



Rethinking adjunctive dobutamine in septic shock: time to individualize, not generalize

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Dobutamine has been used to treat septic shock when tissue hypoperfusion persists despite fluid resuscitation and vasopressor therapy. Activation of β_1 -adrenergic receptors enhances myocardial contractility and stroke volume, whereas β_2 -mediated vasodilation reduces afterload. Early physiological studies reported improvements in gastric mucosal perfusion, sublingual microcirculation, and lactate clearance [1-3]. These findings led to its inclusion in international guidelines as a possible adjunct for patients with sepsis-related myocardial dysfunction [4]. However, the evidence for meaningful clinical benefits remains weak and inconsistent.

Lim et al. [5] reported data from a large nationwide prospective registry. Among 1,800 patients with septic shock treated with norepinephrine, 108 received dobutamine within the first three intensive care unit (ICU) days. After propensity score matching, no significant differences were observed in Sequential Organ Failure Assessment scores, lactate kinetics, ICU mortality, or in-hospital mortality. Subgroup analysis revealed higher mortality in patients with low early fluid balance who received dobutamine, suggesting that inadequate resuscitation may increase vasodilatory risks. These results highlight the complex hemodynamic effects of dobutamine in patients with septic shock.

In a randomized crossover trial, Hernandez et al. observed improved systemic hemodynamics without gains in microcirculatory flow [1]. A multicenter prospective study by Razazi et al. [6] reported poor tolerance in over half of patients with septic cardiomyopathy, with hypotension and tachyarrhythmias being frequent adverse events. Large observational datasets questioned its role: Zhu et al. [7] reported

higher mortality with dobutamine use in the MIMIC cohort, and Martin et al. showed that while norepinephrine plus dobutamine improved left ventricular performance, this did not improve survival [2]. Recent echocardiography-guided studies suggested benefits in patients with left ventricular dysfunction [8]. However, evidence regarding dobutamine use remains preliminary and inconsistent.

The 2021 Surviving Sepsis Campaign suggests only a weak recommendation, based on low-quality evidence, for adding dobutamine when hypoperfusion persists despite fluids and norepinephrine [4]. Epinephrine alone has been reported as an alternative. However, the guidelines emphasize that dobutamine should be discontinued if patients fail to improve or experience side effects.

A study by Lim et al. [5] reported that dobutamine is not universally beneficial in patients with septic shock and may be harmful in certain settings. Clinicians should adopt a cautious and individualized approach until robust data become available. The strengths of this study include the large, prospectively collected cohort, careful matching of baseline characteristics, and novel analysis of the fluid balance as a potential effect modifier. This study had certain limitations. The lack of a systematic echocardiographic assessment limits the ability to draw firm conclusions regarding patients with confirmed septic cardiomyopathy, a group in which dobutamine may still hold therapeutic value. However, information on the timing, dosage, and duration of infusion is lacking. Finally, as this study was limited to Korean tertiary hospitals, its generalizability to other healthcare systems remains uncertain.

Overall, the available evidence, including that of this study, suggests that dobutamine should not be consid-

ered a routine adjunct treatment for septic shock. Instead, its use should be individualized, with careful attention to fluid status and objective evidence of cardiac dysfunction. Future randomized controlled trials, such as the ongoing the ADAPT-dobutamine trial [9], will be crucial to clarify whether a subset of patients with septic cardiomyopathy benefits from this agent. Future research should explore biomarker- and imaging-guided strategies, examine the interactions between fluid balance and vasopressor dosing, and assess long-term functional outcomes.

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