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Sepsis-induced cardiac dysfunction: Pathophysiology, diagnostic challenges, and clinical outcomes: A narrative mini review

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Abstract

Sepsis-induced cardiac dysfunction (SICD), also called septic cardiomyopathy, is a common complication seen in patients with sepsis and septic shock. It means the heart becomes weak during severe infection and cannot pump blood effectively. This condition can happen even in people who did not have previous heart disease. The exact cause of SICD is complex and involves many factors. These include strong inflammation in the body, release of harmful cytokines, reduced energy production in heart cells due to mitochondrial damage, and problems in calcium handling inside the heart muscle. Changes in blood flow at the small vessel level also contribute to heart dysfunction.

Diagnosing SICD is challenging because its signs overlap with other effects of sepsis. Doctors mainly use echocardiography to assess heart function and blood tests such as troponin and B-type natriuretic peptide (BNP) to support diagnosis. However, no single test can confirm the condition. Although SICD is often reversible, it is linked with worse outcomes, including low blood pressure requiring strong medications, longer ICU stays, and higher risk of death.

This mini review summarizes current knowledge about the causes, diagnosis, and outcomes of sepsis-induced cardiac dysfunction. It also highlights the need for better diagnostic tools and early recognition to improve patient care.

Keywords: Sepsis-induced cardiac dysfunction; Septic cardiomyopathy; Sepsis; Echocardiography; Cardiac biomarkers

1. Introduction

Sepsis is a life-threatening syndrome caused by a dysregulated host response to infection, leading to acute organ dysfunction and high mortality worldwide. Despite advances in intensive care, sepsis remains one of the leading causes of death among critically ill patients. Cardiac dysfunction is a common complication of sepsis and contributes significantly to disease severity and poor clinical outcomes. Among the different forms of cardiovascular involvement, right ventricular dysfunction has gained increasing attention because it is associated with significantly higher short-term and long-term mortality in patients with sepsis [1].

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In addition to right ventricular dysfunction, impairment of left ventricular systolic function is frequently observed in patients with severe sepsis and septic shock. Advanced echocardiographic techniques, particularly strain echocardiography, have improved the assessment of myocardial function and have demonstrated that reduced left ventricular systolic function is associated with an increased risk of mortality [2].

Management of sepsis-induced cardiac dysfunction remains mainly supportive, although several pharmacological therapies have been investigated. A recent randomized controlled trial evaluated adjunctive milrinone in patients with septic shock and demonstrated its effects on hemodynamic parameters, highlighting the continued interest in therapies aimed at improving cardiac performance in septic patients [3].

Cardiac dysfunction in sepsis is not limited to systolic impairment. Diastolic dysfunction is also common and has been associated with poor prognosis. Tissue Doppler echocardiography has emerged as a useful tool for assessing diastolic function and identifying septic patients at increased risk of mortality [4].

The mechanisms responsible for sepsis-induced cardiac dysfunction are complex and involve multiple interacting pathways. Inflammatory cytokines, nitric oxide overproduction, mitochondrial dysfunction, oxidative stress, abnormalities in calcium handling, and microcirculatory disturbances all contribute to myocardial depression. A comprehensive understanding of these mechanisms is essential for improving diagnosis and developing targeted therapies [5].

Recent evidence also suggests that sepsis-induced cardiac dysfunction is a heterogeneous condition rather than a single disease entity. Patients may present with isolated left ventricular dysfunction, right ventricular dysfunction, diastolic dysfunction, or combinations of these abnormalities, each with different clinical and prognostic implications [6].

Diagnosis of sepsis-induced cardiac dysfunction remains challenging because no universally accepted diagnostic criteria exist. Echocardiography remains the cornerstone of evaluation, while cardiac biomarkers such as troponin and B-type natriuretic peptide (BNP) provide additional prognostic information. Combining imaging findings with clinical assessment may improve early recognition and risk stratification [7].

Although sepsis-induced cardiac dysfunction is often reversible in survivors, it is associated with increased vasopressor requirements, prolonged intensive care unit stay, and higher mortality. Greater awareness of this condition and early identification are therefore important for optimizing patient management and improving outcomes [8].

This mini review aims to summarize the current evidence on the pathophysiology, diagnosis, and clinical outcomes of sepsis-induced cardiac dysfunction, with a focus on recent advances and remaining knowledge gaps.

2. Methods

This mini review was conducted to summarize the current evidence on sepsis-induced cardiac dysfunction (SICD), focusing on its pathophysiology, diagnostic approaches, and clinical outcomes.

