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Septic Shock Management Using Continuous Renal Replacement Therapy in a Postpartum Patient with Diabetic Ketoacidosis, Acute Kidney Injury, and Ventilator-Associated Pneumonia

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ABSTRACT

Introduction: Postpartum sepsis in patients with pregestational diabetes mellitus is a life-threatening condition that may precipitate acute kidney injury (AKI) and diabetic ketoacidosis (DKA). Sepsis frequently leads to multiorgan dysfunction, with the kidneys being particularly vulnerable. Severe AKI in septic shock often requires renal replacement therapy. Continuous Renal Replacement Therapy (CRRT), particularly in hemodynamically unstable patients, is the preferred modality due to its gradual solute and fluid removal, cytokine modulation, and ability to manage complex acid-base disturbances. In this setting, secondary ventilator-associated pneumonia (VAP) further complicates management.

Case Description: A 35-year-old woman with pregestational type 2 diabetes mellitus developed septic shock with DKA following spontaneous vaginal delivery. She presented with refractory hypotension, severe metabolic acidosis (pH 6.98), hyperglycemia (301 mg/dL), ketonuria (2+), and oliguria (0.3 mL/kg/h). Serum creatinine rose from 0.85 to 3.61 mg/dL, fulfilling KDIGO stage 3 AKI criteria. During ICU stay, the patient developed VAP, necessitating prolonged mechanical ventilation and targeted antimicrobial therapy.

Conclusion: Early initiation of Continuous Veno-Venous Hemodiafiltration (CVVHDF) using bicarbonate-buffered replacement fluid and an oXiris filter effectively corrected severe acidosis (pH 7.255 → 7.359 within 76 hours), removed ketones and inflammatory mediators, stabilized hemodynamics, and facilitated renal recovery while reducing vasopressor requirements. Multidisciplinary management, including strict VAP prevention bundle and culture-directed antibiotics, enabled successful extubation. Timely high-volume CRRT combined with comprehensive critical care is crucial in managing complex postpartum septic shock with DKA and AKI.

Keywords

Acute Kidney Injury, Continuous Renal Replacement Therapy, Septic Shock, Diabetic Ketoacidosis, Pregestational Diabetes, Postpartum, Ventilator-Associated Pneumonia

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INTRODUCTION

Postpartum sepsis remains a leading cause of maternal mortality worldwide, with an incidence ranging from 0.5% to 3% in pregnant women [1]. Despite its relatively low prevalence, it accounts for significant maternal morbidity, mortality, and long-term sequelae [2]. In low-resource settings, reported rates are markedly higher, reaching 12.2% in Kenya and 37.5% in Uganda within two weeks of postpartum [3,4]. Postpartum sepsis ranks as the third or fourth most common cause of maternal death in both developing and developed countries [2]. Among its complications, acute kidney injury (AKI) is common, with sepsis accounting for 70.3% of postpartum AKI.

The pathophysiology of AKI in septic shock is complex and multifactorial. Endothelial dysfunction, renal microcirculatory impairment, and excessive release of inflammatory mediators contribute to kidney injuries. Intriguingly, cortical hypoperfusion may occur despite the preservation of macrovascular renal blood flow [5]. Pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs) trigger an overwhelming inflammatory cascade that exacerbates renal injury [6]. In patients with pregestational diabetes mellitus, the risk of diabetic ketoacidosis (DKA) is heightened during pregnancy and the puerperium, further aggravating AKI in the setting of septic shock [7]. The synergistic effect of septic shock and DKA in postpartum women with pregestational diabetes markedly increases the complexity of management and worsens the prognosis of the condition.

