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CASE REPORT

# Hyperinflammatory septic shock during immune reconstitution: Temporal association with HA-330 hemoadsorption: A case report

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All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author. (Surat Tongyoo, email address: surat.ton@mahidol.ac.th)

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## ABSTRACT:

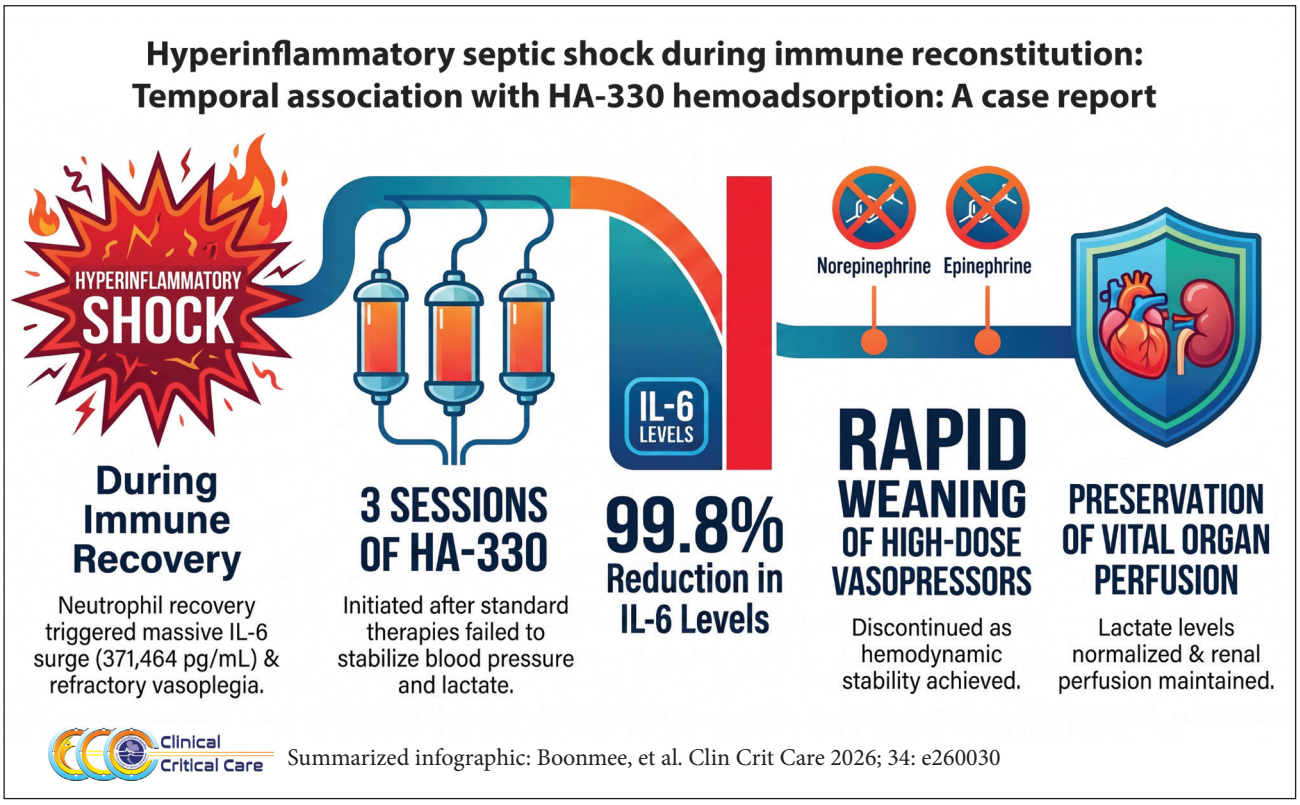
**Introduction:** Immune-recovery phases after intensive chemotherapy can abruptly trigger hyperinflammatory responses that amplify vasoplegia in septic shock. Recognizing this phenotype is crucial, particularly when the inflammatory burden is disproportionate to the source of infection.

