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Review Article

A Comprehensive Review of Fluid Resuscitation Techniques in Sepsis in Patients with Heart Failure

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ABSTRACT

Introduction: Fluid management in sepsis patients with pre-existing heart failure presents a complex clinical challenge, as these patients require adequate resuscitation while avoiding fluid overload that could worsen cardiac function. This article aims to explore optimal fluid resuscitation strategies for patients with pre-existing heart failure who develop sepsis, a group at high risk for fluid management complications.

Methods: We conducted a narrative review of studies published between 2010 and 2024, utilizing PubMed, ScienceDirect, and Google Scholar. We employed Boolean formulas and search terms that evolved from broad to specific, refining the focus on fluid resuscitation in septic heart failure patients. Human studies focusing on fluid resuscitation, sepsis management, and outcomes in heart failure were included. Exclusion criteria included animal studies, non-English articles, and case reports.

Results: Guideline-recommended fluid resuscitation (≥30 mL/kg within 3 hours) shows a neutral or positive effect on mortality in sepsis patients with pre-existing heart failure when monitored appropriately. Patients with reduced ejection fraction (HFrEF) and those with preserved ejection fraction (HFpEF) exhibit different tolerances to fluids. Advanced hemodynamic monitoring — including bedside echocardiography, lactate clearance, central venous pressure, and BNP levels — is essential for personalizing fluid therapy.

Conclusion: Early guideline-compliant fluid resuscitation followed by a conservative, individualized fluid strategy guided by hemodynamic monitoring optimizes outcomes in sepsis patients with heart failure. Future prospective studies are needed to develop standardized protocols.

1. Introduction

Sepsis and septic shock are the significant causes of death globally, with a 25-30% mortality rate, particularly in hospitals [1]. This is especially true in the post-COVID era, underscoring the importance of timely intervention in critical care and highlighting the need for comprehensive research to refine therapeutic approaches and enhance patient outcomes.

If sepsis progresses to septic shock, a type of distributive shock, it will lead to vasodilation, resulting in circulatory, cellular, and metabolic abnormalities, which may lead to multiple organ failure, requiring vasopressor treatment to maintain adequate perfusion [1]. The population at risk for sepsis includes the elderly, intensive care unit patients, immunocompromised individuals, and neonates. When sepsis coincides with heart failure, fluid resuscitation plays a crucial role in preventing fluid overload and acute decompensated heart failure [2]. However, there is a paucity of literature specifically addressing this issue in patients with sepsis and heart failure. This narrative review aims to determine the safety, timing, rates, volumes, and types of fluids needed in patients with heart failure and sepsis, shedding light on an overlooked aspect of critical care and committed to improving outcomes for post-sepsis survivors. Though there are not any standardized definitions for fluid overload, it is majorly a clinical decision based on signs like respiratory distress with coarse crackles, peripheral edema, ascites, hepatomegaly, congestive heart failure, jugular venous distention or quantitative criteria like weight gain of >10%, a positive fluid balance of >5 L, or radiologic evidence of pulmonary edema.

2. Methods

We conducted a narrative review of studies published between 2010 and 2024 using PubMed, ScienceDirect, and Google Scholar, employing Boolean formulas and search terms that evolved from

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Table 1: Sepsis definition [1, 2]

Parameter	qSOFA Score	NEWS Score
Purpose	Rapid assessment for sepsis outside ICU	Early warning score for acute illness
Score Range	0 to 3 2 to 3 high risk 0 to 1 not high risk	0 to ≥7 0–4 low risk 5–6 medium risk ≥7 high risk
Respiratory Rate	≥ 22 breaths/min (1 point)	0: 12–20 breaths/min 1: 9–11 or 21–24 breaths/min 2: ≤ 8 or ≥ 25 breaths/min
Altered Mental Status	GCS < 15 (1 point)	New confusion
Systolic Blood Pressure	≤ 100 mmHg (1 point)	0: ≥ 111 mmHg 1: 101–110 mmHg 2: 91–100 mmHg 3: ≤ 90 mmHg
Heart Rate	–	0: 51–90 bpm 1: 41–50 or 91–110 bpm 2: 111–130 bpm 3: ≤ 40 or ≥ 131 bpm
Oxygen Saturation	–	0: ≥ 96% 1: 94–95% 2: 92–93% 3: ≤ 91%
Temperature	–	0: 36.1–38.0°C 1: 35.1–36.0 or 38.1–39.0°C 2: ≤ 35.0 or ≥ 39.1°C
Use of Supplemental O_2	–	2: Yes
Use in Clinical Setting	Non-ICU setting Screening tool	Hospital and emergency departments Prediction tool

