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EDITORIAL

Ticagrelor-based DAPT after CABG: in whom and for how long?

Mattia Galli[1,2] · **Mattia Vinciguerra**[3] · **Ernesto Greco**[3]

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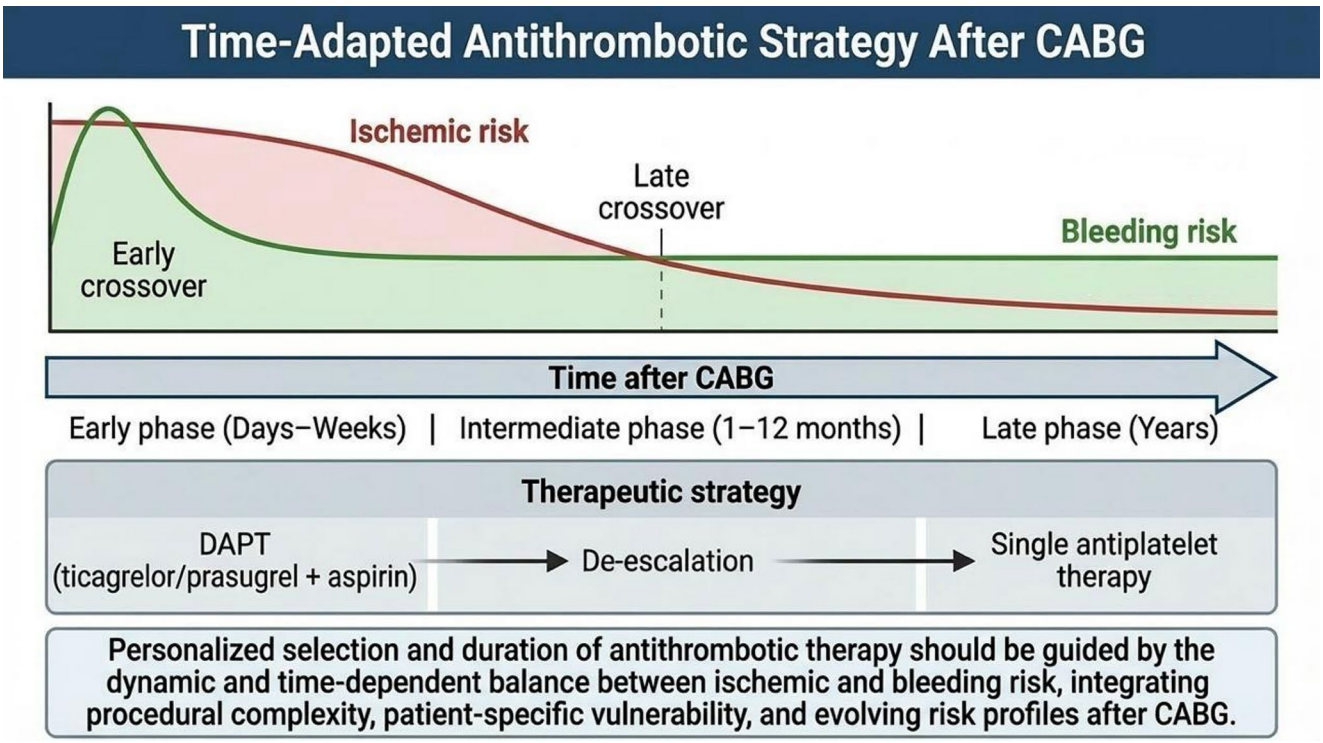
Graphical abstract

Time-Adapted antithrombotic therapy strategy after coronary artery bypass grafting. Abbreviations: CABG= coronary artery bypass grafting; DAPT= dual antiplatelet therapy.



Coronary artery bypass grafting (CABG) remains a cornerstone in the management of complex coronary artery disease, particularly in patients with multivessel disease, diabetes, or left main involvement [1]. Despite substantial advances in surgical techniques, conduit selection, and perioperative care, long-term outcomes remain limited by graft failure and recurrent ischemic events, which occur most frequently within the first year after surgery, especially among patients receiving saphenous vein grafts [2]. Graft failure is driven by distinct, time-dependent pathophysiological mechanisms. Early graft failure, typically occurring within the first months after surgery, is predominantly mediated by acute thrombosis resulting from endothelial injury during

