Archives of Anesthesiology and Critical Care (In Press); x(x): xx-xx.

Available online at http://aacc.tums.ac.ir

Case Report

Heart, Lung, Kidney Interaction Following Urosepsis and Acute Viral Interstitial Pneumonia

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ARTICLE INFO

Article history:
Received 17 January 2026
Revised 11 February 2026
Accepted 26 February 2026

Keywords:
Organ crosstalk;
Sepsis;
Heart-lung-kidney interaction;
Cardiorenal-cardiopulmonary syndrome

ABSTRACT

Organ-organ interactions play a crucial role in the progression and severity of critical illnesses such as sepsis. Dysregulated interaction among the heart, lungs, and kidneys (mediated by inflammatory, neurohormonal, and metabolic processes) can exacerbate organ dysfunction, contributing to the development of multi-organ failure. We report the case of a 70-year-old woman with significant comorbidities, including type 2 diabetes mellitus (DM-II), chronic kidney disease (CKD), heart failure (HF), nephrolithiasis, and morbid obesity, who was admitted with severe urosepsis caused by extended-spectrum β-lactamase (ESBL)-producing Escherichia coli. Her clinical course was further complicated by acute viral interstitial pneumonia progressing to ARDS. During the ICU admission, the patient experienced a complicated clinical course characterized by mixed septic and cardiogenic shock, right ventricular dysfunction, hypoxemic respiratory failure, and acute-on-chronic kidney injury. Treatment included lung-protective mechanical ventilation, hemodynamic support with inodilator therapy, insulin-dextrose-potassium administration, adjunctive therapies targeting inflammation and endothelial dysfunction, and judicious fluid management. Over the course of hospitalization, cardiac, respiratory, and renal function progressively improved, allowing discharge in a stable condition. This case highlights the clinically important role of organ-organ cross-talk in the development of multi-organ dysfunction during severe sepsis complicated by viral pneumonia. Hemodynamic instability, inflammation-related capillary leakage, and increased pressure within encapsulated organs may interact to perpetuate a cycle of organ injury, particularly in patients with underlying conditions such as heart failure, CKD,

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DOI:

or obesity. Patient-specific management strategies may support organ recovery and improve clinical outcomes by disrupting this cycle.

Introduction

Interactions among organs are critical determinants of morbidity and mortality in critically ill patients [1]. The heart, lungs, and kidneys are closely interconnected through inflammatory, neurohormonal, and intracellular signaling pathways. As a result, injury to one organ can adversely affect the others, particularly during systemic conditions such as sepsis, acute respiratory failure, or metabolic derangements [2-3]. While organ-organ cross-talk is crucial for maintaining physiological homeostasis, disease or injury in one or more organs can trigger functional and structural impairments in others [2]. Changes in hemodynamic status, vascular permeability, and the release of proinflammatory cytokines-including interleukins (IL-1β, IL-2, IL-6, IL-8, and IL-12), tumor necrosis factor-α (TNF-α), and interferon-γ-can initiate a chain of events whereby injury in one organ contributes to dysfunction in others [4-5]. Understanding these interconnected mechanisms is essential for guiding management decisions in critically ill patients. Urosepsis is a severe complication of urinary tract infections (UTI) that can progress to septic shock and multi-organ dysfunction, particularly in patients with underlying comorbidities such as chronic kidney disease (CKD) or diabetes [6-7]. Escherichia coli and Klebsiella species are the main pathogens in urosepsis, but some strains produce extended-spectrum β-lactamases (ESBL), conferring resistance to many β-lactam antibiotics. This makes treatment particularly challenging in these patients and consequently increases the risk of subsequent multiorgan damage [8].

Acute respiratory distress syndrome (ARDS) represents a critical inflammatory injury of the lungs, marked by enhanced vascular permeability and inflammatory cytokine release [9]. Severe hypoxemia, bilateral radiographic infiltrates, and associated hemodynamic alterations are characteristic manifestations of ARDS [10-11]. Among causes occurring outside hospital settings, community-acquired pneumonia (CAP) is the most frequent trigger of ARDS. In recent years, respiratory viruses have been increasingly detected in patients suffering from severe CAP, ARDS, and sepsis [12-14].