Continuous renal replacement therapy (CRRT) has emerged as the preferred renal replacement modality for AKI in septic shock [8]. Compared with intermittent hemodialysis, CRRT offers superior hemodynamic stability, precise fluid management, sustained azotemia control, and beneficial immunomodulation through the enhanced clearance of inflammatory mediators [9]. Retrospective data have demonstrated that early CRRT initiation in patients with septic shock and AKI is associated with significantly reduced ICU mortality (27.7% vs. 63.5%; $p<0.001$) [10]. A cutoff analysis identified 16.5 h from AKI onset to CRRT initiation as a critical threshold, beyond which cumulative ICU mortality rises sharply [11]. The use of adsorbent membranes, such as oXiris, further enhances endotoxin and cytokine removal, accelerates lactate clearance, and reduces vasopressor requirements [9].

Diabetic ketoacidosis during pregnancy and the postpartum period carries a high risk of perinatal mortality, although the risk of maternal mortality remains poorly defined. Most pregnant women who developed DKA had pre-existing type 1 diabetes (45.9%), followed by gestational diabetes (40.5%). Infection is the most common precipitant (28%), followed by poor treatment adherence (13.5%) [1]. Euglycemic diabetic ketoacidosis (DKA), characterized by normoglycemia with ketonemia and metabolic acidosis, poses a particular diagnostic challenge during pregnancy. When DKA coincides with postpartum septic shock, comprehensive therapeutic strategies, including CRRT, are essential to improve outcomes [12]. The management of concomitant postpartum septic shock, AKI, and DKA in pregestational diabetes requires a multidisciplinary approach encompassing aggressive fluid resuscitation, early targeted antibiotics, tight glycemic control, and timely CRRT initiation. Continuous veno-venous hemodiafiltration (CVVHDF) has shown promise in hemodynamically unstable patients. In addition to renal support, CRRT contributes to hemodynamic stabilization, cytokine modulation, and overall clinical improvement [12]. Specialized adsorbent filters (e.g., oXiris) augment the removal of endotoxins and proinflammatory mediators, further optimizing outcomes in patients with septic shock. Timely, high-volume CRRT integrated within a comprehensive critical care bundle can substantially improve the prognosis of this highly complex patient population [11].

CASE DESCRIPTION

A 35-year-old woman (G3P2A0) with poorly controlled pregestational type 2 diabetes mellitus was referred by a midwife in West Bandung following irregular antenatal care visits. She presented on May 8, 2025, at 32–33 weeks gestation with premature contractions and a random blood glucose level of 281 mg/dL. Spontaneous preterm vaginal delivery occurred shortly after arrival. Immediately postpartum, she developed severe dyspnea and altered consciousness, necessitating urgent transfer to the medical adult intensive care unit (MAICU). There was no history of cough, fever, or upper respiratory symptoms in the patient. Initial urinalysis revealed significant bacteriuria (746.2/μL), erythrocyturia (990+/μL), ketonuria (2+), and glucosuria (2+), strongly suggesting a urinary tract infection as the source of sepsis and concurrent diabetic ketoacidosis (DKA).

On admission, the patient was intubated for airway protection and placed on mechanical ventilation (PSIMV mode: PC 14 cmH_2O, RR 14, PEEP 5 cmH_2O, FiO_2 100%, and PS 5 cmH_2O). She was hemodynamically unstable, requiring norepinephrine infusion (initially 0.2 mcg/kg/min) with blood pressure 107/82 mmHg, heart rate 158 bpm, and respiratory rate 28/min on the ventilator. Severe metabolic acidosis was confirmed by arterial blood gas analysis (pH 7.051, pCO_2 10.5 mmHg, HCO_3^- 2.9 mEq/L, base excess -

24, lactate was not initially measured). Laboratory findings included leukocytosis (37,000/μL), hyperglycemia (276–400 mg/dL), hyperkalemia (5.6–5.9 mEq/L), and oliguria (0.63 mL/kg/h). An initial chest X-ray (May 9) showed suspected bilateral perihilar lymphadenopathy without consolidation or cardiomegaly (CTR, 49.8%) (Figure 1).