**Case presentation:** A 56-year-old woman with acute B-cell lymphoblastic leukemia developed sudden vasoplegic shock during profound neutropenia with Granulocyte colony-stimulating factor (G-CSF) support following administration of hyperfractionated cyclophosphamide, vincristine, adriamycin (doxorubicin), and dexamethasone ( Hyper-CVAD). She presented with severe hyperlactatemia (11 mmol/L), C-reactive protein (CRP) 232 mg/L, procalcitonin >100 ng/mL, and extreme elevation of interleukin-6 (IL-6) (371,464 pg/mL). Despite timely broad-spectrum antibiotics, hydrocortisone, aggressive fluid resuscitation, and high-dose dual vasopressors, profound vasoplegia persisted. Urine output remained partially preserved (0.33–0.93 mL/kg/h) in the setting of predominantly vasodilatory circulatory failure.

**Intervention and outcome:** Three sessions of HA-330 hemoadsorption (Jafron Biomedical, Zhuhai, China) over 72 hours were initiated after failure of conventional therapy. IL-6 declined from 371,464 to 23,629 pg/mL after the first session and to 517 pg/mL after the third session. This was temporally associated with progressive reduction in vasopressor requirements and lactate normalization. Hemodynamic stability was achieved with improved organ perfusion.

**Conclusions:** This case highlights a cytokine-dominant vasoplegic septic shock phenotype during immune recovery, marked by extraordinary IL-6 elevation despite partially preserved renal perfusion. The temporal alignment between hemoadsorption, cytokine reduction, and hemodynamic improvement is hypothesis-generating and underscores the potential role of inflammatory burden in certain hyperinflammatory shock states.

**Keywords:** Septic shock; Hemoperfusion; HA-330




## INTRODUCTION

Septic shock in immunocompromised patients is shaped by pathogen burden and dynamic fluctuations in immune function. During intensive chemotherapy, the transition from profound neutropenia to early immune recovery can trigger abrupt cytokine surges that can cause hemodynamic deterioration disproportionate to the underlying source of infection [1,2]. This hyperinflammatory phenotype may overlap with conventional septic shock but be amplified during immune recovery, resembling Immune Reconstitution Inflammatory Syndrome (IRIS)-like or neutrophil recovery syndromes [1,3]. Interleukin-6 (IL-6) is a key mediator of sepsis-associated vasoplegia, and transcriptomic data link extreme inflammatory signatures to endothelial dysfunction and catecholamine resistance [4,5]. These insights have prompted investigation of adjunctive cytokine-modulating strategies for selected hyperinflammatory states. The HA-330 hemoadsorption cartridge removes mid–molecular-weight cytokines, with mechanistic and early clinical reports demonstrating reductions in inflammatory markers and clinical stabilization in several observational studies [6,7]. However, descriptions of septic shock occurring during neutrophil recovery with an exceptionally high cytokine burden are rare, and data on the use of HA-330 in this specific phenotype are limited. This case illustrates a severe vasoplegic shock presentation with markedly elevated IL-6 during immune recovery after intensive chemotherapy and describes the clinical course after adjunctive hemoadsorption.

### KEY MESSAGES:

- Early immune recovery following intensive chemotherapy can trigger an extraordinary cytokine-dominant vasoplegic shock phenotype.
- In such highly selected refractory cases, adjunctive hemoadsorption with HA-330 may be considered a "biologically timed" rescue therapy to support hemodynamic stabilization in conjunction with standard care.

## CASE PRESENTATION

In August 2025 a 56-year-old woman was admitted for induction chemotherapy after a diagnosis of B-cell acute lymphoblastic leukemia (B-ALL). The diagnosis was established by bone marrow biopsy after she presented with abnormal uterine bleeding and pancytopenia. Her medical history included ductal carcinoma in situ of the right breast treated surgically in 2021 and followed by adjuvant tamoxifen therapy.