In septic states, maladaptive communication among the heart, lungs, and kidneys contributes to the onset and progression of multiorgan failure [15].

We report the case of a 70-year-old woman with severe urosepsis and concomitant acute viral interstitial pneumonia, whose clinical course was marked by interconnected cardiac, pulmonary, and renal dysfunction. The case emphasizes the difficulties in managing concurrent organ failures and illustrates the critical role of organ-organ crosstalk in patients with severe critically illness.

Case Report

A 70-year-old woman with a 40-year history of type 2 diabetes mellitus (DM-II), CKD, hypertension, chronic heart failure (CHF), obesity (height 160 cm; weight 130 kg; BMI 50.8; intra-abdominal pressure approximately 15 mmHg), and recurrent nephrolithiasis was initially brought to another hospital because of a decreased level of consciousness.

Based on available reports from that facility, she was diagnosed with a UTI, and a urine culture grew ESBL-producing Escherichia coli. Her condition continued to deteriorate, with worsening encephalopathy thought to be secondary to sepsis.

Because of this clinical decline, she was referred to our center, intubated in the emergency department, and then admitted to the intensive care unit (ICU). Baseline and interval laboratory data from the referring hospital, including serum creatinine measurements and detailed neurologic assessments, were not available. Her laboratory findings upon arrival at our hospital are summarized in (Table 1).

Upon arrival in the ICU, the patient was hypotensive, tachypneic, and markedly obtunded, with a Glasgow Coma Scale (GCS) score of approximately 4–5.

Her initial chest X-ray showed bilateral infiltrates (Figure 1). Over the subsequent 48 hours, follow-up CT imaging revealed a rapid progression to acute interstitial pneumonia, ultimately culminating in ARDS, as evidenced by severe hypoxemia with a Partial pressure of arterial oxygen/Fraction of inspired oxygen (PaO_2/FiO_2) ratio below 200.

This cascade, triggered by influenza virus infection, led to diffuse alveolar damage (DAD), hypoxic pulmonary vasoconstriction (HPV), and subsequent right-sided heart failure. Although biomarkers associated with DAD, such as IL-8 or angiopoietin-2, were not assessed, the clinical course and radiologic findings were highly indicative of severe DAD. During this period, the patient developed profound hypoxemia and hypercapnia, resulting in refractory vasodilatory shock that necessitated vasopressor support.

The patient's hemodynamic profile, derived from ultrasonic cardiac output monitoring (USCOM), was as follows: cardiac output (CO) 6 L/min, cardiac index (CI) 2.5 L/min/m², corrected flow time (FTc) 450 ms, and systemic vascular resistance (SVR) 900 dyn·s/cm⁵.

Because she was in septic shock, a norepinephrine infusion was initiated at a rate of 5 µg/min.

Table 1- Laboratory values on admission to the intensive care unit.

Parameter	Value	Reference range	Parameter	Value	Reference range	Parameter	Value	Reference range
Urea (mg/dl)	65	21-43	AST (U/L)	13	Up to 31	Plt (x10^3/µL)	262	150-450
Cr (mg/dl)	2.5	0.8-1.4	ALT (U/L)	17	Up to 31	Neutrophil (%)	92	45-65
CRP (mg/L)	263.6	0-8: Negative	ALP (U/L)	316	110-380	Lymphocyte (0%	4.2	22-40
Troponin-I (ng/ml)	<0.03	Up to 0.1	CPK (U/L)	72	Up to 170	pH	7.11	7.35-7.45
Amylase (lu/L)	38	<100	CPK-MB (U/L)	10	Up to 24	pCO_2 (mmHg)	54.2	35-59
NT-PRO-BNP (pg/ml)	157	Up to 125	WBC (x10^3/µL)	19.8	4-10.5	HCO_3 (mmol/L)	17.1	22-30
LDH (U/L)	471	<530	Hgb (g/dL)	9.3	12.1-15.1			