A literature search was performed in PubMed and Google Scholar to identify relevant English-language studies published through June 2026. The search terms included combinations of: “sepsis,” “septic shock,” “sepsis-induced cardiac dysfunction,” “septic cardiomyopathy,” “myocardial dysfunction,” “echocardiography,” “strain imaging,” “tissue Doppler,” “right ventricular dysfunction,” “left ventricular dysfunction,” “troponin,” and “BNP.”

Studies were selected based on their relevance to SICD and its cardiovascular manifestations. Eligible articles included systematic reviews, meta-analyses, randomized controlled trials, and comprehensive review articles that focused on cardiac dysfunction in adult septic patients. Studies involving pediatric populations, non-cardiac outcomes, animal-only experiments, conference abstracts without full text, and non-English publications were excluded. As this was a narrative mini review, formal systematic review methodology, quality assessment, and risk-of-bias analysis were not performed.

Table 1 Summary of included studies on sepsis-induced cardiac dysfunction

No	Study	Study Type	Population	Key Finding
1	Vallabhajosyula et al., 2021	Meta-analysis	1,373 septic patients	Right ventricular dysfunction is strongly associated with increased short-term and long-term mortality in sepsis
2	Sanfilippo et al., 2018	Systematic review & meta-analysis	Severe sepsis/septic shock patients	Reduced LV systolic function assessed by strain imaging is associated with higher mortality.
3	Tongyoo et al., 2025	Randomized controlled trial	Septic shock patients	Milrinone alters hemodynamics but evidence for outcome benefit remains limited.
4	Sanfilippo et al., 2017	Systematic review & meta-analysis	Critically ill septic patients	Diastolic dysfunction assessed by tissue Doppler is associated with increased mortality.
5	L'Heureux et al., 2020	Comprehensive Review	Septic patients	SICD is caused by inflammatory injury, mitochondrial dysfunction, and metabolic failure.
6	Shvilkina & Shapiro, 2023	Review Article	Septic myocardial dysfunction	SICD is a heterogeneous syndrome with variable ventricular involvement and clinical effects.
7	Ehrman et al., 2018	Review Article	ICU septic patients	Echocardiography and biomarkers are key tools for evaluation and prognosis
8	Sato & Nasu, 2015	Review Article	Septic cardiomyopathy patients	SICD is often reversible and clinically defined as transient myocardial dysfunction.

A total of eight key articles were included in this mini review, which are summarized in Table 1. These studies were selected due to their high methodological quality and direct relevance to cardiac dysfunction in sepsis. The final included literature consisted of meta-analyses on right ventricular and left ventricular dysfunction, studies evaluating diastolic function, a randomized controlled trial assessing hemodynamic therapy, and comprehensive reviews describing the mechanisms and clinical implications of SICD.

The findings from the selected studies were synthesized qualitatively and organized into three major domains: (1) pathophysiology, (2) diagnostic evaluation, and (3) clinical outcomes of sepsis-induced cardiac dysfunction.

3. Results

3.1. Pathophysiology of Sepsis-Induced Cardiac Dysfunction

Sepsis-induced cardiac dysfunction (SICD) is a complex and multifactorial process that results in temporary impairment of myocardial function during sepsis and septic shock. It does not arise from a single pathway but from the interaction of inflammatory, metabolic, cellular, and microcirculatory abnormalities that together lead to reduced cardiac performance. Importantly, these changes are often reversible in survivors, suggesting functional rather than permanent structural myocardial injury [1]. It is summarized in Figure 1.