Induction chemotherapy with the Hyper-CVAD regimen was initiated, together with granulocyte colony-stimulating factor (G-CSF) support. Pancytopenia persisted (white blood cell count 1,400/μL; hemoglobin 8.9 g/dL; platelets 56,000/μL), and by hospital day 9 the absolute neutrophil count (ANC) had declined to 10/μL. On day 12, she developed abrupt clinical deterioration characterized by fever

(38.5 degrees Celsius), tachycardia (128 bpm), hypotension (101/44 mmHg, mean arterial pressure 63 mmHg) with wide pulse pressure (57 mmHg), delayed capillary refill, and severe metabolic acidosis. Laboratory evaluation revealed hyperlactatemia (11 mmol/L) with electrolyte abnormalities (sodium 136 mmol/L, potassium 2.7 mmol/L, chloride 99 mmol/L, bicarbonate 14 mmol/L). Inflammatory biomarkers rose rapidly, with C-reactive protein increasing from 57 to 139 to 232 mg/L and procalcitonin exceeding 100 ng/mL.

Broad-spectrum antimicrobial therapy (meropenem and vancomycin) was administered within one hour, along with aggressive fluid resuscitation, intravenous hydrocortisone, and thiamine. Despite aggressive fluid resuscitation and the administration of hydrocortisone (200 mg/24h), hypotension persisted, requiring escalating doses of norepinephrine up to 0.84 µg/kg/min and epinephrine up to 0.64 µg/kg/min, while the lactate level increased to a peak of 11 mmol/L (time points illustrated in Figure 1, beginning at shock onset, 0 hours). During this refractory period, the patient was intubated for progressive respiratory failure. During the first 24 hours of shock, approximately 8.9 liters of intravenous fluids were administered, including 2.8 liters of crystalloid, blood products, maintenance fluids, and vasopressor carriers. Despite aggressive resuscitation, she remained profoundly vasoplegic. Urine output fluctuated between 25 and 70 mL/h (0.33–0.93 mL/kg/h), indicating partial preservation of renal perfusion despite refractory circulatory failure. Blood cultures were positive for Klebsiella pneumoniae that was sensitive to standard antimicrobial therapy. Urine and sputum cultures were negative, and computed tomography of the abdomen did not reveal an abscess or other source of infection. Bedside echocardiography performed during refractory shock revealed a hyperdynamic left ventricle (LVEF 65%) with a plethoric, non-collapsible inferior vena cava, findings consistent with a vasoplegic rather than cardiogenic shock phenotype (Table 1).

Because vasoplegia and a markedly hyperinflammatory profile persisted (interleukin-6 (IL-6) 371,464 pg/mL, C-reactive protein 232 mg/L, and procalcitonin >100 ng/mL), adjunctive hemoadsorption using the HA-330 cartridge (Jafron Biomedical, Zhuhai, China) was initiated approximately 12 hours after the onset of shock (Figure 2). Three sessions were performed over 48–72 hours via a central venous catheter (Figure 3). Each hemoadsorption session was performed for approximately 4 hours, with an initial blood flow rate of 100 mL/min that was gradually increased to 150 mL/min. The circuit was primed with heparinized saline; however, no systemic anticoagulation was administered due to baseline coagulopathy. No circuit clotting or procedure-related complications were observed. Interleukin-6 (IL-6) was measured using an electrochemiluminescence immunoassay (ECLIA). Because concentrations exceeded the assay range, samples were diluted according to the manufacturer's protocol. No analytical interference was reported, and results were confirmed by repeated measurements. The institutional reference range is 0–7 pg/mL. Meropenem and vancomycin were re-administered at full supplemental doses immediately after each hemoperfusion session to mitigate potential drug adsorption and ensure therapeutic levels. Therapeutic drug monitoring confirmed adequate dosing, with a vancomycin trough level of 19.59 µg/mL measured during the course of hemoadsorption sessions. Serial biomarkers demonstrated a marked decline in IL-6, from 371,464 pg/mL before treatment to 23,629 pg/mL after the first session and 517 pg/mL after the third session. Procalcitonin decreased from >100 ng/mL to 88 ng/mL, while C-reactive protein peaked before gradually improving as the hemodynamic status stabilized.