ALP = Alkaline Phosphatase, ALT = Alanine Aminotransferase, AST = Aspartate Aminotransferase, CPK = Creatine Phosphokinase, CPK-MB = Creatine Phosphokinase-MB, Cr = Creatinine, CRP = C-Reactive Protein, HCO3 = Bicarbonate, Hgb = Hemoglobin, LDH = Lactate Dehydrogenase, NT-PRO-BNP = N-terminal pro–B-type Natriuretic Peptide, pCO2 = Partial Pressure of Carbon Dioxide, pH = Blood pH, Plt = Platelets, WBC = White Blood Cells.

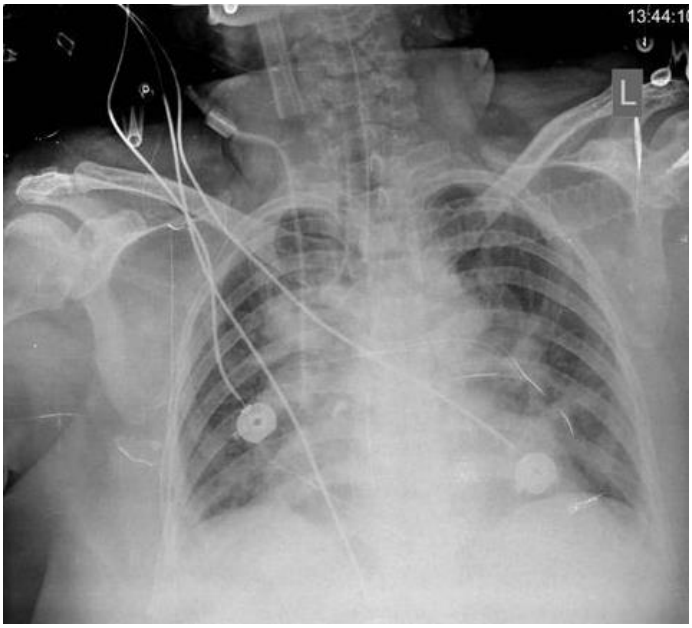

Figure 1- Initial chest CT scan showing bilateral pulmonary infiltrates consistent with viral interstitial pneumonia.

Vasopressin was also administered at a dose of 40 units diluted in 200 mL of normal saline and infused at a rate of 10 mL per hour, adjusted according to mean arterial pressure (MAP), SVR, and oxygen delivery (DO_2). The infusion was to be held if SVR exceeded 1800 dyn·s/cm^5, MAP was above 70 mmHg, and DO_2 was below 300 mL/min.

Two days after initiation of hemodynamic support measures, the patient stabilized, and an echocardiography consultation was requested for prognostication and optimization of pharmacotherapeutic management. Transthoracic echocardiography (TTE) revealed diastolic dysfunction with a reduced ejection fraction (EF) of 40–45%, moderate right ventricular dilation with systolic dysfunction, and right-sided heart failure with an estimated pulmonary artery pressure (PAP) of 40–50 mmHg. These findings were consistent with sepsis-associated cardiomyopathy and right-sided heart failure driven by HPV, sepsis-induced myocardial depression, and possible viral myocarditis.

Consequently, the norepinephrine requirement decreased, vasopressin was discontinued, and dobutamine was initiated at a dose of 3–5 µg/kg/min to enhance global oxygen delivery and ventilation.

A combination of low-dose norepinephrine and dobutamine was maintained for 5 days as part of the management of mixed (septic–cardiogenic) shock. The patient's hemodynamic profile has been improved, and the new tracheal culture was reported to be positive for growing gram-negative coccobacilli.