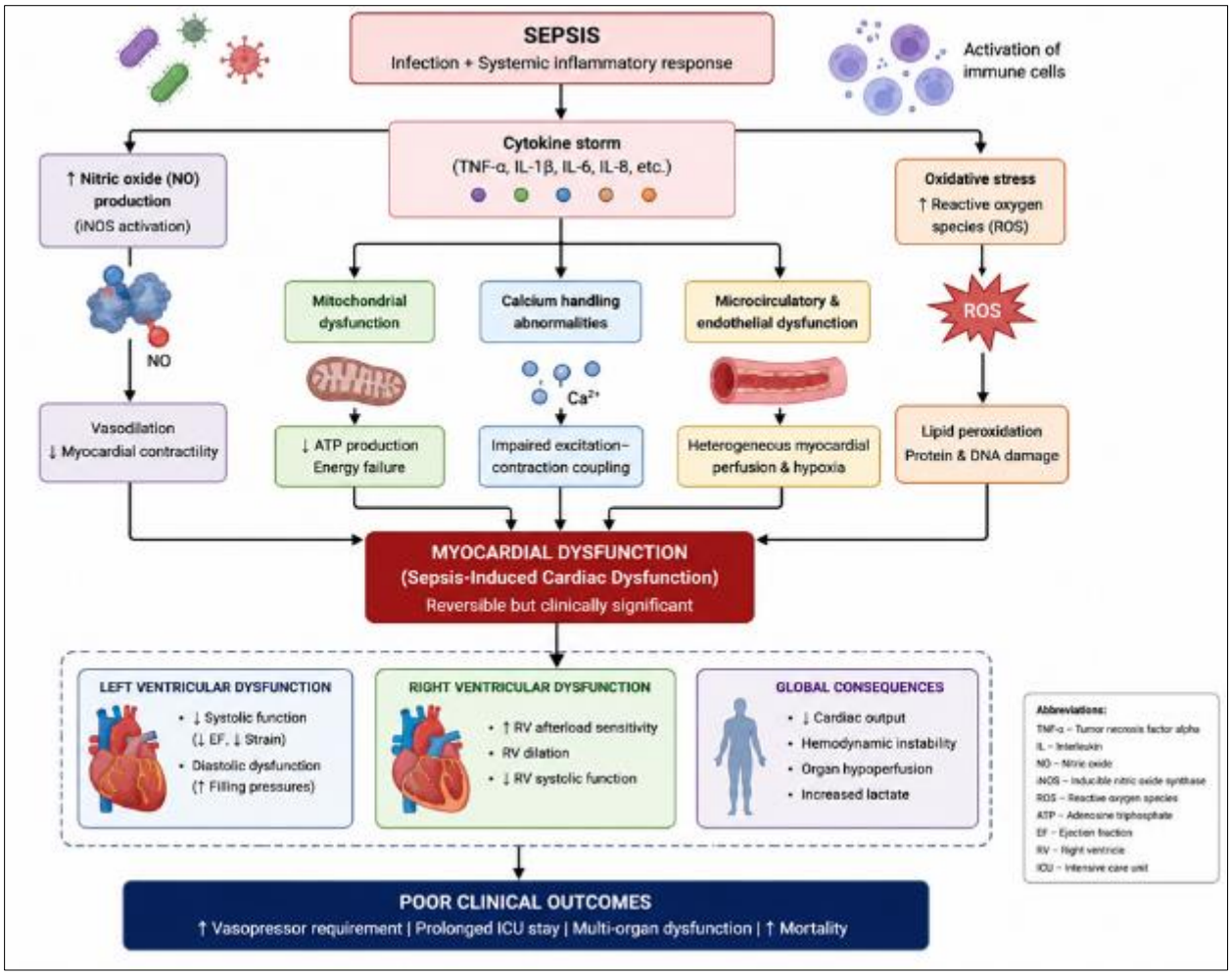

Figure 1 Pathophysiology of Sepsis-Induced Cardiac Dysfunction (SICD)

Sepsis triggers a systemic inflammatory response leading to release of cytokines (TNF-α, IL-1β, IL-6), which initiate myocardial depression. This is followed by nitric oxide overproduction causing vasodilation and reduced myocardial contractility. Concurrent mitochondrial dysfunction leads to impaired ATP production and cellular energy failure. Oxidative stress further damages cardiomyocytes and worsens contractile function.

Calcium handling abnormalities disrupt excitation–contraction coupling, resulting in impaired systolic and diastolic performance. Microcirculatory dysfunction leads to heterogeneous myocardial perfusion despite normal or elevated global cardiac output. These combined mechanisms result in reversible myocardial dysfunction affecting both left and right ventricles, clinically manifesting as sepsis-induced cardiac dysfunction.

This figure is created by Ruchik Kevadiya.

3.1.1. Inflammatory cytokine-mediated myocardial depression

A key mechanism in SICD is the excessive release of inflammatory cytokines during sepsis. Tumor necrosis factor-alpha (TNF-α), interleukin-1β, and interleukin-6 directly impair myocardial contractility and alter β-adrenergic signaling. These cytokines reduce calcium responsiveness in cardiomyocytes and contribute to global myocardial depression rather than focal ischemic injury [7].

3.1.2. Nitric oxide overproduction and oxidative stress

Sepsis induces activation of inducible nitric oxide synthase, leading to excessive nitric oxide production. This results in systemic vasodilation and contributes to reduced myocardial contractility. Nitric oxide and related reactive oxygen species also impair mitochondrial enzymes and worsen oxidative stress, further suppressing cardiac function [7].