✉ Mattia Galli
mattia.galli@uniroma1.it

1 Maria Cecilia Hospital, GVM Care & Research, Cotignola 48033, Italy

2 Department of Medical-Surgical Sciences and Biotechnologies, Sapienza University of Rome, Corso della Repubblica, 79, Latina 04100, Italy

3 Department of Health and Life Sciences, European University of Rome, Via degli Aldobrandeschi 190, 00163, Rome, Italy

conduit harvesting, ischemia–reperfusion injury, altered shear stress, exposure of thrombogenic subendothelial matrix, and technical factors related to graft handling and anastomotic construction [3]. In this context, antiplatelet regimens after CABG may reduce the incidence of early graft failure, with important implications for both short- and long-term outcomes [4]. However, as thrombotic risk declines over time, whereas bleeding risk associated with antiplatelet therapy persists and carries important prognostic implications, careful, individualized balancing of ischemic and bleeding risks—reassessed over time—is required [5].

Aspirin has long been established as the cornerstone of antiplatelet therapy after CABG and is recommended for lifelong use to improve graft patency and reduce major adverse cardiovascular events (MACE) [4, 6]. However, whether intensified antiplatelet regimens - such as dual antiplatelet therapy (DAPT) with aspirin and a $P2Y_{12}$ inhibitor - administered for a defined period after CABG can mitigate residual thrombotic risk and improve outcomes remains uncertain, largely due to the paucity of dedicated randomized controlled trials (RCTs) in CABG populations [4, 7]. Consequently, recommendations have often relied on expert consensus or extrapolation from percutaneous coronary intervention (PCI) studies, despite important differences in risk profiles and underlying pathophysiology [4]. In contrast to PCI, CABG achieves revascularization by bypassing both obstructive and non-obstructive lesions, and graft failure follows distinct pathophysiological mechanisms compared with stent thrombosis, restenosis, or progression of native coronary disease typically observed after PCI.

In this context, the meta-analysis by Hammadeh et al. provides a timely synthesis of the available evidence on intensified antiplatelet therapy after CABG, comparing ticagrelor-based DAPT for 12 months with aspirin monotherapy [8]. Ticagrelor-based DAPT was associated with a significant reduction in trial-defined MACE and stroke, without a corresponding benefit in all-cause mortality, myocardial infarction, or cardiovascular death [8]. Importantly, these ischemic benefits were offset by a significant increase in major bleeding, underscoring the persistent trade-off inherent to intensified antiplatelet therapy [8].

Several important limitations that temper the strength and generalizability of these findings should be acknowledged. First, of the 11,893 patients included, only 4,905 were enrolled in RCTs, limiting the overall quality of the evidence. Second, the primary efficacy endpoint of MACE was derived from only two studies, with most events contributed by an observational study, raising concerns regarding the robustness and precision of the pooled estimate. Third, the study-level nature of the analysis introduces inherent limitations, including heterogeneity in patient populations, variability in endpoint definitions across studies, and the lack of key subgroup analyses, such as clinical setting (acute vs. stable) or graft type (arterial vs. venous). Fourth, this study does not address the optimal duration of DAPT after CABG. Evidence from the PCI setting and emerging data in CABG populations suggest that a shorter course (i.e., 3 months) of ticagrelor-based DAPT may provide the greatest benefit [4, 9]. In the TACSI trial, Kaplan–Meier curves appeared to favor ticagrelor-based DAPT during the first 3 months, supporting the potential benefit of an initial short course after CABG in patients with acute coronary syndrome, and no advantage was observed beyond this early period, compared to aspirin monotherapy [10]. Additionally, in the yet-to-be-published TOP-CABG trial (NCT05380063), which enrolled 2,290 elective CABG patients in China, comparing 12 months of ticagrelor plus aspirin with a shorter regimen consisting of 3 months of DAPT followed by 9 months of aspirin monotherapy, the shorter strategy was shown to be non-inferior for the primary endpoint of graft occlusion at 1 year.

Overall, accumulating evidence from RCTs in both PCI and CABG settings suggests that routine 12-month DAPT with potent $P2Y_{12}$ inhibitors (i.e., prasugrel or ticagrelor) may be reconsidered in favor of de-escalation strategies, with single antiplatelet therapy after short course (i.e., 3 months) of DAPT emerging as one of the most promising approaches [9]. Therefore, the key question is not whether ticagrelor should be used in addition to aspirin after CABG, but rather for how long and in which patients [11]. Ongoing RCTs, including ODIN (NCT05997693) and SDAT-IRC (NCT03789916), are expected to provide further clarity.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

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