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The Beta-Blocker Question in Post-Myocardial Infarction Care

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Take Home Messages

- Long-term beta-blocker therapy after myocardial infarction (MI) may not benefit all patients.
- Recent large trials and meta-analyses show limited mortality or MI reduction in low-risk patients, with benefits concentrated in those with reduced ejection fraction or heart failure.
- Long-term beta-blocker therapy should be tailored to individual risk, prioritising patients with reduced ejection fraction or symptoms.

Introduction

Beta-blockers have long been a mainstay of therapy for patients following myocardial infarction (MI). Their benefits in reducing mortality, limiting infarct size, and preventing arrhythmias were established in the pre-reperfusion era, particularly for patients with anterior MI or reduced left ventricular function. However, the management of post-MI patients has evolved. The widespread use of percutaneous coronary intervention (PCI), modern antiplatelet therapy, and high-intensity statins has improved outcomes, prompting a reassessment of whether all patients require long-term beta-blockers.

Evidence from Contemporary Trials and Meta-Analysis

Recent trials provide valuable insights. The REDUCE-AMI trial enrolled over 5,000 patients with acute MI and preserved left ventricular ejection fraction (LVEF) above 50% who had undergone PCI (1). Patients were randomised to receive beta-blockers or no beta-blockers, with a median follow-up of 3.5 years. No significant difference was observed in all-cause mortality or recurrent MI, with event rates of 7.9% in the beta-blocker group and 8.3% in controls (hazard ratio, 0.96; 95% confidence interval, 0.79 to 1.16; P=0.64) (1). This suggests that routine beta-blockers may not provide additional survival benefit in low-risk patients with preserved ventricular function. Beta-blockers remained effective, however, in patients with reduced LVEF (<40%) or other high-risk features, highlighting the importance of careful patient selection.

The ABYSS trial enrolled over 3,600 stable post-MI patients with left ventricular ejection fraction (LVEF) ≥40% who had been on chronic beta-blocker therapy and were at least six months from their index MI (2). Patients were randomised to beta-blocker interruption versus continuation, with a primary composite endpoint of all-cause death, non-fatal MI, non-fatal stroke, or cardiovascular hospitalisation over a median follow-up of approximately three years. Interruption of therapy did not achieve non-inferiority compared with continuation (23.8% vs 21.1% for the primary endpoint; hazard ratio ~1.16, 95% CI, 1.01 to 1.33; P=0.44 for noninferiority) and did not improve quality of life, providing no evidence that discontinuation is safe in this population and underscoring that

routine withdrawal of long-term beta-blockers cannot be recommended based on these data alone (2).

The REBOOT-CNIC trial examined over 8,000 post-MI patients receiving guideline-directed therapy, including PCI, dual antiplatelet therapy, and statins (3). Among patients discharged with LVEF above 40%, beta-blockers did not affect rates of death, reinfarction, or heart failure hospitalisation (3). As in REDUCE-AMI, protective effects persisted in patients with reduced LVEF, lowering the risk of heart failure and arrhythmic complications. Together, these trials indicate that survival benefits in contemporary practice are concentrated in patients with ventricular dysfunction or higher arrhythmic risk.

Further evidence comes from the BETAMI and DANBLOCK trials in Norway and Denmark (4). Both enrolled patients within one to two weeks of MI who had LVEF 40% or higher and no clinical heart failure. Participants were randomised to long-term beta-blockers or no beta-blockers. The primary endpoint, a composite of all-cause mortality, new MI, unplanned coronary revascularisation, ischaemic stroke, heart failure, or malignant ventricular arrhythmias, occurred in 14.2% of the beta-blocker group and 16.3% of controls (hazard ratio, 0.85; 95% confidence interval [CI], 0.75 to 0.98; P=0.03) (4). All-cause mortality was similar, but the incidence of new MI was lower among patients receiving beta-blockers. Differences in heart failure, arrhythmias, unplanned revascularisation, or stroke were not statistically significant, although the trials were not powered for these individual outcomes. Overall, long-term beta-blockers modestly reduced major cardiovascular events, primarily by preventing recurrent MI, even in patients without reduced LVEF or heart failure.

A large individual patient data meta-analysis pooled data from five contemporary randomised trials (including REBOOT, REDUCE-AMI, BETAMI, DANBLOCK, and CAPITAL RCT) comprising 17,801 post-MI patients with preserved LVEF (≥50%) (5). In this cohort treated with modern revascularisation and guideline-directed therapy, long-term beta-blocker therapy, irrespective of the specific agent, did not significantly reduce the incidence of all-cause death, myocardial infarction, or heart failure compared with no beta-blocker therapy over approximately 3.5 years of follow-up (5). These findings further reinforce the notion that, in patients with preserved ventricular function post–myocardial infarction and no other indications for beta-blockers, routine use provides little incremental benefit in preventing major adverse cardiovascular events, with observed effects reflecting a class effect rather than agent-specific differences. It is worth noting, however, that the median follow-up duration was relatively short.

Conclusion

These findings support a more nuanced approach to beta-blocker use following MI. While early trials demonstrated clear survival benefits, contemporary patients, who are often rapidly revascularised and receive potent medical therapy, may not all require indefinite treatment. Recent large randomised trials and meta-analyses show that routine long-term beta-blocker therapy in patients with preserved ventricular function offers limited additional protection against death or recurrent MI. Nonetheless, beta-blockers remain essential for patients with heart failure, reduced ejection fraction, or persistent symptoms such as angina or arrhythmias. Going forward, beta-blocker therapy should be tailored to individual risk, optimising benefit while minimising unnecessary treatment.

References:

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