3.1.3. Mitochondrial dysfunction and energy failure

Mitochondrial injury plays a central role in SICD. Impaired oxidative phosphorylation leads to reduced adenosine triphosphate (ATP) production, resulting in an "energy-starved myocardium." This metabolic failure contributes to reversible myocardial depression and impaired contractile reserve [5].

3.1.4. Calcium handling abnormalities

Normal cardiac contraction depends on tightly regulated calcium cycling within cardiomyocytes. In sepsis, disruption of sarcoplasmic reticulum function and altered calcium channels impair excitation–contraction coupling. This leads to reduced systolic contraction and impaired diastolic relaxation [5].

3.1.5. Microcirculatory and endothelial dysfunction

Sepsis causes widespread endothelial injury and microvascular dysfunction. Despite preserved or even increased global cardiac output in some patients, regional myocardial perfusion becomes heterogeneous. This imbalance leads to impaired oxygen delivery at the cellular level and contributes to myocardial dysfunction [5].

3.1.6. Ventricular functional abnormalities

SICD manifests as a spectrum of cardiac abnormalities involving both ventricles. Left ventricular systolic dysfunction is commonly detected using strain echocardiography and is associated with increased mortality in septic patients [2]. Diastolic dysfunction, assessed using tissue Doppler imaging, is also common and independently associated with worse outcomes [4].

In addition, right ventricular dysfunction has been identified as a strong predictor of both short-term and long-term mortality in sepsis, highlighting the importance of biventricular assessment [1].

3.1.7. Heterogeneity of myocardial dysfunction

Recent evidence suggests that SICD is not a single uniform entity but rather a heterogeneous condition with variable phenotypes. Patients may present with isolated left ventricular dysfunction, isolated right ventricular dysfunction, diastolic impairment, or mixed patterns. This heterogeneity contributes to variability in clinical presentation and prognosis and reflects the complex nature of septic myocardial injury [6].

3.1.8. Integrated mechanism of dysfunction

Overall, SICD results from the combined effects of inflammatory injury, mitochondrial energy failure, impaired calcium cycling, nitric oxide–mediated depression, and microcirculatory dysfunction. These processes interact to produce a reversible but clinically significant reduction in myocardial performance during sepsis [5].

3.2. Diagnosis of Sepsis-Induced Cardiac Dysfunction

Diagnosing sepsis-induced cardiac dysfunction (SICD) remains challenging because there is no universally accepted definition or single diagnostic criterion. In clinical practice, diagnosis relies on a combination of hemodynamic assessment, echocardiographic evaluation, and cardiac biomarkers, supported by the overall clinical context of sepsis [7].

3.2.1. Echocardiography as the cornerstone

Transthoracic echocardiography is the primary diagnostic tool for assessing myocardial dysfunction in sepsis. It allows real-time evaluation of both systolic and diastolic function, as well as right ventricular performance. Conventional parameters such as left ventricular ejection fraction (LVEF) are widely used; however, LVEF may be misleading in sepsis due to changes in preload and afterload [7].

3.2.2. Strain echocardiography and advanced imaging

Speckle-tracking strain imaging has emerged as a more sensitive method for detecting subtle myocardial dysfunction in sepsis. Left ventricular global longitudinal strain can identify systolic impairment even when ejection fraction appears normal. Reduced strain values have been associated with higher mortality in patients with severe sepsis and septic shock, highlighting its prognostic value [2].

Right ventricular function assessment is also increasingly recognized as essential, as right ventricular dysfunction is strongly associated with both short-term and long-term mortality in septic patients. This emphasizes the importance of biventricular assessment rather than focusing only on the left ventricle [1].

3.2.3. Diastolic dysfunction assessment

Diastolic dysfunction is commonly observed in septic patients and can be evaluated using tissue Doppler imaging. Parameters such as E/e' ratio help estimate left ventricular filling pressures and diastolic performance. Diastolic dysfunction has been independently associated with increased mortality in critically ill patients with sepsis, making it an important component of comprehensive cardiac evaluation [4].

3.2.4. Cardiac biomarkers

Cardiac biomarkers such as troponin and B-type natriuretic peptide (BNP) are frequently elevated in sepsis and reflect myocardial stress or injury. However, their interpretation is complex because elevations may occur due to non-ischemic mechanisms such as inflammation, ventricular strain, and microcirculatory dysfunction. Therefore, biomarkers are best used as adjuncts rather than standalone diagnostic tools [7].