In addition, lung compliance was below 20 mL/cmH_2O (PaO_2/FiO_2 < 200), and airway resistance exceeded 30 cmH_2O/L/s, with a pH < 7.3 and $PaCO_2$ > 47 mmHg. These abnormalities were managed using airway pressure release ventilation (APRV) in conjunction with dexamethasone 8 mg twice daily and, subsequently, aminophylline administered as a 3 mg/kg loading dose over 1 hour, followed by a continuous intravenous infusion of 10 mg/h. The aminophylline infusion was titrated based on the patient's $PaCO_2$ levels and heart rate. Using APRV mode improved functional residual capacity and facilitated gradual improvement in oxygenation and ventilation.

During her ICU stay, the patient developed supraventricular tachyarrhythmia that was initially managed with intravenous digoxin 0.25 mg as a stat dose and an intravenous amiodarone 150 mg bolus, followed by a continuous infusion at 30-50 mg/h for 24 hours.

Amiodarone was subsequently discontinued due to concern for worsening the patient's lung injury. The arrhythmia was then managed with rate control using ranolazine 500 mg daily and bisoprolol 5 mg daily. Magnesium drip was limited because of ongoing acute kidney injury (AKI) secondary to urosepsis.

Neurologically, the patient exhibited persistent unresponsiveness for ten days (GCS 3-5), raising concern for ongoing or subclinical seizure activity (non-convulsive seizure established in electroencephalography (EEG) monitoring) and necessitating initiation of antiseizure therapy with levetiracetam 2 g intravenously as a stat dose followed by 500 mg intravenously twice daily, and lacosamide 200 mg as a stat dose followed by 100 mg twice daily. Supportive neuroprotective measures included limiting environmental stimulation through control of ambient noise (<50 dB) and light exposure (<200 lux). Brain CT imaging demonstrated no structural lesions, supporting a diagnosis of metabolic encephalopathy associated with sepsis.

Management of her AKI on CKD included albumin-assisted diuresis with furosemide, administered as Ringer's lactate (200 mL) combined with 50 mL of albumin and 200 mg of furosemide, at a dose ranging from 0.1 mg/kg/hr up to 0.75 mg/kg/hr. Dosing was adjusted according to urine output, arterial blood gas analysis, and hemodynamic profile. This strategy achieved a daily urine output of 5–6 liters, suggesting fluid toxicity and established volume overload secondary to right-sided heart failure.

The patient's serum creatinine gradually increased during hospitalization, peaking at 3.2 mg/dL, and subsequently improved to 0.8 mg/dL by the time of her first ICU discharge. Over the subsequent days, the patient's oxygenation, ventilation, hemodynamics, cardiac output, and renal function continued to improve (SVR 1700 dyn·s/cm^5, CO=4 L/min, CI=2.5 L/min/m^2, FTc=350 ms, DO_2=560 mL/min). The patient showed progressive clinical improvement, allowing discontinuation of vasopressor support and transition from invasive mechanical ventilation to noninvasive ventilatory assistance. Her level of consciousness returned to normal, blood glucose levels were effectively controlled with insulin treatment, and inflammatory markers gradually improved. Although she was transferred from the ICU in a stable condition, she continued to experience diaphragmatic dysfunction and ICU-acquired weakness, along with exacerbation of her pre-existing chronic heart failure.

Three days after transfer to the ward, the patient was readmitted to the ICU with severe hemodynamic instability, decreased level of consciousness, and respiratory failure, necessitating reintubation, as she was unable to tolerate noninvasive ventilation (NIV) due to profound muscle weakness. USCOM measurements revealed markedly elevated SVR=2700 dyn·s/cm^5 with low CO=2.8 L/min and CI=1.2 L/min/m^2, indicating critically impaired perfusion. Moreover, an inodilator-directed strategy was reinitiated. Dobutamine at 10–20 µg/kg/min, in combination with insulin–dextrose infusion, was initiated, resulting in rapid improvement in CO and urine output, followed by milrinone administration at 2 mg/h. An insulin–dextrose–potassium cocktail was used adjunctively to provide additional inodilator effects.

