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Case Series

CytoSorb hemoadsorption in diverse hyperinflammatory states with multi-organ dysfunction: a clinical case series

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ABSTRACT

Hyperinflammatory syndromes resulting in cytokine storm are a major contributor to morbidity and mortality in critically ill patients with infections and systemic inflammatory conditions. CytoSorb hemoadsorption therapy has been increasingly utilized as an adjunctive modality to mitigate excessive cytokine burden. This study aimed to evaluate the clinical utility of CytoSorb therapy across diverse hyperinflammatory conditions presenting with multi-organ dysfunction. This case series describes three patients with distinct etiologies-severe malaria with acute liver failure, dengue-associated hemophagocytic lymphohistiocytosis (HLH), and septic shock with cardiomyopathy-who developed multi-organ dysfunction and were treated with CytoSorb therapy alongside standard care. Clinical, biochemical, and hemodynamic parameters were monitored before, during, and after CytoSorb intervention. Inflammatory markers including C-reactive protein (CRP), procalcitonin, interleukin-6 (IL-6), and ferritin were evaluated. Across all cases, CytoSorb therapy was associated with marked clinical improvement, including stabilization of hemodynamics, reduction in inflammatory markers (e.g., CRP decreased from 245.4 to 5.8 mg/l), and recovery of organ function. Patient A required three CytoSorb sessions integrated with sustained low-efficiency dialysis (SLED) for severe malaria-associated multi-organ dysfunction. Patient B received two sessions during HLH-associated hyperinflammation. Patient C underwent one session during peak cytokine surge in septic shock with cardiomyopathy. All patients achieved 100% survival with de-escalation to ward care. CytoSorb hemoadsorption demonstrates potential therapeutic utility across heterogeneous cytokine-driven conditions when initiated early and combined with standard care. These findings support the integration of extracorporeal cytokine removal as an adjunctive strategy in critically ill patients with hyperinflammatory states.

Keywords: CytoSorb, Hemoadsorption, Cytokine storm, Multi-organ dysfunction, Sepsis, Severe malaria, Hemophagocytic lymphohistiocytosis, Extracorporeal blood purification, Critical care, Adjunctive therapy

INTRODUCTION

The pathophysiology of critical illness in conditions such as sepsis, severe infections, and hyperinflammatory syndromes is increasingly understood to involve dysregulated host immune responses, particularly the excessive release of pro-inflammatory cytokines, commonly termed a "cytokine storm".[1] This uncontrolled inflammatory cascade leads to endothelial dysfunction, capillary leak, coagulopathy, and ultimately multi-organ dysfunction syndrome (MODS).[2] While conventional management focuses on treating the underlying cause (e.g., antimicrobials, antivirals, supportive care), these strategies often fail to directly address the pathogenic cytokine burden. Consequently, adjunctive extracorporeal therapies aimed at immune modulation have gained attention.[3]

CytoSorb is a hemoadsorption device composed of biocompatible porous polymer beads capable of removing hydrophobic molecules in the 5-60 kDa range, including key inflammatory mediators such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1β (IL-1β).[4] By reducing circulating cytokine levels, CytoSorb aims to restore immune homeostasis, improve microcirculation, reduce vasopressor requirements, and limit progression of organ dysfunction.[5] Although most evidence supports its use in septic shock, its application has expanded to non-bacterial hyperinflammatory conditions, including HLH, severe malaria, and viral infections.[6]

Severe malaria, particularly Plasmodium falciparum infection, is characterized by a profound inflammatory response with elevated levels of TNF-α, IL-1β, IL-6, and IL-10, contributing to acute lung injury, acute kidney injury (AKI), and hepatic dysfunction.[7] Hemophagocytic lymphohistiocytosis represents a life-threatening hyperinflammatory syndrome driven by excessive immune activation, with markedly elevated ferritin, soluble IL-2 receptor, and cytokine levels.[8] In septic shock, the interplay between pathogen-associated molecular patterns and damage-associated molecular patterns triggers a systemic inflammatory response that frequently progresses to MODS and death.[9] Despite the expanding body of literature on cytokine-directed therapies, robust evidence from low- and middle-income countries remains limited, particularly regarding the application of CytoSorb in tropical infectious diseases and resource-limited settings.[10] This case series presents three patients with distinct etiologies but a common pathophysiological denominator-cytokine storm leading to organ failure-treated with CytoSorb therapy, providing insight into its broader clinical applicability in diverse hyperinflammatory conditions.