3.2.5. Hemodynamic and functional assessment

In addition to imaging and biomarkers, continuous hemodynamic monitoring plays a key role in identifying SICD. Parameters such as cardiac output, systemic vascular resistance, and response to fluid resuscitation or vasopressors help characterize the functional impact of myocardial depression in septic shock. Advanced monitoring techniques may further assist in differentiating distributive shock from cardiogenic components [5].

3.2.6. Emerging role of artificial intelligence

Artificial intelligence (AI) is increasingly being explored in cardiovascular imaging and critical care. Machine learning algorithms can assist in automated interpretation of echocardiographic images, detection of subtle ventricular dysfunction, and integration of multi-parameter clinical data. AI-enabled models have the potential to improve early recognition of SICD by combining imaging, laboratory, and clinical variables into predictive risk scores, although most applications are still in early development stages [6].

3.2.7. Diagnostic challenges and limitations

Despite advances in imaging and biomarkers, diagnosis of SICD remains difficult due to overlapping features with other forms of sepsis-related circulatory failure. Changes in preload, afterload, and vasoactive therapy can mask or mimic myocardial dysfunction. The absence of standardized diagnostic criteria further contributes to variability in diagnosis and reported incidence [7].

3.2.8. Integrated diagnostic approach

A comprehensive diagnostic strategy combining echocardiography (including strain imaging), diastolic assessment, biomarkers, and clinical hemodynamic evaluation provides the most accurate assessment of SICD. This multimodal approach improves early detection and helps identify high-risk patients who may benefit from closer monitoring and targeted therapeutic strategies [2,4,7].

3.3. Clinical Outcomes of Sepsis-Induced Cardiac Dysfunction

Sepsis-induced cardiac dysfunction (SICD) is strongly associated with adverse clinical outcomes in critically ill patients. Its presence reflects more severe disease physiology and is linked to increased morbidity and mortality in both the short-term and long-term course of sepsis.

3.3.1. Mortality outcomes

Cardiac dysfunction in sepsis has been consistently associated with higher mortality. Among the different phenotypes, right ventricular dysfunction has emerged as a particularly strong predictor of poor prognosis. A meta-analysis of 1,373 patients demonstrated that right ventricular dysfunction significantly increases both short-term and long-term mortality in sepsis, highlighting its independent prognostic value [1].

Left ventricular systolic dysfunction assessed by strain imaging has also been associated with increased mortality in patients with severe sepsis and septic shock, suggesting that even subclinical myocardial impairment carries important

prognostic implications. [2] Similarly, diastolic dysfunction identified by tissue Doppler imaging is linked with higher mortality in critically ill septic patients [4].

3.3.2. Hemodynamic instability and vasopressor requirement

Patients with SICD often exhibit greater hemodynamic instability compared to those without myocardial dysfunction. Reduced myocardial contractility contributes to impaired cardiac reserve, leading to hypotension and increased dependence on vasopressor therapy to maintain organ perfusion. This reflects the combined impact of both systolic and diastolic impairment on global cardiovascular function [5].

3.3.3. ICU length of stay and disease severity

SICD is associated with more severe disease progression and prolonged intensive care unit (ICU) stay. Patients frequently require longer durations of mechanical ventilation, advanced hemodynamic monitoring, and multi-organ support. The presence of myocardial dysfunction often indicates a more severe systemic inflammatory response and higher overall illness burden [5].

3.3.4. Reversibility of myocardial dysfunction

A characteristic feature of SICD is its potential reversibility in survivors. Unlike ischemic cardiomyopathy, septic myocardial dysfunction is often transient and may resolve as the underlying sepsis is controlled. However, the recovery period varies, and persistent subclinical dysfunction may remain in some patients, contributing to long-term cardiovascular risk [8].

3.3.5. Ventricular-specific prognostic implications

Both left and right ventricular dysfunction contribute to adverse outcomes, but right ventricular dysfunction appears to carry a particularly strong prognostic burden. This may be due to its sensitivity to changes in pulmonary vascular resistance and its critical role in maintaining cardiac output during septic shock [1].

Left ventricular dysfunction, although often reversible, remains clinically significant because it reduces cardiac output and impairs tissue perfusion during the acute phase of sepsis [2].

3.3.6. Overall prognostic significance

Overall, SICD represents a marker of severe systemic illness and is associated with worse clinical trajectories. The presence of myocardial dysfunction identifies a high-risk subgroup of septic patients who require closer monitoring, aggressive hemodynamic management, and early echocardiographic evaluation [6].

