

The synergistic value of mechanical circulatory support and fluid resuscitation in the treatment of sepsis-induced cardiomyopathy

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Sepsis, the leading cause of death among critically ill patients worldwide, can induce sepsis-induced cardiomyopathy (SICM), which is characterized by reversible acute impairment of biventricular systolic and diastolic functions. SICM has a high incidence in sepsis patients and significantly increases mortality, and it has emerged as one of the key factors contributing to death in septic shock.^[1] The pathological manifestations of SICM are highly heterogeneous, encompassing various forms such as left ventricular systolic dysfunction and diastolic dysfunction, which pose significant challenges to early identification and treatment. Fluid resuscitation serves as the cornerstone of sepsis treatment, yet there remains ongoing controversy regarding its optimal volume and timing.^[2] Meanwhile, mechanical circulatory support technologies such as venoarterial extracorporeal membrane oxygenation (VA-ECMO) have been successfully applied in refractory septic shock.^[3] These 2 approaches exert a synergistic effect based on complementary hemodynamic regulation mechanisms. Fluid resuscitation optimizes preload to improve cardiac filling while mechanical support directly replaces cardiac pump function to reduce afterload. This bidirectional regulation is particularly crucial in patients with mixed shock (septic combined with cardiogenic) as it helps resolve the contradiction between fluid resuscitation and diuretic therapy.^[4] In current clinical practice, controversies including the lack of individualized volume standards for fluid resuscitation, no consensus on the timing of mechanical support intervention, and difficulties in balancing fluid administration and diuresis in mixed shock states highlight the evidence gap in this field and the urgency for further research.

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The pathogenesis of SICM involves multiple molecular abnormalities, such as the release of inflammatory mediators, mitochondrial dysfunction, and calcium homeostasis imbalance. Mechanisms like toll-like receptor-mediated pyroptosis further exacerbate myocardial injury, ultimately leading to hemodynamic changes, including reduced cardiac output and ventricular dilatation.^[5] The hemodynamic effects of fluid resuscitation exhibit a double-edged sword property. During the initial phase, fluid infusion increases preload to enhance cardiac output, but excessive resuscitation can exceed the compensatory range of the Frank-Starling curve. This not only aggravates myocardial oxygen consumption and interstitial edema but may also worsen right heart dysfunction.^[2] Among mechanical circulatory support modalities, VA-ECMO exerts a compensatory role by maintaining systemic perfusion pressure, reducing cardiac workload, and protecting end-organ perfusion. It is more suitable for patients with biventricular dysfunction compared with venovenous-ECMO, though it may also introduce new challenges, such as increased left ventricular afterload.^[6] Evidence-based studies indicate that lactated Ringer's solution may be superior to normal saline for initial resuscitation.^[7] After the implantation of mechanical circulatory support devices, dynamic indicators such as stroke volume variation should be used to assess fluid responsiveness, and hemodynamic parameters should be interpreted in conjunction with a multiparameter monitoring system.^[8] Treatment timing directly affects prognosis. Early fluid resuscitation within 8 hours of onset can improve mortality, but large-volume resuscitation may increase risks when mechanical ventilation resources are limited. The association between SICM and mortality, as well as its dynamic changes, also needs to be incorporated into prognostic assessments.^[9]

Currently, the treatment of SICM still lacks high-quality evidence-based individualized protocols. Key issues such as the optimal support strategy for different hemodynamic subgroups and the balance between fluid administration and diuresis in mixed shock states remain unclear.^[4] Precision therapy guided by biomarkers is limited by insufficient mechanistic research. For instance, the mechanisms underlying the impact of diabetes on SICM have not been fully elucidated.^[10] Monitoring technologies face multiple bottlenecks. The predictive value of traditional parameters decreases during mechanical circulatory support, and there is a lack of bedside quantitative tools for assessing microcirculatory function.^[8] Multicenter studies have poor result comparability due to inconsistent definitions of SICM and varied resource accessibility, and most evidence comes from single-center studies with insufficient follow-up on long-term cardiac function recovery.^[11] Future efforts should focus on developing intelligent decision support systems that integrate multimodal data to resolve contradictions in mixed shock treatment and optimize the judgment of optimal

intervention timing for mechanical support. They should also explore biomarker combinations that distinguish pathological subtypes to guide the prediction of fluid resuscitation response and sequential application of treatments. Additionally, studies on the timing optimization of mechanical support and pharmacotherapy are needed to clarify the microcirculatory effects of different support modalities and the principles of fluid management after device implantation.^[2] At the same time, unified diagnostic criteria for SICM should be established to reduce research heterogeneity.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Meng C and Sun J conducted searches of the scientific literature and developed the draft of the manuscript. Miao G provided contributions to the conception and design of the study, in addition to revising the manuscript with a focus on critical intellectual content. All authors reviewed and approved the final version of the manuscript.

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Ethical approval of studies and informed consent

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