Serum lactate initially decreased but rose transiently during attempts to wean vasopressors, coinciding with evolving digital ischemic changes. Computed tomography of the abdomen did not show evidence of mesenteric ischemia. Vasopressor requirements declined in a stepwise fashion,

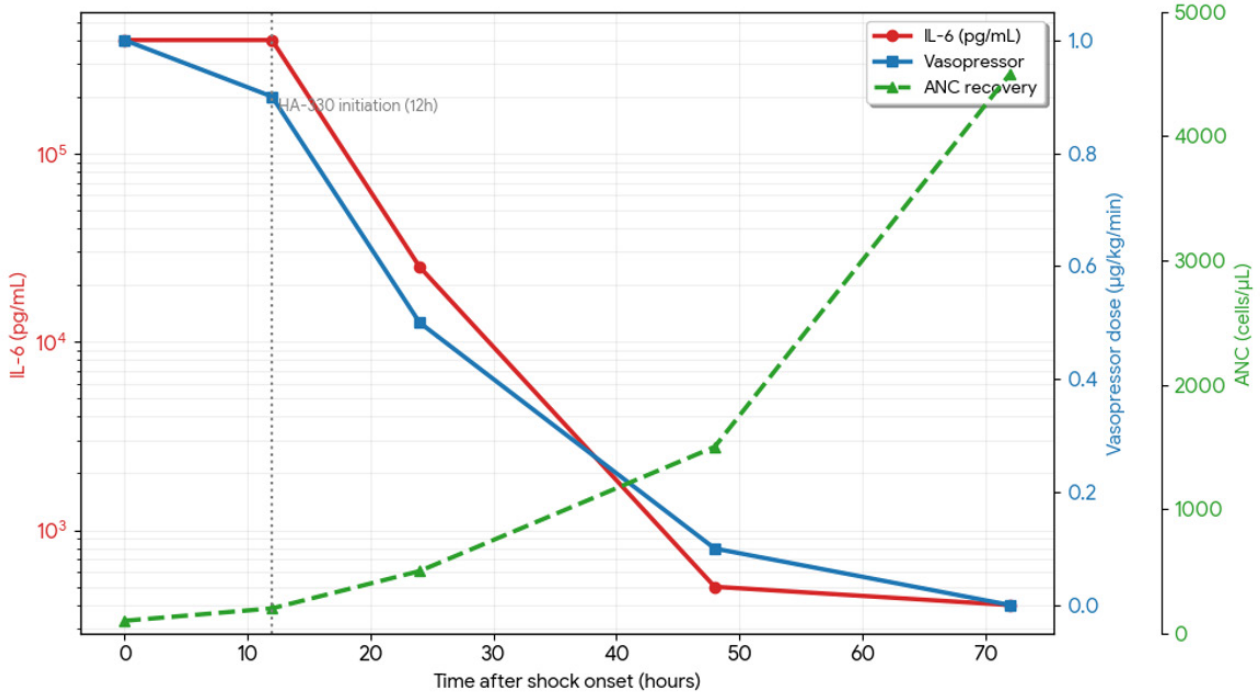



**Figure 1.** Temporal trend of IL-6 concentration (logarithmic scale), absolute vasopressor doses (µg/kg/min), and absolute neutrophil count (ANC) recovery following HA-330 hemoperfusion. Time 0 hours represents the onset of shock.