In addition to standard hemodynamic and supportive care, the patient received adjunctive therapies, including intravenous vitamin B12 (2000 µg daily for 3 days), intravenous immunoglobulin (IVIG; 5 g infused over 12 hours), and melatonin (9 mg three times daily for 3 days), to help stabilize the endothelium, reduce inflammation, and support multiorgan recovery during her ICU stay.

Renal function subsequently recovered, with serum creatinine decreasing from 1.9 to 0.9 mg/dL. Hemodynamic status stabilized and oxygenation improved, allowing transfer to the ward and eventual discharge home in stable condition.

At outpatient follow-up, the patient demonstrated features consistent with post-intensive care syndrome (PICS), including nightmares, insomnia, iron deficiency anemia, depression, fatigue, and intermittent exacerbations of heart failure. Her daughter also experienced significant anxiety consistent with PICS-Family (PICS-F). She remains under multidisciplinary follow-up (Figure 2).

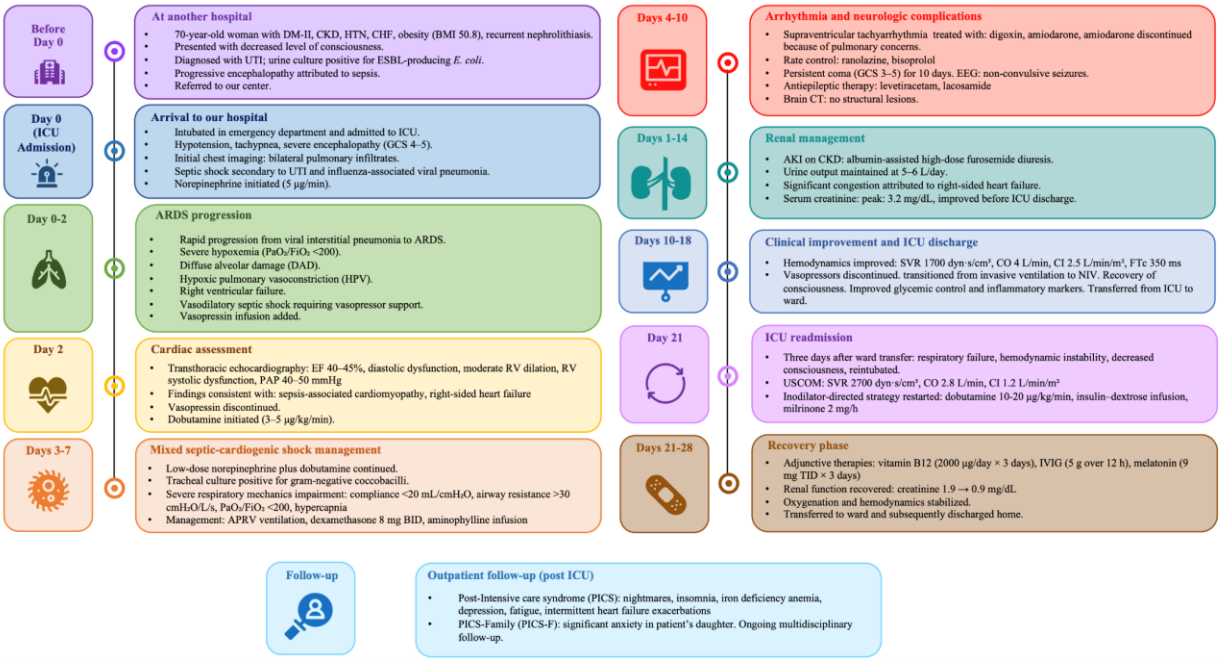

Figure 2- Timeline of major clinical events, hemodynamic changes, and organ function during hospitalization.

Discussion

Here, we describe the complex and dynamic interplay between sepsis, ARDS, and multi-organ dysfunction in a 70-year-old woman with significant cardiac dysfunction, in whom initial urosepsis caused by ESBL-producing *Escherichia coli* triggered a cascade of systemic inflammation, immune dysregulation, and hemodynamic collapse. As her condition progressed, she developed a combination of septic and cardiogenic shock, hypoxemic respiratory failure, and acute-on-chronic kidney injury.

