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Letter: Beta blocker withdrawal post-MI – the missed dimension of patient symptoms

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I commend Rossello et al for their important analysis of the REBOOT trial[1]. Their findings – that beta blocker (BB) withdrawal in patients with myocardial infarction (MI) and preserved ejection fraction is not associated with an increased risk of recurrent ischaemic events (hazard ratio [HR] 0.98, 95% confidence interval [CI]: 0.82-1.16) – are particularly practice-changing. The significance is underscored in the subgroup of patients already on chronic BB therapy, where randomisation to stop medication was not associated with an increased risk of the composite ischaemic endpoint (HR 0.93, 95% CI: 0.64-1.34). This directly mitigates the long-held clinical fear of a harmful rebound phenomenon.

However, the study's exclusive focus on hard endpoints risks creating an incomplete definition of "safety". In clinical practice, the impetus for deprescribing often arises not from a change in guideline indications but from the patient's lived experience with the medication – reporting intolerable side-effects like fatigue, dizziness, or sexual dysfunction[2]. The primary goal in these scenarios is to improve quality of life, a dimension on which the REBOOT analysis, while reassuring on ischaemic risk, is necessarily silent.

A patient whose MI has been successfully treated but who now experiences daily palpitations post-BB withdrawal is not a therapeutic success. These symptoms, while not qualifying as a major adverse event, are deeply consequential. They trigger a cascade of clinical activity – phone calls, office visits, Holter monitors, and emergency department evaluations – that generates significant patient anxiety and healthcare costs. By omitting this layer of patient experience, the study cannot inform clinicians about the true net benefit or harm of a withdrawal strategy.

Essentially, the REBOOT analysis has firmly established the first pillar of deprescribing safely: ischaemic safety. It confirms that withdrawing a BB is unlikely to cause a heart attack or dangerous arrhythmia. But it leaves the second pillar, symptomatic and functional safety, unexamined. This second pillar addresses the questions that matter most to a patient's daily life: will I feel better or worse? Will I have new, troubling symptoms like palpitations or shortness of breath? Will I be able to return to my desired level of activity? Therefore, while Rossello et al provide a critical safety net against catastrophic events, their work should be seen not as the final word but as the essential first chapter in the story of post-MI deprescribing. The next chapter must be written by trials that integrate major adverse cardiovascular event endpoints with validated patient-reported outcome measures. Only by capturing both dimensions can we develop truly patient-centred guidelines that define success not just by the absence of reinfarction but by the restoration of well-being.

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Conflict of interest statement

A.B. Shamsulddin has no conflicts of interest to declare.

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