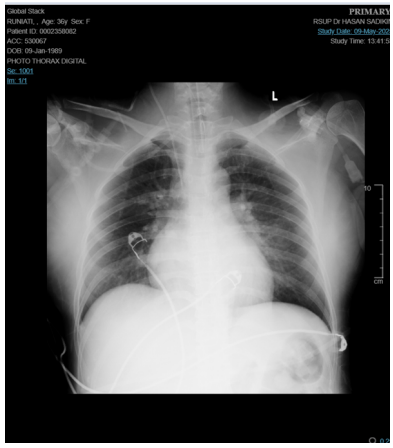

Figure 1. Chest X-ray on hospital day 1

During the first 72 h (H0–H3), refractory hypotension prompted vasopressin addition (0.04 units/h), and broad-spectrum antibiotics were escalated from ceftriaxone to meropenem on day 3 after procalcitonin peaked at 253.94 ng/mL. Aggressive fluid resuscitation, bicarbonate infusion, and sliding-scale insulin were administered according to the DKA protocol; however, acidosis persisted (pH, 6.98–7.10). Progressive abdominal distension with weakened bowel sounds raised concerns of paralytic ileus secondary to septic shock. From hospital days 4–5 (H4–H5), clinical deterioration ensued with refractory hypotension (80/53 mmHg despite norepinephrine 0.5 mcg/kg/min), deepening coma (GCS E1M1V_{ett}), and acute kidney injury progression. Serum creatinine increased from a baseline of 0.85 mg/dL to 3.61 mg/dL with persistent oliguria (<0.3 mL/kg/h), fulfilling the KDIGO stage 3 AKI criteria. Serum lactate levels reached 4.0 mmol/L, and the severe acidosis worsened (pH 6.98). Sedation was switched from midazolam to dexmedetomidine.

Table 1. Key Laboratory and Acid-Base Trends Before and During Continuous Renal Replacement Therapy (CVVHDF)

Parameter	Pre-CRRT Day 5	CRRT Day 1	CRRT Day 2	CRRT Day 3	CRRT Day 5	Post-CRRT (Day 11)
Urea (mg/dL)	67.1	79.8	56.3	55.1	48.4	42.1
Creatinine (mg/dL)	3.61	3.14	1.04	1.22	0.90	0.78
pH	6.98	7.25	7.35	7.42	7.448	7.41
HCO_3^- (mEq/L)	10.1	11.6	15.8	14.7	19.6	24.2
Base Excess	–20	–13	–8	–7.5	–2.8	+1.2
Lactate (mmol/L)	4.0	—	—	2.7	—	1.1
Urine Output (mL/kg/h)	<0.3	0.3	0.6	0.97	1.35	2.79

Noted: CRRT initiated on hospital day 6 using CVVHDF mode (oXiris filter, bicarbonate-buffered fluids, effluent dose ~40 mL/kg/h). Rapid correction of severe mixed acidosis and stage 3 AKI was observed within 76 h.

Continuous Renal Replacement Therapy (CRRT) was initiated on hospital day 6 (May 14) using the continuous veno-venous hemodiafiltration (CVVHDF) mode with bicarbonate-buffered solutions, post-dilution replacement (600 mL/h), dialysate flow 600 mL/h, blood flow 120–150 mL/min, net ultrafiltration 50 mL/h, and systemic heparin anticoagulation (Table 1). An oXiris filter was used to enhance the adsorption of endotoxins and cytokines. Within 76 hours of CRRT, acid–base status markedly improved (pH 7.255 → 7.359, HCO_3^- 11.6 → 15.8 mEq/L), renal parameters normalized (creatinine 3.61 → 0.9 mg/dL, urea 79.8 → 48.4 mg/dL), and urine output progressively increased to 2.79 mL/kg/h by day 11 (Table 2). The vasopressor requirements steadily declined, and norepinephrine was discontinued on day 11.

