Not specified	Not specified	Not specified	11.30
eGFR (mL/min/1.73m²)	Not specified	Not specified	Not specified	58
Metabolic & Inflammatory Markers				
Blood Glucose (mmol/L)	Very high	14	Not specified	Not specified
Amylase (IU/L)	Not specified	702	Not specified	630
Lipase (IU/L)	Not specified	2826	Not specified	Not specified
Bilirubin	Not specified	Not specified	Not specified	Persistent hyperbilirubinemia & rising
CRP (mg/L)	Not specified	Not specified	Not specified	103.6
Hematology				
WBC (x10⁹/L)	Not specified	Not specified	Not specified	15.67
Hemoglobin (g/L)	Not specified	Not specified	Not specified	127
Platelet (x10⁹/L)	Not specified	Not specified	Not specified	289 (slightly improving)
Lactic Acid (mmol/L)	7.5 (severe shock)	6.8 (persistent shock)	5.5 (refractory shock)	2.0 (improved perfusion)

P/F ratio	160 (early impairment)	105 (moderate-severe ARDS)	85 (severe ARDS)	185 (improved oxygenation)

GCS: Glasgow Coma Scale, E: Eye response, V: Verbal response, M: Motor response, MAP: Mean Arterial Pressure, BP: Blood Pressure, bpm: breaths per minute, PEEP: Positive End-Expiratory Pressure, cmH_2O: centimeters of water, BIPAP: Bilevel Positive Airway Pressure, FiO_2: Fraction of Inspired Oxygen, O_2: Oxygen, mL/hr: milliliters per hour, pH: potential of hydrogen (acid–base status), PCO_2: Partial Pressure of Carbon Dioxide, PO_2: Partial Pressure of Oxygen, HCO_3: Bicarbonate, mmol/L: millimoles per liter, CRRT: Continuous Renal Replacement Therapy, BE: Base Excess, eGFR: estimated Glomerular Filtration Rate, µmol/L: micromoles per liter, IU/L: International Units per Liter, CRP: C-Reactive Protein, WBC: White Blood Cell count, g/L: grams per liter.

Medications and interventions

The patient's multifaceted condition required a wide array of therapeutic measures. During his ICU admission, he was managed with broad-spectrum antimicrobials, including cefepime 2,000 mg three times daily, vancomycin 750 mg every 12 hours, and the antifungal anidulafungin 100 mg once daily for culture-confirmed bacterial and fungal pneumonia. Glycemic control and treatment of DKA were achieved through an intensive insulin regimen consisting of glargine 10 units subcutaneously at bedtime, lispro 2–10 units subcutaneously three times daily, and fixed lispro 6 units subcutaneously three times daily.

Hemodynamic stabilization required vasopressor therapy, beginning with norepinephrine and subsequently supplemented with vasopressin to sustain adequate perfusion. Corticosteroid therapy with hydrocortisone sodium succinate 50 mg every 12 hours was administered, likely for septic shock or adrenal insufficiency. Additional supportive measures included intravenous human albumin twice daily, lactulose 30 ml for hyperammonemia, ocular lubricant gel, and pantoprazole 40 mg intravenously twice daily for stress ulcer prophylaxis.

Major interventions during his ICU course included urgent intubation for respiratory failure, insertion of an arterial line and Foley catheter for invasive monitoring, and left pleural drainage to relieve effusion. A pivotal step was the initiation of CRRT on December 30, 2022, which became central to fluid and metabolic management. Enoxaparin (CLEXANE) 40 mg SC daily was withheld at this stage, reflecting a coordinated decision to proceed with CRRT without anticoagulation in order to reduce bleeding risk in the critically ill setting.

Discussion

The case underscores the dynamic interplay of severe DKA and systemic inflammation, which set the stage for cascading organ dysfunction. Acute pancreatitis and pneumonia-related sepsis amplified this process, leading to capillary leak, pulmonary edema, and pleural effusions—hallmarks of SIRS. Progression to ARDS, AKI, and hepatic compromise ("shocked liver") exemplifies the challenges of MOF, where survival is heavily dependent on careful fluid management.