**Table 1.** Bedside echocardiographic findings during refractory shock.

| Parameter | Finding | Clinical interpretation |
|---|---|---|
| Left Ventricle (LV) | Hyperdynamic (LVEF 65%) | Preserved systolic function; argues against predominant LV systolic failure as the cause of shock. |
| Right Ventricle (RV) | Non-distended | No signs of acute right heart failure or massive pulmonary embolism. |
| Valvular Function | No significant abnormalities | No significant valvular abnormality identified on bedside echocardiography. |
| IVC Diameter | 2.1 cm | Plethoric IVC; suggests adequate/high preload. |
| IVC Distensibility Index | < 18% | Plethoric, minimally variable IVC, suggesting elevated right-sided filling pressure. |
| Hemodynamic Phenotype | Vasoplegia | Findings consistent with a vasodilatory hemodynamic profile. |



**Figure 2.** HA-330 hemoadsorption cartridge and circuit set-up.

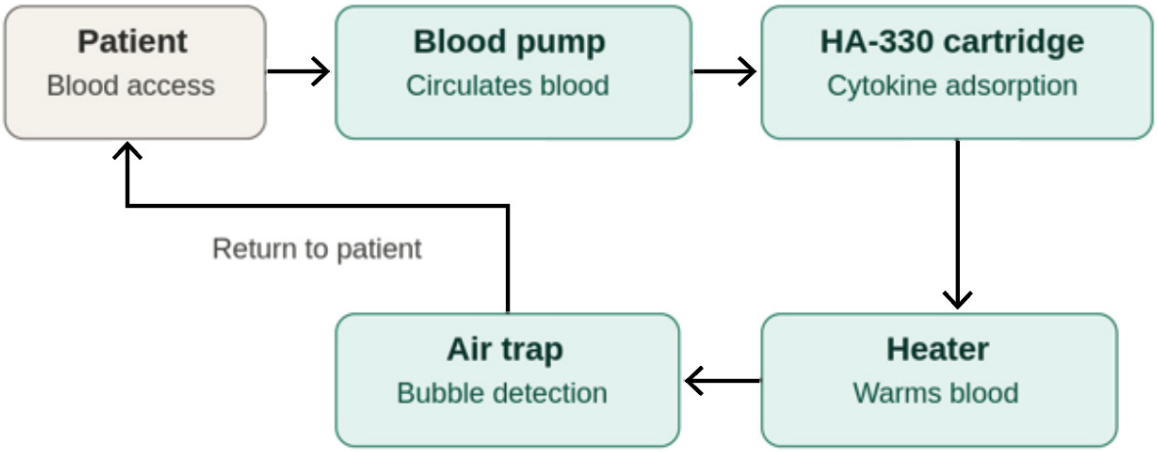



**Figure 3.** Schematic of the standalone HA-330 hemoperfusion circuit.

**Table 2.** Illustrates the temporal relationship between cytokine burden, hemoadsorption sessions, vasopressor requirements, and hematologic recovery, demonstrating a stepwise reduction in IL-6 and catecholamine dependency following early HA-330 initiation.

| Day | Intervention summary | IL-6 (pg/mL) | PCT (ng/mL) | Lactate (mmol/L) | Temp (degree celsius) | $ScvO_2$ (%) | Vasopressor Dose (µg/kg/min) | MAP (mmHg) | WBC ($\times 10^3$/µL) | ANC (cells/µL) | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 0 | Baseline neutropenia | - | <0.5 | 1.2 | 40 | | - | 70–75 | 0.3 | 80 | Hemodynamically stable |
| 1–4 | Sepsis onset continuous IV hydrocortisone (200 mg/24h) | >370,000 | >100 | 11 | 38.5 | 67.5 | NE 0.84 + Epi 0.64 | 55–70 (labile) | 0.2 | <100 | Refractory vasople-gia |
| 5 | 1st HA-330 | 371,464 | 88 | 7.5 | 38.1 | 81.6 | NE 0.68 + Epi 0.52 | 60–70 (vasopressor dependent) | 0.3 | 120 | Modest improvement |
| 6 | 2nd HA-330 | 23,629 | 58 | 4.8 | 37.7 | | NE 0.42 + Epi 0.24 | ≥65 with support | 0.8 | 400 | MAP improved |
| 7 | 3rd HA-330 | 517 | 22 | 1.9 | 37.8 | 66.2 | NE 0.08 | Stable >65 | 1.5 | 1,200 | Weaning vasopressors (NE 0.08 µg/kg/min) vancomycin trough level was therapeutic (19.59 ug/mL), |
| 8–9 | Weaning | - | - | <2 | 36.8 | | - | Stable | 3.8 | 4,500 | Clinical stabilization |