ICU, Intensive Care Unit; qSOFA, Quick Sequential Organ Failure Assessment score; NEWS, National Early Warning Score

broad to specific to refine the focus on fluid resuscitation in septic heart failure patients. Human studies focusing on fluid resuscitation, sepsis management, and outcomes in heart failure were included. Exclusion criteria included animal studies, non-English articles, and case reports. Articles were analyzed with a focus on the research question: the safety of fluid administration in sepsis with heart failure. Relevant data were extracted and cited using Vancouver style via Zotero.

2.1. Background

The diagnosis of sepsis has evolved over time, the most recent one being the Sepsis-3 definition as measured by the Sequential Organ Failure Assessment score (SOFA) score and National Early Warning Score (NEWS), is presented in Table 1- which emphasizes the presence of organ dysfunction, particularly cardiovascular dysfunction, like decreased peripheral resistance and increased vascular permeability, leading to tissue hypoperfusion, oliguria, elevated lactate and creatinine levels, coagulopathies, and subsequent organ failure. While effective fluid resuscitation is a mainstay of sepsis management, aiming to restore hemodynamic stability and improve tissue perfusion, this also raises concern in heart failure patients [3].

Cardiovascular insufficiency in heart failure can arise from structural or functional heart disorders that impair the heart's ability to fill with or eject blood. The pathophysiology of HF is complex, involving hemodynamic, neurohormonal, and cellular mechanisms. Hemodynamic changes include a reduced cardiac output and elevated ventricular filling pressures due to volume overload and impaired relaxation. Neurohormonal activation significantly impacts HF, with the renin-angiotensin-aldosterone system (RAAS) causing vasoconstriction, sodium retention, and fluid overload [4]. The sympathetic nervous system (SNS) increases catecholamine levels, thereby raising heart rate and contractility, but also contributes to myocardial damage over time [5]. On a cellular level, myocytes undergo hypertrophy to handle the increased workload, fibrosis stiffens the myocardium due to excess collagen, and apoptosis, or the programmed cell death of myocytes, further impairs cardiac function [6].

Sepsis is a life-threatening condition resulting from a dysregulated immune response to infection, leading to widespread inflammation, tissue damage, and organ dysfunction. The immune response in sepsis involves both pro-inflammatory and anti-inflammatory processes. Pro-inflammatory responses include the release of cytokines such as TNF-α, IL-1, and IL-6 [7]. Anti-inflammatory responses involve the production of IL-10 and other mediators to counteract inflammation [7]. Endothelial dysfunction is a key feature of sepsis, with increased permeability leading to fluid leakage, edema, and hypotension [7], and microvascular thrombosis occurring due to coagulation activation and impaired fibrinolysis [7]. Metabolic changes in sepsis include mitochondrial dysfunction, which reduces ATP production and contributes to cellular energy failure [7], and hyperglycemia resulting from stress-induced insulin resistance and increased gluconeogenesis [7]. These processes contribute to multiple organ dysfunction syndrome (MODS), characterized by impaired perfusion and oxygen delivery to tissues [7].

Table 2: Mechanisms of Cardiac Dysfunction alongside Sepsis [5, 6, 7]

Mechanism	Description	Impact
Myocardial Depression	Cytokines (TNF-α, IL-1β, IL-6) and nitric oxide reduce myocardial contractility.	Decreased cardiac output, hypotension.
Autonomic Dysfunction	Altered autonomic regulation leads to impaired heart rate variability and reduced baroreflex sensitivity.	Tachycardia, arrhythmias.
Microvascular Dysfunction	Endothelial damage and microthrombi reduce coronary perfusion.	Ischemia, myocardial infarction.
Mitochondrial Dysfunction	Impaired oxidative phosphorylation leads to reduced ATP production.	Energy deficit, impaired contractility
Increased Afterload	Systemic vasodilation and hypotension initially, followed by increased vascular resistance.	Increased workload on the heart, heart failure
Electrolyte Imbalance	Sepsis-induced AKI and other factors cause electrolyte disturbances	Arrhythmias, impaired contractility