4. Discussion

Sepsis-induced cardiac dysfunction (SICD) is increasingly recognized as a major contributor to poor outcomes in critically ill patients with sepsis. Rather than being a uniform disease, SICD represents a heterogeneous spectrum of myocardial involvement affecting both ventricles and varying across systolic, diastolic, and right ventricular function. This variability explains why diagnosis and reporting remain inconsistent across studies.

Current evidence suggests that right ventricular dysfunction may have the strongest association with mortality among all cardiac phenotypes in sepsis. This highlights the importance of comprehensive biventricular assessment rather than focusing solely on left ventricular ejection fraction [1]. In parallel, advanced echocardiographic techniques such as strain imaging have revealed that subclinical left ventricular dysfunction is common and clinically relevant, even when conventional ejection fraction appears preserved [2]. These findings suggest that traditional diagnostic approaches may underestimate the true burden of SICD.

Diastolic dysfunction is also frequently observed and independently associated with poor outcomes, further supporting the concept that SICD involves global myocardial impairment rather than isolated systolic failure [4]. Together, these findings emphasize the need for integrated cardiac assessment in septic patients.

Despite advances in imaging, diagnosis remains challenging due to the dynamic nature of sepsis-related hemodynamic changes. Fluid resuscitation, vasopressor therapy, and mechanical ventilation can significantly alter cardiac performance, making interpretation of echocardiographic findings difficult [7]. In addition, the absence of standardized diagnostic criteria contributes to variability in reported incidence and limits comparison across studies.

Therapeutic strategies remain largely supportive. Although interest in inotropic agents such as milrinone exists, evidence for mortality benefit remains limited and inconsistent [3]. This reflects the ongoing uncertainty regarding optimal management strategies for SICD and highlights the need for well-designed randomized controlled trials.

Mechanistically, SICD is driven by complex interactions involving inflammation, mitochondrial dysfunction, oxidative stress, and altered calcium handling [5]. These processes result in a reversible form of myocardial depression, distinguishing SICD from classic ischemic cardiomyopathy. However, the reversibility does not eliminate its clinical significance, as acute dysfunction is strongly associated with worse outcomes and increased resource utilization.

Recent literature also emphasizes the heterogeneity of SICD, suggesting that it should be considered a syndrome rather than a single disease entity [6]. This concept may explain why therapeutic trials have shown inconsistent results and underscores the need for phenotyping patients based on ventricular involvement and severity of dysfunction.

Overall, SICD remains an under-recognized but clinically important condition. Improved understanding of its pathophysiology and incorporation of advanced imaging techniques are essential for earlier detection, better risk stratification, and future development of targeted therapies.

Limitations

This narrative mini review did not include a formal quality assessment or risk-of-bias analysis of the included studies. In addition, only English-language articles identified through PubMed and Google Scholar were considered, which may have excluded relevant studies from other sources.

5. Conclusion

Sepsis-induced cardiac dysfunction (SICD) is a common and clinically important complication of sepsis that significantly contributes to poor outcomes in critically ill patients. It is a heterogeneous and dynamic condition involving systolic, diastolic, and right ventricular dysfunction, often occurring together and reflecting the severity of systemic illness.

Current evidence shows that right ventricular dysfunction, left ventricular systolic impairment, and diastolic dysfunction are all independently associated with increased mortality in sepsis. Advanced imaging techniques such as strain echocardiography and tissue Doppler imaging have improved the detection of both overt and subclinical myocardial dysfunction, although diagnostic challenges still remain due to the absence of standardized criteria.

The pathophysiology of SICD is multifactorial, involving inflammatory injury, mitochondrial dysfunction, oxidative stress, and abnormalities in calcium handling, resulting in a reversible but clinically significant form of myocardial depression. Despite this reversibility, SICD is strongly associated with increased vasopressor requirements, prolonged ICU stay, and higher mortality.

Future research should focus on establishing standardized diagnostic criteria, validating multimodal diagnostic strategies, and developing targeted therapeutic approaches tailored to distinct SICD phenotypes. Advances in these areas may facilitate earlier recognition, improve risk stratification, and ultimately enhance outcomes for patients with sepsis-induced cardiac dysfunction.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare that they have no competing financial or non-financial interests related to this work.

Statement of ethical approval

Ethical approval was not required for this study because it is a narrative review based exclusively on previously published literature.

Statement of informed consent

No human participants, animals, or identifiable patient data were involved.

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