Table 2. Continuous Renal Replacement Therapy (CRRT) Prescription and Settings

Parameter	Setting
Modality	Continuous Veno-Venous Hemodiafiltration (CVVHDF)
Access	Primed double-lumen catheter
Filter	oXiris (adsorptive polymethylmethacrylate membrane)
Blood flow rate	120–150 mL/min
Dialysate flow rate	600 mL/h
Replacement fluid flow	600 mL/h (post-dilution)
Total effluent dose	~40 mL/kg/h
Net ultrafiltration	50 mL/h
Replacement/dialysate fluid	Bicarbonate-buffered solution (Prismasol/Biphozyl)
Anticoagulation	Unfractionated heparin 5–10 IU/kg/h (target aPTT 45–80 s)
Duration	5 days (total running time ≈120 hours)

Note: High-volume CVVHDF with the oXiris filter was chosen for combined convective-diffusive clearance, enhanced endotoxin/cytokine adsorption, precise fluid management, and hemodynamic tolerability in refractory septic shock with stage 3 AKI and severe metabolic acidosis. No circuit clotting or significant bleeding was noted.

Serial chest radiographs obtained on day 10 (May 18) revealed new bilateral consolidations consistent with ventilator-associated pneumonia (VAP) (Figure 2).

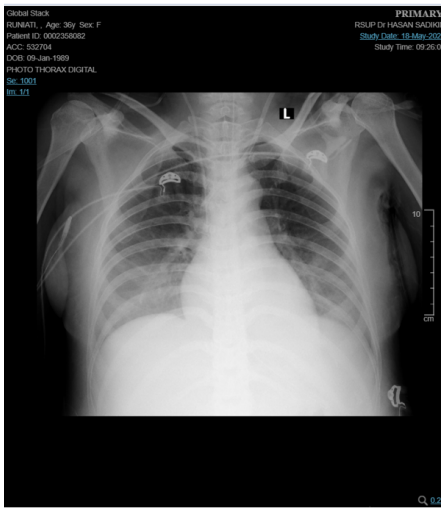

Figure 2. Chest X-ray on hospital day 10

Strict VAP prevention bundle measures were intensified (head-of-bed elevation 30°–45°, oral care, thromboprophylaxis, proton-pump inhibition, and daily interruption of sedation). Antibiotic therapy was continued with meropenem, guided by subsequent culture results. The patient was successfully extubated on day 12 (May 20) with full consciousness (GCS 15), stable hemodynamics without vasopressors, and adequate oxygenation on a simple mask. She was transferred to the regular ward on day 13 with oxygen supplied via a nasal cannula and subsequently discharged in a stable condition.

DISCUSSION

Pregestational type 2 diabetes mellitus confers persistent β-cell dysfunction and insulin resistance that extend into the postpartum period, rendering patients highly susceptible to metabolic decompensation [13]. Although placental diabetogenic hormones (human placental lactogen and cortisol) rapidly decline after delivery, the underlying pancreatic insufficiency and peripheral insulin resistance remain, necessitating vigilant glycemic control [14]. In this patient, poor adherence to antenatal care and insulin therapy precipitated severe hyperglycemia (up to 301 mg/dL), ketonuria, and profound metabolic acidosis (pH 6.98, HCO_3^- 2.9 mEq/L), fulfilling the diagnostic criteria for diabetic ketoacidosis (DKA) [15]. Relative insulin deficiency, coupled with elevated counter-regulatory hormones (glucagon, catecholamines, cortisol, and growth hormone), drives

gluconeogenesis, lipolysis, and ketogenesis in the liver. The resultant osmotic diuresis and volume contraction exacerbated hypoperfusion, whereas β-hydroxybutyrate accumulation directly worsened acidosis and endothelial dysfunction [16]. Unlike gestational diabetes, which typically resolves postpartum, pregestational disease carries irreversible β-cell damage, explaining the extreme glycemic volatility observed despite the use of sliding-scale insulin and dextrose-containing fluids [14].