CASE SERIES

Patient overview

Three patients aged 25, 30 and 32 years presented with severe systemic illness characterized by fever, organ dysfunction, and laboratory evidence of hyperinflammation. Despite differing etiologies, all patients exhibited progressive clinical deterioration with features of cytokine-mediated injury, necessitating escalation to high-dependency or intensive care settings. Table 1 summarizes the comparative clinical characteristics of the three patients.

Table 1: Comparative clinical characteristics of patients treated with CytoSorb hemoadsorption.

Parameters	Patient A	Patient B	Patient C
Age in years/Sex	30/M	32/M	25/F
Etiology	Severe malaria	Dengue + HLH	Septic shock + cardiomyopathy
Key complications	Liver failure, AKI	HLH, hepatic dysfunction	MOF, cardiomyopathy
Respiratory involvement	CAP with effusion	Pleural effusion	Severe pneumonia+ventilation
Renal involvement	AKI (dialysis)	Mild	AKI (improved)
ICU/HDU care	Yes	Yes	Yes

*CAP: Community-acquired pneumonia; AKI: Acute kidney injury; HLH: Hemophagocytic lymphohistiocytosis; MOF: Multi-organ failure; ICU: Intensive care unit; HDU: High dependency unit.

Table 2: CytoSorb intervention profile and treatment characteristics.

Parameters	Patient A	Patient B	Patient C
CytoSorb sessions	3	2	1
Adjunct therapy	SLED (9 sessions)	HDU care	SLED + plasma exchange
Indication	Severe malaria + MODS	HLH cytokine storm	Septic shock + MOF
Timing	During organ failure	During deterioration	During peak cytokine surge
Outcome	Recovered	Recovered	Recovered

*SLED: Sustained low-efficiency dialysis; MODS: Multi-organ dysfunction syndrome; HLH: Hemophagocytic lymphohistiocytosis; MOF: Multi-organ failure; HDU: High dependency unit

Clinical spectrum at presentation

Patients demonstrated persistent high-grade fever, respiratory compromise (hypoxia, pleural effusion, consolidation), hepatic dysfunction (elevated transaminases, jaundice), renal impairment (AKI requiring dialysis in selected cases) and hematological abnormalities (anemia, thrombocytopenia). Inflammatory markers were markedly elevated, with CRP 245.4 mg/l, procalcitonin 17.9 ng/ml, IL-6 129 pg/ml, and ferritin 2169 ng/ml, strongly indicating a hyperinflammatory cytokine-driven state.

CytoSorb intervention profile

All patients received CytoSorb therapy as part of extracorporeal treatment, integrated with renal replacement therapy (RRT) or intensive care support. Patient A required three CytoSorb sessions due to severe malaria-associated cytokine burden, integrated with nine sessions of sustained low-efficiency dialysis (SLED). Patient B received two CytoSorb sessions targeting HLH-associated hyperinflammation during clinical deterioration. Patient C required early intervention with one CytoSorb session during septic shock with extremely elevated inflammatory markers, combined with SLED and plasma exchange. The differential number of CytoSorb sessions required across patients may reflect varying degrees of inflammatory burden, suggesting a potential dose–response relationship that warrants further investigation. Table 2 summarizes the CytoSorb intervention profiles.