Sepsis is defined as a dysregulated host response to infection that leads to multiorgan dysfunction [16]. Rather than arising from isolated organ injury, this dysfunction reflects the convergence of interconnected inflammatory pathways and profound cellular and metabolic reprogramming. These changes extend beyond the primary infectious focus and propagate across organs through integrated communication networks involving cellular signaling, circulating inflammatory mediators, and neurohormonal regulation. As a result, impairment in one organ system may initially elicit compensatory responses in others; however, when sustained, these responses become maladaptive, intensifying tissue damage and ultimately driving progressive multi-organ failure [15,17].

Since cases similar to our patient have been reported rarely, we were unable to perform a direct comparison with previously published cases. Therefore, instead of a case-to-case comparison, we focused on analyzing the underlying nature of organ-to-organ interactions in sepsis and the organ cross-talk notion as they relate to our patient.

In the setting of sepsis following a severe viral infection, a complex interplay emerged among the heart, lungs, and kidneys [18-19]. Severe viral respiratory infections, particularly viral pneumonia, may result in widespread injury to the alveolar–capillary membrane, commonly known as diffuse alveolar damage (DAD). This pathological process affects both the alveolar epithelial cells and the pulmonary vascular endothelium, disrupting surfactant activity and increasing vascular permeability. Consequently, protein-rich fluid accumulates within the alveoli, leading to alveolar collapse, reduced gas exchange efficiency, and the development of hypoxemia.

Moreover, heterogeneous involvement of alveoli produces uneven ventilation, giving rise to ventilation-perfusion mismatch. Collectively, vascular compression secondary to edema, together with inflammation-induced disturbances in vasomotor regulation, leads to a significant increase in pulmonary vascular resistance [20-23].

A reduction in alveolar oxygen tension triggers hypoxic pulmonary vasoconstriction (HPV), a physiological reflex involving constriction of small pulmonary arteries in hypoxic regions of the lung. This response redistributes blood flow away from poorly ventilated alveoli toward better-ventilated areas, thereby improving ventilation-perfusion matching and gas exchange efficiency [24-26]. At the cellular level, hypoxia modulates pulmonary arterial smooth muscle cell function through disruption of mitochondrial redox

homeostasis. This leads to inhibition of potassium channel activity, membrane depolarization, and increased calcium influx, ultimately resulting in vasoconstriction. When hypoxia is sustained or diffuse, as in severe pneumonia and diffuse alveolar damage (DAD), this normally adaptive mechanism becomes generalized, contributing to a marked rise in pulmonary vascular resistance [22,24,26-27].

The combined effects of alveolar fluid accumulation, inflammation-induced pulmonary damage, and widespread hypoxic vasoconstriction lead to a rise in pulmonary vascular resistance, resulting in increased pulmonary artery pressure and a significant burden on the right ventricle [22].

Persistent elevation of pulmonary vascular resistance imposes a substantial pressure load on the right ventricle, leading to progressive dilation and decline in right ventricular function. When compensatory mechanisms are exhausted, right ventricular failure may develop, presenting as acute cor pulmonale. These hemodynamic effects are often exacerbated by positive-pressure mechanical ventilation, which increases intrathoracic pressure and further impairs venous return [22,28-29]. Additionally, concurrent sepsis plays a critical role in worsening myocardial dysfunction and pulmonary vascular dysregulation, thereby establishing a self-perpetuating cardiopulmonary cycle referred to as sepsis-induced cardiomyopathy (SICM) [30].

SICM is a well-recognized but often underappreciated manifestation of severe sepsis and septic shock, typically characterized by reversible myocardial depression that may affect both systolic and diastolic function [31-32]. In our patient, the interpretation of myocardial dysfunction was particularly challenging due to the coexistence of pre-existing heart failure and superimposed myocarditis. Despite this adverse cardiac milieu, the patient gradually recovered to her pre-admission baseline cardiac function. This pattern of improvement supports a substantial contribution of sepsis-induced cardiomyopathy (SICM) rather than irreversible structural myocardial injury. The reversible nature of SICM likely facilitated hemodynamic stabilization, vasopressor weaning, and successful ICU discharge.