Postpartum sepsis in patients with diabetes most commonly originates from urinary tract infection, facilitated by physiological hydronephrosis, glucosuria serving as a bacterial growth substrate, and hyperglycemia-induced neutrophil dysfunction [17-21]. Significant bacteriuria on admission confirmed UTI as the primary septic focus, rapidly evolving into urosepsis and distributive shock. Chronic hyperglycemia impairs phagocytosis and promotes oxidative stress, amplifying endothelial permeability and systemic inflammatory responses [18]. Cytokine storm (TNF-α, IL-6) induces refractory vasodilatation and capillary leakage, necessitating escalating vasopressor support [22-24]. Paralytic ileus, manifesting as progressive abdominal distension, reflects sepsis-mediated microvascular gut dysfunction and bacterial translocation, further aggravating the inflammatory cascade [25]. Although genital tract sources were considered, microbiological evaluation supported UTI as the dominant etiology.

The patient's stage 3 AKI exemplified synergistic injury due to septic shock and DKA [26,27]. Sepsis triggers microcirculatory failure, endothelial damage, and tubular apoptosis via pathogen- and damage-associated molecular patterns, reactive oxygen species, and proinflammatory cytokines, often independent of macroscopic hypoperfusion [5,6]. Concurrent DKA contributes to prerenal azotemia through osmotic diuresis and effective volume depletion, whereas severe acidosis impairs renal autoregulation and mitochondrial function [28]. Advanced glycation end-products from chronic hyperglycemia activate NF-κB pathways, promoting intrarenal inflammation and tubular cell death [29]. This dual pathology rapidly escalated creatinine levels from 0.85 to 3.61 mg/dL with oliguria, underscoring why combined metabolic and septic insults portend a dismal renal prognosis without prompt renal replacement therapy.

Early continuous veno-venous hemodiafiltration (CVVHDF) using an oXiris adsorptive membrane has proven lifesaving [8,9]. Combined diffusive-convective clearance efficiently removed β-hydroxybutyrate (104 Da) and middle-molecular-weight cytokines, while bicarbonate-buffered replacement corrected acidosis within 76 h (pH 7.255 → 7.359; HCO_3^- 11.6 → 15.8 mEq/L) [30-32]. High-volume effluent (40 mL/kg/h) and endotoxin/cytokine adsorption reduced vasopressor requirements and restored hemodynamic stability, consistent with evidence that early high-dose CRRT significantly improves survival in patients with septic AKI [33]. Urine output recovery to 2.79 mL/kg/h by day 11 confirmed renal recovery, which was facilitated by precise fluid management and immunomodulation therapy. Ventilator-associated pneumonia emerged as a late complication, as evidenced by new bilateral consolidations on chest imaging performed on day 10. Strict adherence to the VAP prevention bundle—head-of-bed elevation 30°–45°, daily sedation interruption, oral chlorhexidine, and peptic ulcer/deep vein thrombosis prophylaxis— minimized disease progression and enabled successful extubation on day 12 despite prolonged mechanical ventilation.

This case demonstrates that multidisciplinary management integrating source control, targeted antimicrobials, tight glycemic control, vasopressor titration (norepinephrine plus vasopressin), and timely high-volume CVVHDF with oXiris can achieve full recovery in the highly lethal triad of postpartum septic shock, DKA, and stage 3 AKI complicating pregestational diabetes [34-38]. Early CRRT not only provides metabolic correction and organ support but also exerts beneficial extracorporeal cytokine removal, highlighting its pivotal role in hyperinflammatory states where conventional therapy fails to work.

CONCLUSION

This case demonstrates that early high-volume CVVHDF with an oXiris filter, combined with aggressive multidisciplinary care, rapidly reversed the lethal triad of septic shock, severe DKA, and stage 3 AKI in a postpartum diabetic patient, leading to full recovery by day 12. Timely cytokine-adsorptive CRRT is the cornerstone of patient survival.

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None

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The Authors agree to be published in the Journal of Society Medicine.

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The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS
All authors made substantial contributions to the case report. PIP was responsible for patient management, data collection, and the initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity, and are accountable for all aspects of the work.

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