NE: norepinephrine; Epi: epinephrine. Cytokine measurements were performed using the same assay platform throughout. *Day 5 IL-6 reflects the pre-treatment baseline; Day 6 and Day 7 values reflect levels measured after the preceding HA-330 session (i.e., 23,629 pg/mL after the 1st session and 517 pg/mL after the 3rd session).

with epinephrine discontinued after 55 hours and norepinephrine after 72 hours. The lactate level normalized, urine output remained preserved, and overall organ perfusion improved. Serial hemodynamic monitoring demonstrated a transition from a supranormal $ScvO_2$ (81.6 percent) during refractory shock to physiological levels (66.2 percent) in parallel with lactate clearance, reflecting restored systemic oxygen utilization (Table 2).

## DISCUSSION

In immunocompromised hosts, septic shock is a dynamic syndrome influenced by pathogen burden and fluctuations in immune function. In patients who receive intensive chemotherapy, the transition from profound neutropenia to early immune recovery can trigger abrupt inflammatory escalation, occasionally resulting in circulatory deterioration disproportionate to the source of infection [1,2]. In this case, the close temporal association between neutropenia, early immune recovery, and rapid progression to vasoplegic shock suggests an immune-recovery–associated inflammatory surge contributed to the clinical presentation. Rather than representing a distinct entity from conventional septic shock, this presentation may reflect an immune-recovery–associated amplification of the inflammatory response to infection.

A striking feature of this case was the extraordinarily elevated interleukin-6 (IL-6) concentration (371,464 pg/mL), which markedly exceeded the critical mortality-associated threshold of 15,000 pg/mL identified in a large cohort of patients with severe sepsis and septic shock, beyond which mortality risk has been reported to exceed survival probability [8].

Although IL-6 itself is not a direct therapeutic target, transcriptomic and systems-level analyses have shown that severe inflammatory signatures correlate with vasoplegia, endothelial dysfunction, and catecholamine resistance [4,5]. The coexistence of profound vasoplegia and fluctuating urine output suggests a circulatory state dominated by macrovascular vasodilation rather than global hypoperfusion. Simultaneous abnormalities in lactate levels and peripheral perfusion indices suggested concurrent microcirculatory dysfunction. The combination of fluctuating macrocirculatory and microcirculatory parameters, a gram-negative infection, and the patient being in the early immune recovery period raised concern for a cytokine-dominant vasoplegic phenotype. This supported the decision to use HA-330 hemoadsorption as an adjunctive intervention after conventional therapy, including antimicrobial treatment, corticosteroids, fluid resuscitation, and high-dose vasopressors failed to achieve hemodynamic stabilization.

In this case, treatment failure was defined as the persistence of vasoplegic shock and hyperlactatemia despite optimized standard therapy, including empirical antimicrobial therapy, adequate fluid resuscitation, and early corticosteroid administration. The decision to initiate hemoadsorption was driven by the failure to respond to escalating vasopressor support, while the remarkably high IL-6 level (>370,000 pg/mL) further supported the presence of a hyperinflammatory phenotype.