Beyond myocardial dysfunction, severe sepsis profoundly disrupts systemic fluid homeostasis. In critical illness such as sepsis and other severe inflammatory states, as seen in this patient, normal fluid balance is significantly altered [33-34]. Under physiological conditions, circulating blood volume and interstitial fluid distribution are tightly regulated to maintain adequate organ perfusion and oxygen delivery. In contrast, widespread inflammation (driven by cytokine release and endothelial injury) disrupts this balance by increasing capillary permeability, permitting fluid to escape the intravascular space and accumulate within surrounding tissues. In severe inflammatory states such as sepsis and septic shock, the release of inflammatory mediators further damages the endothelial glycocalyx, thereby exacerbating capillary leak and vascular permeability that cause third-spacing of fluid [35-36]. This third-spacing of fluid is particularly harmful in organs with tight anatomical boundaries, such as the kidneys, lungs, brain, and abdominal viscera, because they cannot easily expand to accommodate excess fluid [36-39]. As a result, these organs experience swelling and rising interstitial pressures, which compress small blood vessels and reduce effective perfusion. These interconnected mechanisms can lead to clinical syndromes such as cardio-renal, cardio-intestinal, and cardio-cerebral dysfunction, whereby impairment of one organ system exacerbates injury in others through common hemodynamic and inflammatory pathways [36,40-43].

Furthermore, excessive fluid administration or continued fluid accumulation secondary to persistent capillary leak can lead to increased pressure within these closed anatomical compartments, most notably the abdominal cavity. Elevated intra-abdominal pressure compromises venous return and compresses the vascular supply to the kidneys, gastrointestinal (GI) tract, and other encapsulated organs, thereby further impairing regional perfusion and promoting acute organ dysfunction. This pathophysiological process is well recognized in conditions such as intra-abdominal hypertension and abdominal compartment syndrome, both of which are associated with reduced multi-organ perfusion and, if left untreated, progressive organ failure [44-47].

Our patient was morbidly obese, with an intra-abdominal pressure of approximately 15 mmHg. Growing evidence supports the concept that adipose tissue acts as an active metabolic and immunologic organ rather than serving solely as an energy reservoir [48-49]. During severe infections such as sepsis and periods of intense systemic stress, hypoxia and dysfunction within adipose tissue can promote the release of damage-associated molecular patterns (DAMPs), proinflammatory cytokines, including IL-6, TNF-α, and high mobility group box 1 (HMGB1), as well as free fatty acids. These mediators amplify the systemic inflammatory response and may contribute to injury in distant organs, particularly the lungs, heart, and kidneys. In addition, adipose tissue-derived inflammatory and metabolic factors can worsen endothelial dysfunction, activate coagulation pathways, and promote insulin resistance [50]. Collectively, these mechanisms may help explain the pronounced vasoplegia, hypermetabolic state, and metabolic derangements observed in our patient. Severe obesity may further worsen intra-abdominal hypertension, impair venous return, and intensify adverse cardio-renal and cardio-pulmonary interactions. While mild to moderate adiposity has been associated with

improved outcomes in some critically ill populations, a phenomenon commonly described as the "obesity paradox", severe obesity [50], as observed in our case, has consistently been associated with an increased risk of multiorgan failure and mortality. Thus, the combined effects of hemodynamic instability, inflammation-induced capillary leak, and pressure-related tissue compression promote fluid accumulation within vital organs. This not only impairs organ function directly but also perpetuates a vicious cycle in which progressive organ failure further worsens circulatory dysfunction.

