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Albumin–Crystalloid Synergy for Fluid-Restricted Case of Diabetic Ketoacidosis: A Volume-Sparing Strategy in a Critically Ill Patient

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Abstract
Aggressive fluid resuscitation remains a cornerstone in the management of diabetic ketoacidosis (DKA) besides insulin therapy and electrolyte correction. However, patients with fluid-sensitive comorbidities like heart failure and chronic kidney disease, where the boundary between benefit and harm is dangerously thin, may find this approach hazardous. We present a critically ill 62-year-old man with poorly controlled type 2 diabetes mellitus, heart failure with reduced ejection fraction (HFrEF), and stage V chronic kidney disease (CKD) who developed DKA with urosepsis. Given his fluid restriction, conventional therapy risked catastrophic fluid overload. A volume-sparing approach was employed, combining 5% human albumin, followed by a carefully titrated balanced crystalloid. This strategy achieved rapid metabolic correction without precipitating pulmonary or cardiac decompensation. This case presents a volume-sparing strategy: the synergistic administration of albumin and crystalloids. This tailored approach offers a safe, effective, and physiologically sound alternative to conventional high-volume protocols, addressing a critical therapeutic gap in fluid-restricted DKA patients.

Keywords
Diabetic Ketoacidosis; Albumin; Fluid; Heart Failure; Case Report

Introduction

Diabetic ketoacidosis (DKA) remains one of the most dramatic, acute, life-threatening metabolic complications of diabetes mellitus besides hyperosmolar hyperglycaemic state (HHS). It is characterised by hyperglycaemia, ketosis, and metabolic acidosis.[1, 2] It is typically seen in patients with type-1 diabetes mellitus, though it may also occur in patients with type-2 diabetes mellitus. The examples of causes are infection including fungal, stress, certain drugs and non-adherence to anti-hyperglycaemic agents.[2] Management is typically formulaic: restore volume, reverse acidosis, and replace electrolytes.[2, 3] Yet, in

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patients whose hearts or kidneys can no longer tolerate standard fluid volumes, the age-old question resurfaces: *how do we rehydrate without drowning the patient?*

Traditional guidelines advocate liberal fluid replacement to counteract osmotic diuresis. However, for patients with heart failure and advanced CKD, this approach risks precipitating pulmonary congestion, worsening renal perfusion, and multi-organ compromise.[3, 4] In such cases, fluid administration becomes both a cure and a curse. In this report, we aim to share a case of a patient with fluid restriction (heart failure and CKD) who developed DKA in the background of an active infection (community-acquired pneumonia and urosepsis) and how the patient responded to the treatment given, which contributed to the resolution of DKA. The patient was successfully managed through a low-volume albumin–crystalloid synergy strategy.

This approach marries the intravascular potency of colloids with the biochemical gentleness of balanced crystalloids. Beyond its immediate clinical success, this case invites a paradigm shift: that *resuscitation should be guided not by fixed volumes but by physiologic needs.*

Case Summary

We present a clinically challenging case of DKA in a 62-year-old male with type 2 diabetes mellitus, complicated by heart failure with reduced ejection fraction (HFrEF) and stage V CKD, necessitating strict fluid restriction to 1 L/day. His latest formal echo showed ejection fraction (EF) of 15-20%, poor left ventricular (LV) function, global hypokinetic with regional wall motion abnormalities (RWMA) seen and trivial mitral regurgitation (MR). His baseline HbA1c is 11.8% with oral hypoglycemic agents and insulin. He presented to the emergency department with lethargy, vomiting, and poor oral intake following a recent discharge for urosepsis. Previously, the patient was treated for septic shock secondary to community-acquired pneumonia (CAP) and urosepsis with intravenous (IV) antibiotics (tazobactam/piperacillin) and discharged with oral antibiotics (ampicillin/sulbactam) to complete a total course of two weeks. Despite antibiotic therapy, his infection has recurred, leading to progressive dehydration, hypotension, and metabolic acidosis.

On arrival, he appeared frail but alert, with clinical signs of dehydration. He was afebrile with a temperature of 36.3. Cardiac monitoring revealed the patient was having rate-controlled atrial fibrillation (AF) but was otherwise haemodynamically stable, not requiring inotropes or vasopressors. Oxygenation status was unremarkable. The abdomen was soft and non-tender on palpation, and the renal punch test was negative. Laboratory results revealed leukocytosis, elevated inflammatory markers (C-reactive protein), and worsening renal function. His renal profile depicted derangement with raised urea and creatinine levels and hypokalaemia. The liver profile was fairly normal except for hypoalbuminemia. Urine analysis showed signs of bacteriuria. The patient was admitted to the ward with a diagnosis of partially treated urosepsis, and the antibiotic was escalated from oral ampicillin/sulbactam to IV meropenem.