TNF, Tumor Necrosis Factor; IL, Interleukin; ATP, Adenosine Triphosphate; AKI, Acute Kidney Injury

2.2. Impact of Sepsis on Cardiac Function

Sepsis exerts profound effects on cardiac function, leading to septic cardiomyopathy, a reversible dysfunction of the heart. Hemodynamically, the combined effects of heart failure and sepsis lead to decreased preload and mean arterial pressure (MAP), increased heart rate, neutral effect on systemic vascular resistance, and varying effect on contractility [4]. The mechanisms of cardiac dysfunction in sepsis are summarized in Table 2: The pathophysiological processes in a patient with heart failure complicated by sepsis are multifaceted and interlinked. The systemic inflammatory response of sepsis exacerbates cardiac dysfunction, precipitates coagulopathy, and leads to multi-organ failure. Understanding these mechanisms is crucial for managing such critically ill patients and highlights the need for integrated therapeutic approaches to mitigate the impact of these concurrent conditions. The challenge lies in finding the delicate balance between providing sufficient fluid to support circulation without exacerbating heart failure; careful monitoring is essential, as fluid overload in the above condition has been associated with exacerbation of heart failure with vasodilation, pulmonary edema, peripheral edema, elevated jugular venous pressure, increased intra-abdominal pressure leading to liver and kidney injury-hyponatremia in critically ill patients, and increased mortality [8]. Heart failure can manifest as either a reduced ejection fraction or a preserved ejection fraction. Both types are further complicated by sepsis, because the inflammatory response can worsen cardiac function and lead to fluid overload.

However, evidence suggests that guideline-based fluid resuscitation ($\geq$30 mL/kg within 3 hours) is associated with lower in-hospital mortality compared to restrictive approaches in sepsis patients with HF [9, 10]. The use of careful management of chronic HF medications and consideration of β-blockers after hemodynamic stabilization with balanced crystalloids and albumin may be beneficial [10]. Additional research is needed to determine optimal fluid resuscitation strategies in this population, as clinicians must balance the need for volume expansion against the risk of fluid overload and worsening of cardiac dysfunction.

2.3. Safety profile and other outcomes

The Surviving Sepsis campaign recommends giving at least 30ml/kg fluid bolus, preferably with IV Crystalloids, in septic shock patients within 3 hours as a best practice measure to correct hypotension, but this recommendation is criticized as based on poor quality evidence, as it doesn't take other patient comorbidities into consideration [11]. Additionally, the Centers for Medicare and Medicaid Services also mandated this recommendation as part of its SEP-1 sepsis management bundle. Despite these guidelines, many physicians are often hesitant to administer fluid resuscitation to sepsis patients with heart failure due to concerns about potential fluid overload [12]. In line with this, several retrospective cohort studies have indicated that patients with heart failure often experience delays in fluid administration [13] those who are more likely to fail to comply with guidelines for fluid resuscitation [14, 15, 16] and generally receive less aggressive resuscitation compared to sepsis patients without heart failure [17, 18].

In a study of 552 patients, Wardi et al. in a retrospective cohort study found that individuals with sepsis and heart failure with reduced ejection fraction (HFrEF) received an average of 9.8 mL/kg less fluid compared to those without HFrEF (31.7 mL/kg versus 41.5 mL/kg, respectively; $p = 0.03$). Moreover, among the heart failure patients, those who did not survive received 21 mL/kg of fluid, whereas survivors with HFrEF received 35 mL/kg ($p = 0.16$). These findings suggest that administering a fluid bolus of at least 30 mL/kg could be beneficial for this subgroup of patients. However, the study's findings may be constrained by the relatively small number of heart failure patients included [19].