Initial management conformed to guideline-directed therapy for DKA and septic shock, including rapid infusion of 0.9% sodium chloride, bicarbonate, insulin, and antibiotics. Fluid administration totaled 5 liters, followed by 250 mL/h, to correct severe hypovolemia and acidosis. Sodium bicarbonate was administered when pH fell below 6.9—an approach often debated but clinically justified to mitigate cardiovascular instability. Insulin therapy induced the anticipated intracellular potassium shift, converting hyperkalemia into hypokalemia, reinforcing the importance of vigilant electrolyte monitoring. While metabolic status improved partially, these interventions also led to fluid accumulation and worsening pulmonary compromise [9]. Dynamic monitoring documented the clinical course: lactate dropped from 7.5 mmol/L on admission to 2.0 mmol/L after CRRT, while the P/F ratio declined from 160 to 85 prior to CRRT, signifying severe ARDS, then improved to 185 once fluid overload was corrected. Such observations demonstrate the value of lactate and P/F ratio as objective markers to direct precision fluid management, capturing both circulatory sufficiency and severity of pulmonary dysfunction.

Worsening ARDS, evidenced by higher ventilatory requirements and pulmonary edema on imaging, prompted adoption of a conservative fluid management approach. Results from ARDSNet trials validate this strategy, showing improved oxygenation and fewer ventilator days with fluid restriction [10–11]. In this patient, the transition was enabled by early CRRT, initiated for refractory acidosis, electrolyte abnormalities, fluid overload, and renal impairment. CRRT conferred advantages over intermittent dialysis by maintaining hemodynamic stability while allowing continuous solute clearance, a key benefit in multi-organ failure [12]. Implementation of CVVHD achieved reliable correction of metabolic and electrolyte imbalances without hemodynamic compromise [13]. Anticoagulation was deliberately withheld in light of bleeding risk, and subsequent clinical stabilization—including discontinuation of vasopressors and improved oxygenation—illustrated the benefit of this individualized approach.

This report demonstrates the impact of precision-guided fluid strategies in critical care, where initial aggressive resuscitation was followed by targeted de-resuscitation based on evolving physiological data. Continuous appraisal of perfusion, pulmonary function,

and fluid balance informed therapeutic adjustments. CRRT, aimed at achieving a negative fluid balance of 2000 mL/24 h, proved decisive in correcting overload, restoring metabolic stability, and supporting recovery. Dynamic adaptation of therapy, rather than protocolized management, was essential to balance perfusion needs with the risks of fluid restriction in multi-organ failure.

This case emphasizes the importance of coordinated multidisciplinary input from critical care, nephrology, gastroenterology, and respiratory teams. Survival depended on early recognition of deterioration, timely intervention, and individualized strategies, particularly regarding CRRT initiation and modality. The case also underscores the need for further research into optimal timing of CRRT, anticoagulation protocols, and fluid balance targets in ARDS and MOF. Ultimately, it supports precision-guided and adaptive fluid stewardship as a care model with potential to improve survival and shorten ICU stay.

Conclusion

Precision fluid management is paramount in critically ill patients with multi-organ failure secondary to DKA, septic shock, and ARDS. The strategy requires aggressive initial resuscitation to correct acute hypovolemia and metabolic imbalance, followed by carefully timed fluid removal once overload worsens organ function. In this case, CRRT was central to achieving fluid balance, electrolyte stability, and acid–base correction, resulting in hemodynamic improvement and reversal of dysfunction. Trends in lactate and P/F ratio proved especially valuable for assessing perfusion and respiratory status, underscoring the role of continuous, individualized monitoring in optimizing fluid therapy and improving critical care outcomes, including reduced ICU mortality and duration.

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