

Importance of Beta-Blocker Use in Acute Heart Failure: Article Review

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ABSTRACT

Acute heart failure (AHF) is a clinical syndrome characterized by the sudden onset or worsening of signs and symptoms of ventricular dysfunction, often associated with high morbidity and mortality, as well as elevated hospital costs. Traditionally, the pharmacological management of AHF includes loop diuretics, vasodilators and, in selected cases, inotropes. However, the use of beta-blockers well-established drugs in the treatment of chronic heart failure during the acute phase has gained increasing interest, as they could neurohormonally modulate the adrenergic system, improve hemodynamics and reduce arrhythmias. Nevertheless, initiating or continuing beta-blockers during acute decompensation poses challenges, particularly regarding hemodynamic tolerability and the risk of excessive hypotension or bradycardia. Recent systematic reviews and meta-analyses suggest that in hemodynamically stable patients, the continuation or early initiation of beta-blockers before hospital discharge is associated with lower readmission rates and prolonged survival. On the other hand, in patients with compromised perfusion pressure, temporary withdrawal may be necessary, with gradual reintroduction indicated as soon as clinical stability is achieved.

Keywords: Acute heart failure; Beta-blockers; Cardiac decompensation; Pharmacological management; Neurohormonal

Introduction

Acute heart failure (AHF) is one of the leading causes of hospital admissions among adults and the elderly worldwide. It is considered a high-complexity medical emergency with significant morbidity and mortality. The disease is marked by a rapid decline in cardiac function, often overlapping with a prior history of chronic heart failure or presenting as a first-time event in patients without known ventricular dysfunction. In developed countries, it is estimated that 1% to 2% of the adult population is affected by heart failure, with projections of continued growth due to population aging, increased prevalence of cardiovascular

comorbidities and extended survival of heart disease patients¹. The management of AHF has traditionally focused on symptom control and hemodynamic stabilization through diuretics, vasodilators and inotropic agents. These interventions aim to relieve congestion, restore tissue perfusion and prevent organ dysfunction caused by hypoperfusion. However, hemodynamic strategies alone do not always ensure sustained improvement in clinical outcomes, underscoring the need for therapies that also target the neurohormonal mechanisms underlying the syndrome.

Beta-blockers are widely used in chronic heart failure with reduced ejection fraction, supported by large multicenter trials

and international guidelines. Drugs like metoprolol, carvedilol and bisoprolol have demonstrated significant reductions in mortality and hospitalizations in studies such as CIBIS-II and COPENICUS by blocking the harmful effects of sympathetic overactivity and adverse ventricular remodeling^{2,3}. However, AHF presents additional challenges, as introducing or maintaining beta-blockers may compromise hemodynamic stability, increasing the risk of bradycardia, hypotension and worsening systemic perfusion. Despite these risks, recent data from meta-analyses and observational studies indicate that in hemodynamically stable patients, continuing previously prescribed beta-blocker therapy or even initiating it during hospitalization, after stabilization, may be associated with reduced mortality, fewer readmissions and improved quality of life. Abrupt discontinuation of these agents during hospitalization can lead to rebound tachycardia, heightened sympathetic activity and electrical myocardial instability.

Objectives

This article aims to critically review the available scientific evidence on the use of beta-blockers in acute heart failure, discussing their mechanisms of action, indications, risks and potential benefits, in light of the latest guidelines from the European Society of Cardiology (ESC) and the American Heart Association (AHA).

Materials and Methods

A literature review was conducted using the databases PubMed, SciELO, Google Scholar and ScienceDirect.

Discussion

The use of beta-blockers during the acute phase of heart failure is controversial, given that clinical instability can be exacerbated by any intervention that reduces cardiac output or affects heart rate. Nonetheless, it is important to consider that sustained adrenergic activation, common in most AHF patients, worsens the condition through mechanisms such as increased afterload, subendocardial ischemia and arrhythmogenesis⁴. By antagonizing β_1 -adrenergic receptors, beta-blockers may attenuate these harmful effects, reduce myocardial oxygen consumption and promote favorable ventricular remodeling. Several observational studies show that discontinuing beta-blockers during decompensation is associated with increased adverse events and in-hospital mortality. Malik, et al. and Beygui, et al., for example, found that continuing beta-blockers in clinically stable patients was linked to lower in-hospital mortality and improved functional recovery^{5,6}. Smith, et al. meta-analysis reinforces this by indicating that early initiation or resumption of therapy before discharge significantly reduces 30-day readmission rates. However, there is no absolute consensus on the optimal timing to reintroduce beta-blockers after AHF control⁷.