Among a retrospective cohort of 505 patients, Singh et al. found that sepsis patients with HFrEF who received more than 3 liters of fluid had a higher in-hospital mortality and longer hospital stays. While Singh et al.'s findings suggest an association between higher fluid volumes and increased mortality in septic patients with HFrEF, the retrospective design and limited adjustment for clinical variables, such as sepsis severity or vasopressor use, introduce potential confounding elements. The absence of a detailed methodology and a small sample size further limits the generalizability of these results. Therefore, these observations should be viewed as hypothesis-generating. Future prospective studies with proper control of confounders are needed to clarify the causal relationship between fluid resuscitation strategies and outcomes in this high-risk population [20].

Additionally, current guidelines may allow for the administration of more than 3 liters of fluid in patients with a body weight of 100 kg, which means that many individuals may not reach the threshold for excessive fluid according to these guidelines. But most importantly, the study did not report the period over which the fluid was administered, making it challenging to apply these findings in clinical practice [20]. Additionally, in a more comprehensive retrospective study, Tam et al. found that patients with sepsis who had a known history of heart failure (HF) received less fluid than those without HF at 6, 12, 24, and 48 hours post-injury. Despite this reduced fluid volume, patients with HF still received over 40 mL/kg of fluids within the first 6 hours of sepsis onset. Nevertheless, there were no significant differences between the HF and non-HF groups in terms of net fluid balance at 6 and 48 hours, hospital length of stay (LOS), ICU LOS, rates of persistent hypotension at 48 hours, intubation, CPAP/BIPAP use, cardiovascular complications, acute kidney injury (AKI), or mortality [21]. In this context, administering at least the recommended 30 mL/kg of fluid resuscitation within the first six hours of sepsis might be safe for patients with septic shock or severe sepsis and a known history of HF. A recent case-control study of 671 patients revealed that compliance with ≥30ml/kg fluid resuscitation was lower in sepsis patients, with even lower compliance among heart failure patients with sepsis. However, the study also found that the use of a fluid bolus (≥30 mL/kg) in heart failure patients presenting with severe sepsis or septic shock appeared to reduce the risk of in-hospital mortality (OR 0.95, 95% CI 0.90–0.99, p<0.05). Additionally, there was no increased risk of mechanical ventilation associated with the fluid bolus (OR 1.01, 95% CI 0.96–1.06, p = 0.70) [10].

In a retrospective chart review, Boccio et al. found that the only significant difference between sepsis fluid bolus-compliant and non-compliant patients with congestive heart failure (CHF) was a shorter stay in the intensive care unit (ICU) for the compliant group. Additionally, there were no statistically significant differences in mortality rates, the probability of intubation within 72 hours or during hospitalization, or inpatient length of stay among CHF patients who were compliant versus those who were noncompliant. However, the sample size may have been insufficient to detect a meaningful difference in mortality.[14]. In their retrospective chart review, Akhter et al. categorized sepsis patients with a history of CHF and/or ESRD into two groups based on their fluid intake: those who received at least 30 ml/kg and those who received a fluid-restrictive regimen of < 30 ml/kg. They found that the compliance group did not have a higher likelihood of intubation compared to the other group. There was also no significant difference in hospital length of stay or mortality. However, the study has limitations, including potential baseline differences- unadjusted confounders between the two patient groups, like sepsis severity or vasopressor use, and insufficient sample size(for calculating mortality), which may have influenced the results [22]. However, in a separate study with a larger sample size and similar patient grouping in a prospective observational design, Akhter et al. utilized a multivariate logistic regression model to account for confounding factors and showed that sepsis fluid bolus administration does not elevate the risk of mortality or intubation [23]. Similarly, in a retrospective cohort study, Duttuluri et al demonstrated that in heart failure patients with severe sepsis or septic shock presenting with hypotension, an adequate fluid bolus (≥30 mL/kg) decreased the risk of in-hospital mortality and intubation [24]. In a smaller prospective observational study by Ehrman et al., patients with reduced left ventricular ejection fraction (rLVEF) and those without rLVEF received similar volume fluid boluses. Interestingly, despite this similarity in fluid administration, both groups had comparable hospital days, ICU days, and ventilator days [25].