Since admission he still has vomiting episodes in the ward until day two of admission; oral intake improved but is still minimal and maintained with 500 ml of IV fluid over 24 hours. On day three of admission, the patient underwent a left nephrostomy and intraoperatively revealed turbid urine. The subsequent workup confirmed an infected, poorly functioning left kidney. Microbiological results supported the presence of a resistant gram-negative organism *Pseudomonas Aeruginosa*, guiding targeted therapy while the primary focus remained on haemodynamic stabilisation and fluid optimisation

On day four of admission, the patient had a hypotensive episode and was resuscitated with 500 ml of fluid and maintained with 1 L of drips over 24 hours. A day after that, the patient desaturated under room air,

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and oxygenation was escalated to a high-flow mask. Clinically, the patient was in fluid overload with evidence on chest X-ray (**figure 1**) and haemodynamically required inotropes.

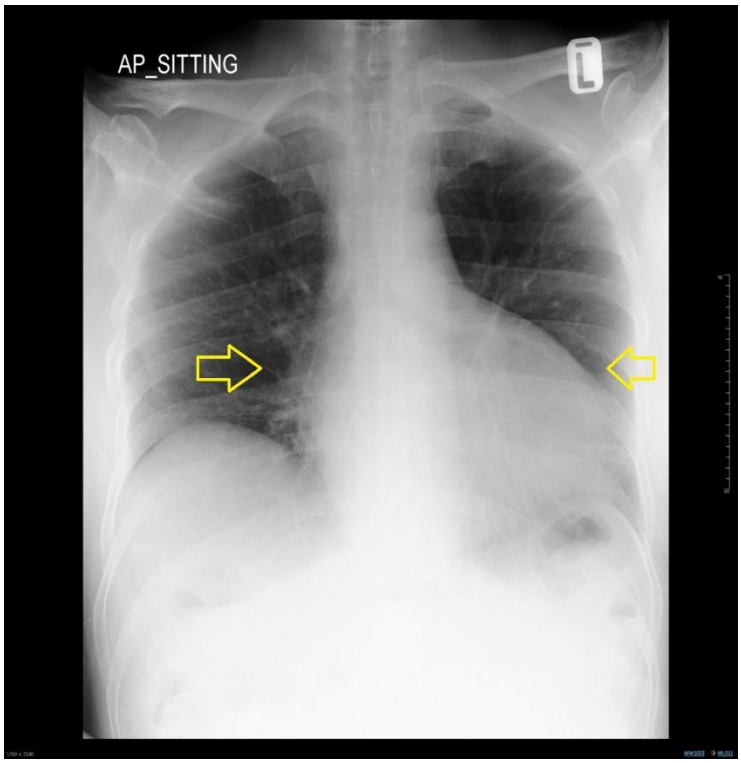


Figure 1: Chest X-ray with evidence of pulmonary congestion (right arrow) and cardiomegaly (left arrow).

The patient was admitted to the intensive care unit (ICU) for close monitoring. Upon arrival to the ICU, he required escalating inotropic support with a noradrenaline IV infusion (IVI) at 0.5 µg/kg/min and started IVI dobutamine up to 5 µg/kg/min. Non-invasive ventilation (NIV) was initiated due to worsening respiratory distress. Bedside lung ultrasound demonstrated bilateral B-lines suggestive of pulmonary congestion, and echocardiography revealed globally reduced cardiac contractility. The first blood gas in ICU revealed pH 7.20, bicarbonate 16.6 mmol/L, glucose 18.6 mmol/L, and ketone 3.1 mmol/L, confirming acute DKA. In light of his severe volume restriction and cardiac vulnerability, a volume-sparing strategy was adopted using albumin–crystalloid synergy.

He received an initial IV fluid of 5% human albumin (250 mL over two hours) and a fixed-rate insulin infusion (6 U/h) equal to 0.1 U/kg/h. Re-evaluation two hours later revealed persistent acidosis (pH 7.26) but sustained urine output, prompting cautious administration of 500 mL Sterofundin over the next two hours. The assessment is continuous with inferior vena cava (IVC) diameter and collapsibility index. Enteral feeding was started, maintaining the glucose level ranging from 6 to 12 mmol/l with frequent monitoring hourly. Follow-up blood gas showed marked metabolic improvement; pH normalised to 7.36, and serum ketones declined to 0.4 mmol/L, signifying resolution of DKA within four hours of guided resuscitation. Fluid support was subsequently maintained with 500 mL Sterofundin over 24 hours alongside continued enteral nutrition and insulin infusion.

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Electrolytes were maintained within normal range and potassium management followed DKA protocol adaptations with target potassium 4.0–5.0 mmol/L. Serial blood gases over the day demonstrated sustained acid-base stability (**table 1**). The patient was successfully weaned off NIV and vasopressor support without requiring intubation and was discharged from ICU care after a three-day admission.