The widely accepted approach is “start low, go slow,” initiating with minimal doses and progressively uptitrating based on hemodynamic tolerance^{8,9}. Drug selection is also relevant: beta-blockers with higher β_1 selectivity, such as bisoprolol, tend to have better tolerability in relatively unstable settings. ESC (2021) and AHA/ACC (2022) guidelines emphasize maintaining beta-blocker therapy if already prescribed before the acute event¹⁰, provided the patient is normotensive, with no signs of hypoperfusion, cardiogenic shock or dependence on inotropes. Initiation during hospitalization should occur after clinical stabilization,

preferably within the last 48-72 hours before discharge to ensure structured outpatient continuation.

Certain patient subgroups require additional caution. In elderly individuals and those with renal dysfunction, the risk of hypotension and bradycardia is more pronounced, requiring careful dose adjustment. Conversely, patients with concurrent atrial fibrillation may derive dual benefits from beta-blockers due to their heart rate control and neurohormonal modulation¹¹. Additionally, the field lacks robust randomized clinical trials specifically designed to evaluate beta-blockers in the acute phase. Most evidence comes from studies on chronic heart failure or secondary analyses of heterogeneous cohorts. This gap limits direct extrapolation and highlights the need for individualized treatment guided by objective clinical parameters and intensive support. Ultimately, decisions on whether to maintain, initiate or suspend beta-blockers in AHF should be based on careful assessment of hemodynamic stability, peripheral perfusion, renal function and absence of absolute contraindications. A multidisciplinary team and well-structured protocols are essential for guiding management and reducing care variability.

Conclusion

A thorough review of the scientific literature shows that beta-blockers can be beneficial in acute heart failure under specific clinical conditions, especially in patients with adequate hemodynamic stability. The well-established understanding of the harmful neurohormonal effects of chronic sympathetic activation justifies the continued use and, in some cases, cautious initiation of beta-blockers during hospitalization provided there is intensive clinical monitoring and appropriate dosage adjustments. Recent studies suggest that abrupt discontinuation of previously prescribed beta-blockers may worsen outcomes, including increased in-hospital mortality and early readmissions^{12,5}. Conversely, early initiation during hospitalization particularly in patients requiring vasopressors or showing signs of hypoperfusion can be hazardous and should be avoided. The “start low, go slow” strategy remains essential, allowing for safe and gradual beta-blocker initiation with titration only after full clinical stabilization. This approach facilitates progressive cardiovascular adaptation while preserving long-term neurohormonal benefits^{8,9}.

International guidelines strongly recommend that clinical decisions be based on objective hemodynamic stability parameters, such as stable blood pressure, absence of inotropes and adequate peripheral perfusion^{10,1}. Patients with cardiogenic shock, unstable tachyarrhythmias or severely impaired renal function require frequent reassessments, with beta-blocker reintroduction postponed until clinical conditions permit. Moreover, implementing standardized protocols and continuous monitoring in intensive care units can optimize the introduction of these agents, enhancing clinical safety. Staff education, pharmacist involvement and structured outpatient follow-up are key complementary strategies to ensure adherence, proper titration and adverse effect surveillance. The future of beta-blocker therapy in AHF depends on the development of randomized trials that stratify patients by hemodynamic profile, age, comorbidities and ventricular dysfunction type. These studies will expand the evidence base and refine guidelines, advancing personalized, data-driven medicine. In conclusion, beta-blockers remain a valuable tool in managing acute heart failure, provided their use is guided by scientific evidence,

rigorous monitoring and a patient-centered approach to maximize benefits and minimize risks in this complex clinical scenario¹³⁻¹⁵.

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