Khan et al., in a retrospective study, found that administering more than 30 mL/kg of fluid resuscitation was not independently linked to intubation (aOR 0.75, 95% CI 0.41-1.36, p = 0.34) after adjusting for confounders using multivariable generalized estimating equations. The number of ICU-free days at 28 days was similar between the restricted and standard fluid groups (17±10 days vs. 17±11 days, p=0.64). Additionally, there was no significant difference in the number of days on mechanical ventilation between the groups (11±16 days vs. 10±12 days, p=0.96), and hospital mortality rates were comparable (45 [21%] vs. 19 [18%], p=0.21), but an insufficient sample size may limit the study [26]. Ouellette and Shah, in their case-control study, used total intravenous fluid volume administered during the first 24 hours of patient admission as exposure and found no correlation between intravenous fluid volume and the PO2/FiO2 (oxygenation) at 24 hours in either cohort (r2 = 0.019 for cases, r2 = 0.001 for controls). In-hospital mortality rates (P = 0.117) and intubation rates at 24 hours (P = 0.687) were not significantly different between cases and controls either. However, the study may have been underpowered to detect differences in mortality [27].

In a retrospective cohort study Kuttab et al. found that, after accounting for various confounders, patients who did not receive the 30-by-3 fluid resuscitation protocol had higher odds of in-hospital mortality (OR=1.52, 95% CI 1.03–2.24), delayed onset hypotension (OR=1.42, 95% CI 1.02–1.99), and a longer ICU stay among those admitted to the ICU (mean increase of about 2 days, β=2.0, 95% CI 0.5–3.6). The study also showed that patients with severe sepsis and septic shock who were elderly, male, obese, with documented volume "overload" from bedside examination, and had a history of heart failure or end-stage renal disease were less likely to receive the 30-by-3 protocol [12]. An older retrospective study by Shah and Ouellette found that adherence to early goal-directed therapy improved in-hospital mortality in patients with a reduced LVEF and sepsis [28]. In a retrospective study using claims data from elderly patients with a history of congestive heart failure (<35% EF) who presented with severe sepsis or septic shock, adherence to the initial fluid resuscitation guidelines outlined in the 3-hour sepsis bundle was associated with better in-hospital and [29] one-year mortality outcomes [29]. These results are also validated by a massive multisite observational study of nearly 15,000 severe sepsis/septic shock patients across three independent, prospective cohorts. Strict adherence to a 3-hour sepsis bundle, emphasizing rapid intervention that is not reliant on physiological endpoints, demonstrated lower in-hospital mortality, even after adjusting for confounding factors. This compliance also yielded significant cost savings in cohorts 2 and 3, which included over 7,500 patients. Furthermore, these results were validated within the CHF subpopulation of the study [30].

Liu et al. assessed the effects of implementing a treatment bundle for sepsis patients with intermediate lactate levels across multiple centers. This retrospective study found that the bundle implementation significantly improved compliance and brought down hospital mortality rates. Crucially, this drop in mortality did not result in longer hospital stays or more ICU transfers. When patients were stratified based on pre-existing heart failure or kidney disease, those with these conditions experienced significant reductions in both hospital and 30-day mortality post-implementation. Compliance with antibiotic timing and lactate reassessment was similar in these patient groups before and after implementation, highlighting that the improved outcomes were largely due to increased fluid

administration for patients with heart failure and/or chronic kidney disease [31].

A recent large retrospective cohort study revealed that patients with septic shock and HFrEF were less likely to receive guideline-recommended intravenous fluids compared to those with septic shock but without HFrEF. Even after adjusting for confounding factors, HFrEF was linked to a reduced likelihood of receiving 30 mL/kg of intravenous fluid within the first 6 hours of sepsis onset (aOR-0.63; 95% confidence interval [CI], 0.47-0.85; P = 0.002). However, the adjusted risk of mortality did not significantly differ between patients with HFrEF (aOR-0.92; 95% CI, 0.69-1.24; P = 0.59) and those without it, and there was no interaction with the volume of intravenous fluid administered (aOR-1.00; 95% CI, 0.98-1.03; P = 0.72) [15].