The HA-330 cartridge is designed to adsorb mid–molecular-weight inflammatory mediators [9,10]. Mechanistic and early clinical evidence describe the attenuation of the cytokine burden and associated hemodynamic improve-

ment in certain hyperinflammatory states [6,7]. Case series in pediatric oncology and abdominal septic shock populations have also reported reductions in inflammatory markers with accompanying hemodynamic improvement [7,11]; for example, Sazonov et al. reported mean reductions of approximately 72% in CRP and 89% in procalcitonin following HA-330 hemoadsorption in septic pediatric oncology patients [7], a magnitude of biomarker clearance broadly comparable to the 99.8% reduction in IL-6 observed in the present case. In this case, IL-6 levels decreased substantially following hemoadsorption sessions that were temporally associated with reductions in vasopressor requirements and lactate normalization. However, temporal association does not establish causality, and clinical improvement likely reflects the combined effects of antimicrobial therapy, corticosteroids, and natural immune recovery. Unlike renal replacement therapy (e.g., CRRT), hemoadsorption does not allow conventional dose titration and is primarily determined by treatment timing and duration. In this case, early initiation of treatment was aligned with the presumed hyperinflammatory phase. There are several limitations that must be acknowledged. Clinical improvement likely reflected the combined effects of pathogen clearance, natural immune recovery, corticosteroid therapy, supportive management, and hemoadsorption. The intermittent measurement of cytokines limited the interpretation of removal kinetics, and the proportion of IL-6 decline attributable to the HA-330 cartridge cannot be determined. Hemoadsorption cartridges are also recognized as potentially procoagulant, making anticoagulation and circuit monitoring important considerations [12]; in this case, the absence of systemic anticoagulation due to baseline coagulopathy necessitated close monitoring of the circuit and coagulation parameters throughout treatment, although no circuit clotting or bleeding complications occurred.

Additionally, hemoadsorption using resin-based hemoperfusion cartridges may alter antibiotic pharmacokinetics. Previous studies have reported variable degrees of antimicrobial removal across hemoadsorption platforms, including HA-330 [13,14]. Although antibiotics were readministered after each session in this case, the extent of drug clearance is uncertain.

Furthermore, the possibility of regression to the mean cannot be excluded because the intervention was initiated at the peak of clinical and biochemical severity. The observed improvement might also be subject to time-lag bias, where the resolution of shock coincides with the cumulative effects of standard therapies and the natural course of the disease over time, rather than being solely attributable to the extracorporeal therapy.

Despite these limitations, several practical insights emerge from this case study. First, immune recovery after chemotherapy may precipitate abrupt hyperinflammatory deterioration, and recognizing this pattern may assist clinicians to interpret sudden vasoplegia that appears disproportionate to the source of infection. Second, when considered alongside bedside indicators such as partially preserved urine output despite high vasopressor requirements, extremely elevated inflammatory biomarkers may help identify cytokine-dominant shock states that are not fully captured by conventional severity scores [4,5]. Third, in carefully selected patients with compatible physiology, adjunctive hemoadsorption may be considered together with careful monitoring of hemodynamics, inflammatory markers, and antimicrobial therapy.

This case does not define patient selection criteria or the optimal timing for hemoadsorption. Instead, it highlights the physiological complexity of shock in immunocompromised hosts and underscores the importance of integrating clinical, biochemical, and temporal information when evaluating refractory vasoplegic presentations. The observations should be viewed as hypothesis-generating rather than evidence of therapeutic efficacy.

### Clinical implications

1. Immune-recovery phases after chemotherapy can trigger extreme hyperinflammatory shock.
2. Extremely high IL-6, interpreted alongside hemodynamic and perfusion parameters, may help characterize a cytokine-dominant vasoplegic phenotype.
3. Adjunctive hemoadsorption may be considered in carefully selected cases when conventional therapy fails, although its routine use cannot be recommended outside of research protocols.

## CONCLUSION

This case highlights a cytokine-dominant vasoplegic septic shock phenotype during immune recovery, marked by extraordinary IL-6 elevation with preserved renal perfusion. The temporal alignment between hemoadsorption, cytokine reduction, and hemodynamic improvement is hypothesis-generating and underscores the potential role of inflammatory burden in certain hyperinflammatory shock states.