Table 1: serial ABG taken during event

Events/Timeline	Initial Event	2-hours post albumin 5%	2-hours post 500 mls sterofundin over 2 hours	8 hours during IV sterofundin maintenance over 24 hour	Normal Range
pH	7.20	7.26	7.36	7.42	7.35 – 7.45
pCO2 (mmHg)	30.5	38.5	37.2	37.2	35 – 45
pO2 (mmHg)	191.6	269.1	145.4	181.8	75 – 100
Lactate (mmol/L)	3.2	3.52	3.43	2.05	0.5 – 2.0
Base excess (mmol/L)	-11.7	-10.1	-4.9	-0.9	–2 to +2
HCO3 (mmol/L)	16.6	17.1	21	24.1	22 – 26
Serum glucose (mmol/L)	15.6	16.2	14.9	15.1	6 – 10
Serum ketone (mmol/L)	3.1	3.1	0.4	Low	<0.6
Serum Potassium (mmol/L)	4.08	3.35	3.71	3.26	3.5-4.5

Discussion

DKA remains a significant clinical concern, contributing substantially to hospitalisations, recurrence, and mortality. Globally, the mean prevalence of DKA is approximately 30% across Europe, Australia, New Zealand, and the United States, with notable regional variation ranging from 19.5% in Sweden to 43.8% in Luxembourg.[5] Managing DKA in patients with fluid-sensitive comorbidities represents a true therapeutic paradox. Fluid resuscitation is the cornerstone of DKA management, yet for fluid-sensitive patients, it is a double-edged sword, essential for reversing hypoperfusion but potentially fatal if it precipitates pulmonary congestion or cardiac decompensation.[3, 4] This case illustrates that metabolic rescue in DKA is still achievable under strict volume restriction, provided the resuscitation strategy is guided by physiology rather than rigid protocol.

The imperative for rapid intravascular repletion must be weighed carefully against the ever-present danger of precipitating pulmonary oedema particularly in individuals with HFrEF and advanced CKD. Human albumin, a naturally occurring colloid, offers unique pharmacokinetic and haemodynamic properties that make it particularly suitable in such cases. With approximately 40–45% of its pool residing intravascularly and a large molecular size limiting transcapillary escape, albumin provides sustained plasma expansion with minimal infused volume.[6, 7] In this case, 5% albumin acted not merely as a resuscitation fluid but as a strategic bridge stabilising circulation and maintaining preload while minimising the risk of pulmonary oedema. Beyond its oncotic function, albumin possesses important biological effects, including antioxidant activity, free radical scavenging, and endothelial protection.[6, 7] These properties are particularly valuable

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in septic and inflammatory states, where capillary leak and oxidative stress exacerbate haemodynamic instability.

Nevertheless, its use in HFrEF and CKD is not without hazard.[6, 7] The inherent sodium load content with colloid solution may exacerbate congestion, its cost remains substantial, and excessive or rapid administration risks overshooting intravascular volume in patients with limited cardiac reserve. Impaired renal clearance further amplifies these concerns, underscoring the necessity of slow infusion, vigilant haemodynamic monitoring, and dynamic tools such as IVC ultrasound assessment to guide responsiveness. Hence, in a patient with both urosepsis and cardiac dysfunction, albumin infusion offered a physiologically sound, volume-efficient first step in resuscitation.

Following initial albumin resuscitation, the subsequent administration of a balanced crystalloid (Sterofundin) allowed fine-tuning of acid–base equilibrium. Crystalloids are typically the mainstay of DKA management.[3, 4] However, their extracellular distribution results in only one-third remaining intravascularly, demanding large infusion volumes to restore perfusion. Unlike isotonic saline, which carries a high chloride content and predisposes to hyperchloremic acidosis, Sterofundin closely mimics plasma electrolyte composition with an appropriate strong ion difference (SID).[8] Its inclusion of acetate and malate as metabolisable anions supports endogenous bicarbonate generation, contributing to the correction of acidosis. Furthermore, Sterofundin's modest potassium content counterbalances the intracellular potassium shifts induced by insulin therapy, mitigating hypokalaemia, a subtle yet clinically important advantage in the management of DKA. The combined use of colloid followed by balanced crystalloid thus represents a sequential, physiology-aligned fluid strategy: albumin restores haemodynamic stability, and crystalloid sustains biochemical recovery.

Large-scale trials such as the SAFE Study (2004) and ALBIOS Study (2014) provide reassuring safety data on albumin administration in critically ill patients, including those with sepsis.[9, 10] Although DKA-specific data remain limited, the underlying pathophysiological rationale aligns with these findings, particularly in patients where fluid overload risk outweighs the benefits of liberal crystalloid infusion. Recent literature in critical care also advocates for individualised, dynamic fluid therapy over algorithmic approaches.[11] The present case supports this shift, demonstrating that small-volume, targeted interventions can yield rapid metabolic correction without jeopardising cardiopulmonary stability. This aligns with the growing concept of "precision fluid therapy", where the type, timing, and titration of fluid are as crucial as the total volume administered.

Conclusion

The successful management of DKA in this fluid-restricted patient underscores the importance of personalised fluid therapy. This case demonstrates that DKA in fluid-restricted patients can be safely corrected through a physiology-guided, volume-sparing strategy. A hybrid strategy integrating 5% albumin and balanced crystalloid achieved metabolic recovery and haemodynamic stability without fluid overload. This approach challenges conventional high-volume protocols, emphasising that fluid therapy should be individualised and precision-driven, particularly in cardiorenal-compromised patients.

Declaration of Competing Interests

There is no actual or potential conflict of interest in relation to this article.

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