Letters

Ischemia and Infarction in STEMI Patients With Multivessel Disease

Insights From the CvLPRIT Nuclear Substudy

The CvLPRIT (Complete versus Lesion-only PRimary PCI Trial) trial was undertaken in 7 UK centers (1, 2). Patients with ST-segment elevation myocardial infarction (STEMI) and multivessel coronary stenoses were randomized to primary percutaneous coronary intervention (PPCI) to the infarct-related artery (IRA) only, or complete revascularization. At 12-month follow-up, the rate of the combined primary endpoint (all-cause mortality, recurrent MI, heart failure, ischemia-driven revascularization) was lower after complete revascularization. All surviving patients were asked to undergo myocardial perfusion scintigraphy (MPS) 6 to 8 weeks post-admission. It was expected that this a priori nuclear substudy would provide mechanistic insights into the outcome of the main trial, and help to define the clinical role of MPS in the PPCI era.

Stress-rest MPS was performed according to local departmental practice: technetium-99m-tetrofosmin 95%, 2-day protocol 84%, vasodilator stress 84%, glyceryl trinitrate at rest 59%. Blinded semi-quantitative analysis was performed in a central core lab (A.D.K.), and summed scores were expressed as percentages of the left ventricular myocardium (%LV). Separate scores were calculated for IRA and non-IRA territories. Supervising physicians were blinded to the results of MPS unless inducible hypoperfusion exceeded 20%LV (no patient), or symptoms developed within 1 month such that another ischemia test would otherwise have been required (3 patients, all IRA only, no significant inducible hypoperfusion, no further revascularization).

Of 296 CvLPRIT trial patients, 205 (69%) underwent MPS as intended; they were broadly similar to those in the overall study cohort (1). The vast majority were asymptomatic and on optimal medical therapy at the time of MPS. IRA-only patients had more extensive resting defects (infarction) than complete revascularization patients (Table 1). This was associated with a nonsignificant trend toward more extensive infarction in the territory of the index IRA rather than that of a non-IRA. The extent of inducible hypoperfusion (ischemia) was small, and exceeded 10%LV in only 14 patients (7%). There was no difference between the IRA-only and complete revascularization groups (Table 1).

Sixteen patients experienced a late cardiac event following MPS. No scintigraphic variable was predictive of the combined primary endpoint. However, the extent of infarction was greater in patients experiencing death, MI, or heart failure than in those who had no event or a revascularization event: median 23.5 (interquartile range [IQR]: 19.1 to 35.3) versus 8.8 (IQR: 4.4 to 16.2) versus 7.4 (IQR: 2.9 to 10.3) %LV (p < 0.01), whereas resting LV ejection fraction was lower: 43 (IQR: 30 to 45) versus 57 (IQR: 51 to 62) versus 59 (IQR: 46 to 62) % (p = 0.01). The extent of inducible hypoperfusion was similar: 0 (IQR: 0 to 1.5) versus 1.5 (IQR: 0 to 4.4) versus 2.9 (IQR: 0 to 7.4) %LV (p = 0.26).

The reduction in infarct size after complete revascularization might represent early improvement in collateral perfusion from treated non-IRAs to the watershed of the IRA territory. “Hard” cardiac events (as opposed to revascularization) occurring after MPS
were associated with more extensive infarction and more severely impaired LV systolic function. It is therefore plausible that a small reduction in median infarct size explains the lower rate of early heart failure events and death seen in the complete revascularization arm of the CvLPRIT trial (1,3). Interestingly, the CvLPRIT cardiac magnetic resonance substudy showed no significant difference in infarct size between the randomized groups prior to hospital discharge (2). This discrepancy probably reflects differences in the substudy populations, and the likelihood that early imaging overestimated infarct size.

All patients had undergone PCI to the IRA and were receiving contemporary optimal medical therapy. This may explain the limited inducible hypoperfusion seen even in the IRA-only group, and the inability of complete revascularization to reduce it further (4). Therefore residual ischemia is unlikely to be an important driver of further events post-PCI for STEMI, and its suppression alone cannot explain the reduced event rate in the complete revascularization arm of CvLPRIT. Finally, routine ischemia testing in asymptomatic patients following hospital discharge after PCI for STEMI may have a limited yield, even in those with unrevascularized non-IRAs.

**REFERENCES**


**Persistent Atrial Fibrillation**

**Time to Stop Comparing Apples With Oranges**

We read with interest the study by Vogler et al. (1), which showed no benefit of full defragmentation versus pulmonary vein isolation (PVI) in patients who underwent catheter ablation of persistent atrial fibrillation (AF). We believe that there are some aspects of this study that deserve reflection, which may account for the observed lack of benefit of the full defragmentation strategy and may lead to the failure of other strategies under evaluation.

This study clearly illustrates that the population of patients with persistent AF is highly heterogeneous; 52 of 205 patients (25.4%) in the initial cohort reverted to sinus rhythm while they underwent PVI, and thus, they were classified by the investigators as having PV-dependent AF. However, we cannot rule out that even among the remaining patients (who did not revert to sinus rhythm after PVI), some of them may have also presented with PV-dependent AF, because after induction of AF from PV foci, AF may have self-perpetuated in the atria even after PVI (2). In such patients, direct-current cardioversion after successful PVI would likely be enough to allow long-term persistence of sinus rhythm, and this seemed to be the case in most of the patients in the PVI-only group (75% of those who underwent a single ablation procedure). Logically, there would be no benefit of additional defragmentation in this subset of patients, which may have occurred in some patients in the full defragmentation group. The key is to try and identify such patients prospectively.