In addition, non-calcium-based nephrolithiasis was identified, likely secondary to recurrent urinary tract infections in the setting of immune dysfunction, which ultimately contributed to the development of sepsis [51-53]. The interplay of sepsis, nephrolithiasis, elevated intra-abdominal pressure, abdominal compartment syndrome, and excessive fluid accumulation ultimately culminated in acute tubular necrosis (ATN) and acute-on-chronic renal failure [54-59].

Given this context, meticulous fluid management was a cornerstone of therapy for these patients. Continuous infusion of furosemide in combination with hypertonic saline facilitated restoration of fluid balance and stabilization of acid-base disturbances in our patient [60].

Complementing this, the use of APRV as a lung-protective strategy for DAD, together with dobutamine for inotropic support and minimization of norepinephrine dosage to improve tissue perfusion, contributed to improved hemodynamics and oxygenation and to reduced ICU stay and mortality [61-64].

Furthermore, administration of insulin, dextrose-potassium as an ionodilator, and continuous heparin infusion, aimed at preservation of the endothelial glycocalyx and attenuation of inflammatory markers, was associated with improved respiratory mechanics, increased lung compliance, and reduced airway resistance [65-69]. The anti-inflammatory and endothelial-stabilizing effects of colchicine, along with its urate-lowering properties, likely provided additional therapeutic benefit in this patient [70-71].

Taken together, adjunctive therapy with vitamin B12, heparin, colchicine, and melatonin, alongside insulin, facilitated successful weaning from norepinephrine and transition to midodrine (5 mg three times daily), which was subsequently tapered and discontinued, reflecting sustained improvement in vascular tone and hemodynamic stability.

Numerous studies have supported the therapeutic role of high-dose vitamin B12 in refractory vasodilatory shock associated with myocardial dysfunction [72-73]. In addition, melatonin was administered to support mitochondrial recovery and improve mitochondrial function in the setting of sepsis-induced myocardial dysfunction [74-75].

Moreover, sepsis-related complications, including encephalopathy, cardiomyopathy, and renal dysfunction, showed gradual improvement following IVIG therapy and comprehensive supportive care [63]. This case underscores the importance of recognizing intricate multiorgan interactions, implementing individualized fluid and hemodynamic management strategies, and applying lung-protective ventilation in patients with severe viral pneumonia complicated by sepsis and multiorgan dysfunction.

This case report has several limitations. First, it involves a single patient, and thus the observed associations between interventions and clinical improvement cannot be generalized or interpreted as causal. Second, the lack of specific inflammatory and endothelial biomarkers limits mechanistic interpretation. Further studies are needed to validate and extend these findings.

Conclusion

This case underscores the importance of organ-organ interactions in driving the progression of multi-organ dysfunction in patients with severe sepsis and concomitant viral pneumonia. A multidisciplinary therapeutic approach incorporating lung-protective ventilatory support, tailored fluid and hemodynamic management, inodilator therapy, and adjunctive treatments targeting inflammation and endothelial dysfunction may facilitate organ stabilization and enhance patient outcomes. Awareness of these complex pathophysiological relationships is crucial for guiding management decisions and optimizing care in critically ill individuals.

Acknowledgment

The authors acknowledge the use of ChatGPT (GPT-5 mini) for native English language editing of the manuscript.

Ethics approval and consent to participate

This case report was carried out in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent for participation and publication of clinical information was obtained from the patient and their legal guardian.

Consent for publication

Written informed consent for publication of this case report, including any accompanying images, was obtained from the patient and their legal guardian, confirming their agreement to share the medical information.

Authors' contributions

FA: formal analysis, investigation, methodology, visualization, writing original draft, review, and editing. **MM:** conceptualization, methodology, project administration, supervision, validation, review, and editing. **LSH:** review and editing, visualization, data curation. **KS:** review and editing, visualization, data curation. **MA:** review and editing, visualization, data curation. **AN:** writing original draft, visualization, literature review, and editing. **SHS:** review and editing. **PK:** review and editing. **AS:** patient management. **AM:** review and editing.

Data availability

Available from the corresponding author upon request.

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