In a retrospective study by Herndon et al., involving a cohort primarily consisting of patients with various types of heart failure and sepsis-induced hypoperfusion, no difference in mortality was found between those who received the recommended 30 mL/kg fluid bolus and those who did not. The study observed shorter hospital and ICU lengths of stay, as well as a higher incidence of new invasive or noninvasive mechanical ventilation. But multivariate analysis indicated that these outcomes were influenced by factors unrelated to the initial fluid bolus. This suggests that administering a 30 mL/kg fluid bolus did not significantly impact outcomes in patients with mixed types of heart failure and sepsis-induced hypoperfusion [32].

In contrast to the above studies, Wiczorek et al., in a retrospective study of sepsis patients with CHF/CKD, found a significant correlation between receiving more than 30 mL/kg of intravenous fluids at 3 and 6 hours from emergency department arrival and the need for BiPAP (p-value 0.006, p 0.02, respectively). Surprisingly, there was no statistically significant difference in in-hospital mortality between the sepsis patients with CHF/CKD and those without CHF/CKD, with regard to receiving more than 30 mL/kg of IVF at either 3 or 6 hours from ED (p-value 0.614, p-value 0.115, respectively) [33].

Multiple studies have found that higher fluid balance is associated with negative outcomes. A recent retrospective cohort study by Dong et al. sought to investigate the link between fluid management and in-hospital mortality in sepsis patients with heart failure. The study aimed to identify a superior indicator for fluid management among fluid balance (FB), fluid intake (FI), and the fluid intake ratio (FB/FI), termed the fluid accumulation index (FAI). The findings revealed that a high FAI (FB/FI ratio) correlated with an increase in in-hospital mortality in these patients [34]. To understand the link between fluid balance (FB) and in-hospital mortality, a retrospective study by Zhang et al. categorized sepsis patients with heart failure into two groups based on FB levels: high ($\geq$ 55.85 ml/kg) and low ($<$ 55.85 ml/kg). Their research uncovered that a high FB was an independent predictor of both in-hospital and 30-day mortality and was also linked to extended ICU and hospital stays. These conclusions remained solid and consistent even after adjusting for potential confounders, making them a dependable measure for evaluating the impact of FB on patient outcomes in clinical practice [35]. In a study of 633 patients, Dhondup et al. revealed that each 1-liter increase in daily negative fluid balance during the de-escalation phase significantly lowered mortality rates across several time points: ICU, hospital, 90-day, and 1-year [36].

Overall, research on the safety, complications, and outcomes of fluid resuscitation in heart failure patients with sepsis is still limited. Septic shock treatment is structured into four phases: Resuscitation, Optimization, Stabilization, and Evacuation. Based on existing literature, following SCC fluid resuscitation guidelines is advisable, as they are likely to produce favorable or neutral outcomes with minimal adverse effects. Nonetheless, the risk of volume overload persists, as highlighted by recent studies [36, 34, 35]. Thus, fluid management should be optimized in heart failure patients with septic shock, and it is advisable to adopt a conservative approach during the other phases of septic shock treatment. New prospective studies are essential to conclusively determine the safety and effectiveness of fluid management in this subgroup.

3. Discussion

Managing fluid resuscitation in patients with sepsis and underlying heart failure remains one of the more delicate challenges in acute care. Although the Surviving Sepsis Campaign recommends administering 30 mL/kg of intravenous fluids within the first three hours—a protocol now widely adopted into quality metrics—this directive often raises concern among clinicians, especially when heart failure complicates the clinical picture. Clinicians are often caught balancing the urgency of reversing hypoperfusion against the risk of fluid overload and its potential consequences. Our review draws on a growing body of evidence indicating that a standardized initial fluid bolus, even in patients with known heart failure, may not only be safe but also potentially beneficial when administered with careful oversight. Several studies suggest that failure to comply with early fluid resuscitation guidelines in this population is common and often driven by fear of worsening cardiac function. Yet, data increasingly indicate that withholding or delaying fluids may be more detrimental, and that compliance with the recommended 30 mL/kg bolus does not uniformly increase adverse outcomes like intubation or mortality. What this review brings to the table is a cohesive, evidence-informed perspective that acknowledges the complexity without defaulting to blanket caution. We argue that early adherence to fluid resuscitation guidelines—guided by dynamic monitoring and clinical judgment—should remain a priority in the initial phase of sepsis management for patients with heart failure. Importantly, we advocate for a shift from one-size-fits-all protocols to phase-specific strategies: aggressive resuscitation when perfusion is threatened, followed by deliberate, conservative fluid strategies during stabilization and recovery. Balanced crystalloids may reduce acid-base disturbances compared to saline, though data in HF patients are limited.