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## REFERENCES


1. Mokart D, Kipnis E, Guerre-Berthelot P, Vey N, Capo C, Sannini A, et al. Monocyte deactivation in neutropenic acute respiratory distress syndrome patients treated with granulocyte colony-stimulating factor. Crit Care. 2008;12:R17. doi: https://doi.org/10.1186/cc6791.
2. Azoulay E, Darmon M. Acute respiratory distress syndrome during neutropenia recovery. Crit Care. 2010;14:114. doi: https://doi.org/10.1186/cc8198.
3. Chousterman BG, Arnaud M. Is there a role for hematopoietic growth factors during sepsis? Front Immunol. 2018;9:1015. doi: https://doi.org/10.3389/fimmu.2018.01015.
4. Moore AR, Zheng H, Ganesan A, Hasin-Brumshtein Y, Maddali MV, Levitt JE, et al. A consensus immune dysregulation framework for sepsis and critical illnesses. Nat Med. 2025;31:4084–4096. doi: https://doi.org/10.1038/s41591-025-03956-5.
5. Antcliffe DB, Burnham KL, Al-Beidh F, Santhakumaran S, Brett SJ, Hinds CJ, et al. Transcriptomic signatures in sepsis and a differential response to steroids. From the VANISH randomized trial. Am J Respir Crit Care Med. 2019;199:980–986. doi: https://doi.org/10.1164/rccm.201807-1419OC.
6. Waalders NJB, Kox M, Pickkers P. Haemoadsorption to remove inflammatory mediators in sepsis: past, present, and future. Intensive Care Med Exp. 2025;13:38. doi: https://doi.org/10.1186/s40635-025-00740-0.
7. Sazonov V, Abylkassov R, Tobylbayeva Z, Saparov A, Mironova O, Poddighe D. Hemoadsorption with HA-330 adsorber in septic pediatric patients with cancer. Front Pediatr. 2021;9:672260. doi: https://doi.org/10.3389/fped.2021.672260.
8. Ruiz-Rodríguez JC, Chiscano-Camón L, Ruiz-Sanmartin A, Costa-Allué N, Bajaña I, Nicolas-Morales P, et al. Critical hypercytokinemia in sepsis and septic shock: identifying interleukin-6 thresholds beyond which mortality risk exceeded survival probability. J Clin Med. 2026;15(3):1057. doi: https://doi.org/10.3390/jcm15031057.
9. Xu X, Jia C, Luo S, Li Y, Xiao F, Dai H, et al. Effect of HA330 resin-directed hemoadsorption on a porcine ARDS model. Ann Intensive Care. 2017;7:84. doi: https://doi.org/10.1186/s13613-017-0287-0.
10. Ronco C, Bonello M, Bordoni V, Ricci Z, D'Intini V, Bellomo R, et al. Extracorporeal therapies in non-renal disease: treatment of sepsis and the peak concentration hypothesis. Blood Purif. 2004;22(1):164–174. doi: https://doi.org/10.1159/000074937.
11. Sánchez-Morán F, Mateu-Campos ML, Bernal-Julián F, Gil-Santana A, Sánchez-Herrero Á, Martínez-Gaspar T. Haemoadsorption combined with continuous renal replacement therapy in abdominal sepsis: case report series. J Pers Med. 2023;13(7):1113. doi: https://doi.org/10.3390/jpm13071113.
12. Li Y, Han M, Yang M, Su B. Hemoperfusion with the HA330/HA380 cartridge in intensive care settings. Blood Purif. 2025;54(2):122–137. doi: https://doi.org/10.1159/000542469.
13. Paul R, Ansari ASA, Ballesteros D, Klaus T, Honoré PM. Current knowledge on the impact of extracorporeal blood purification devices on drug clearance: systematic search and narrative review. Blood Purif. 2025;54(12):733–744. doi: https://doi.org/10.1159/000547477.
14. Wang C, Li C, Yang P, Liu K, Xiong X, Liu Y, et al. High adsorption capacity of hemoperfusion on imipenem in critically ill patients with septic shock: a case report. BMC Infect Dis. 2024;24:894. doi: https://doi.org/10.1186/s12879-024-09774-3.


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