Clinical and biochemical outcomes

All patients demonstrated consistent improvement following CytoSorb therapy. Hemodynamics stabilized in all patients. Inflammatory markers showed significant reduction, exemplified by CRP decreasing from 245.4 to 5.8 mg/l. Organ function recovered across renal, hepatic, and respiratory systems. Oxygen requirements were reduced and subsequently discontinued. ICU courses were successfully de-escalated to ward care. Notably, this case series achieved 100% survival. Table 3 summarizes clinical and biochemical outcomes following CytoSorb therapy.

Table 3: Clinical and biochemical outcomes following CytoSorb therapy.

Outcomes	Observations
Hemodynamics	Stabilized in all patients
Inflammatory markers	Significant reduction (e.g., CRP ↓ from 245.4 → 5.8 mg/L)
Organ function	Renal, hepatic, and respiratory recovery
Oxygen requirement	Reduced and discontinued
ICU course	De-escalation to ward care
Survival	100% survival in this series

*CRP: C-reactive protein; ICU: Intensive care unit

DISCUSSION

This case series illustrates the potential utility of CytoSorb hemoadsorption therapy across diverse hyperinflammatory conditions unified by a common pathophysiological mechanism-cytokine storm leading to multi-organ dysfunction syndrome (MODS). Despite differing etiologies, including severe malaria, dengue-associated hemophagocytic lymphohistiocytosis (HLH), and septic shock with cardiomyopathy, all patients exhibited features of dysregulated immune activation characterized by elevated inflammatory biomarkers, organ failure, and clinical deterioration. The observed clinical improvements following CytoSorb therapy-including hemodynamic stabilization, reduction in oxygen requirements, and recovery of renal and hepatic function-support the hypothesis that extracorporeal cytokine removal may play a crucial adjunctive role in managing such critically ill patients.

CytoSorb functions by adsorbing middle molecular weight inflammatory mediators such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and other cytokines implicated in the propagation of systemic inflammation.[11] In the present series, the marked reduction in inflammatory markers, particularly C-reactive protein (CRP), parallels findings from previous studies where cytokine adsorption was associated with improved clinical outcomes.[12] Importantly, this case series expands the scope of CytoSorb beyond conventional septic shock to include tropical infectious diseases and immune dysregulation syndromes such as HLH, where excessive immune activation plays a central role.[4,6]

The differential number of CytoSorb sessions required across patients may reflect varying degrees of inflammatory burden, suggesting a potential dose–response relationship that warrants further investigation. Additionally, the integration of CytoSorb with other extracorporeal modalities, such as sustained low-efficiency dialysis (SLED) and plasma exchange, may have synergistically enhanced toxin and cytokine clearance, contributing to improved outcomes.[6] Another key observation is the apparent benefit of early initiation during the peak inflammatory phase, which aligns with

emerging evidence that timely cytokine modulation may prevent irreversible organ damage.[10]

While randomized controlled trials have yielded mixed results regarding mortality benefit, accumulating real-world data suggest that selected patient populations with pronounced hyperinflammation may derive the greatest advantage.[11] However, the findings of this series must be interpreted cautiously due to inherent limitations, including small sample size, heterogeneity of disease conditions, and absence of a comparator group. Furthermore, the inability to isolate the independent effect of CytoSorb from concurrent therapies limits definitive causal inference. Nevertheless, the consistent pattern of clinical and biochemical improvement across all three patients provides supportive evidence for its role as an adjunctive therapy. Future large-scale, controlled studies are essential to establish standardized indications, optimal timing, and treatment protocols for CytoSorb therapy.

CONCLUSION

This case series highlights the broad therapeutic potential of CytoSorb hemoadsorption in managing cytokine storm across diverse etiologies. Despite differing underlying diseases, all patients exhibited rapid clinical stabilization, significant reduction in inflammatory markers, and recovery from multi-organ dysfunction. CytoSorb appears to be a promising adjunctive therapy in critically ill patients with hyperinflammatory states, particularly when initiated early and combined with standard care. Future randomized controlled trials are needed to establish definitive clinical guidelines.

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