Monitoring of fluid overload is needed for assessing fluid overload during the administration of fluids, and it requires a multimodal approach of clinical examination along with hemodynamic and imaging tools. Central venous pressure (CVP), while historically used, offers limited predictive value when used alone and must be interpreted using other clinical data. We recommend using point-of-care ultrasound, with lung ultrasound identifying B-lines consistent with pulmonary edema and inferior vena cava (IVC) assessment suggesting volume status based on diameter and collapsibility. Focused cardiac ultrasound for the evaluation of ventricular function and preload. Ultimately, combining these with laboratory markers, such as elevated BNP, and bedside clinical findings provides a more comprehensive and accurate evaluation of fluid overload.

By weaving together fragmented data from diverse clinical settings, we provide a clearer roadmap for frontline providers —one that

respects the nuances of heart failure but doesn't paralyze action. We also highlight gaps in the literature, including inconsistent definitions of fluid overload, limited reporting of fluid timing and monitoring, and a general lack of prospective trials tailored to this vulnerable subgroup. This is what future research must focus on. In short, our review reinforces that timely, well-monitored fluid resuscitation is not only possible in patients with sepsis and heart failure—it may be key to improving outcomes when approached with the nuance and precision these patients deserve.

This review is limited by heterogeneity in study designs, patient populations, and the lack of standardized definitions for fluid overload. Some studies had insufficient statistical power, which limited the conclusions drawn from non-significant outcomes. Additionally, the variability of heart failure phenotypes and the impact of different fluid types were not addressed due to limited data.

4. Conclusions

Fluid resuscitation in sepsis patients with pre-existing heart failure remains a complex clinical challenge, marked by a tension between the urgency of restoring perfusion and the risk of fluid overload. While traditional caution has often led to under-resuscitation in this population, emerging evidence reviewed in this manuscript suggests that early administration of 30 mL/kg of intravenous fluids, when coupled with careful monitoring, may be both safe and beneficial. Our review consolidates the available literature to emphasize that guideline-directed fluid resuscitation does not uniformly result in adverse outcomes among heart failure patients, and in some cases, may even improve mortality and reduce the duration of ICU stays.

By framing fluid management through a phase-specific lens, we advocate for a more dynamic, patient-tailored approach: one that prioritizes timely intervention during the resuscitation phase, followed by thoughtful de-escalation as the clinical picture evolves. This perspective challenges the conventional hesitancy surrounding fluid therapy in heart failure and calls for a shift toward evidence-guided flexibility rather than rigid restraint. Ultimately, our review not only synthesizes key findings across existing studies but also highlights critical areas for future research, particularly the need for prospective trials that can refine volume thresholds, optimal timing, and monitoring strategies, as well as differentiate between different types of heart failure in this high-risk subgroup. With a more nuanced and structured approach to fluid resuscitation, we can move toward safer and more effective sepsis care for patients with underlying heart failure.

Conflicts of Interest

The authors declare no conflicts of interest related to this manuscript.

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Institutional Review Board (IRB)

This review analyzed only previously published literature. No human-subjects research was conducted and no institutional review board approval was required.

Large Language Model

We have employed an advanced large language model to enhance and refine English language writing. This process focused solely on improving the text's clarity and style, without generating or adding any new information to the content.

Authors Contribution

MSO and ST contributed to the conceptualization of the work. Literature search, review of articles, and analysis of fluid resuscitation outcomes were carried out by MSO, ST, AVM, NM, N, and STi. MSO and ST were responsible for the write-up and synthesis of inferences and recommendations, as well as editing the manuscript for journal submission. AVM contributed to the introduction and background writing and provided key inputs during editing. NM was involved in writing the abstract and pathophysiology section and editing. N contributed to conclusion writing and inference synthesis. STi assisted in literature search and proofreading for reference consistency. Project supervision, guidance, and final review were completed by MSO and ST.

Data Availability

This review article does not contain any new primary data. All information discussed is derived from previously published sources and publicly available databases, as cited in the manuscript.

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