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EDITORIAL COMMENT

Timely Primary Percutaneous Coronary Intervention

A Call to Action in the Post-Coronary Artery Bypass Graft Patient*

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Patients who have previously undergone coronary revascularization bring a variable degree of added complexity to the delivery of timely reperfusion in ST-segment elevation myocardial infarction (STEMI). Patients with previous revascularization are more likely to be elderly and to have comorbidities that blunt the symptomatic presentation of ischemia and its recognition and to be at increased risk. Knowledge of abnormal baseline electrocardiograms and details of previous revascularization are frequently absent in previously revascularized patients, and diagnostic procedures are often more lengthy. Arterial access options may be limited by peripheral vascular disease and other patient-specific factors. Delays to reperfusion (increased ischemic time) translate into increased infarct size and mortality. Recent studies in which the time of onset of myocardial infarction was measured objectively by backward extrapolation of troponin curves suggested that the biological onset of ischemia and infarction was substantially earlier than the patient-recognized symptom onset and that the differences were more pronounced in older patients and those with more complexity (1). Interestingly, and perhaps not surprisingly, patients having undergone a previous percutaneous coronary intervention (PCI) were more likely to recognize the onset of ischemia, which may lead to better outcomes in these patients.

Current guideline statements do not provide specific recommendations for STEMI patients with previous surgical revascularization, a patient subgroup with a number of issues not encountered in nonsurgical patients, including the need to treat bypass grafts. In the APEX-AMI (Assessment of PEXelizamab in Acute Myocardial Infarction) trial of 128 post-coronary artery bypass graft (CABG) patients, 63 (41%) had saphenous vein graft (SVG) culprit vessels, and the 90-day mortality rate was significantly increased in SVG-treated patients (19% vs. 5.7%; $p = 0.05$) (2). Similarly, in 192 post-CABG patients who underwent primary PCI, Gaglia et al. (3) reported a significantly higher 30-day mortality rate in patients with SVG compared with native vessel PCI (14.3% vs. 8.4%; $p = 0.03$). In a more recent single-center, retrospective study, Kohl et al. (4) reported that 249 of 3,542 consecutive STEMI patients (7%) had a previous CABG, that the culprit vessel was an SVG in 34%, a native vessel in 42%, and no clear culprit in 24%. Despite higher comorbidity, patients with a previous CABG had a similar mortality rate compared with those without a previous CABG in-hospital (4.8% vs. 5.2%; $p = 0.82$) and at 1 year (10.8% vs. 9.1%; $p = 0.36$). The mortality rate was numerically higher (but not statistically) with an SVG PCI compared with a native vessel PCI at 30 days (8.3% vs. 3.9%; $p = 0.19$) and at 1 year (14.3% vs. 9.0%; $p = 0.35$). The American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction) classified a PCI of an SVG as Class IIa, "acceptable, of uncertain efficacy and may be controversial; weight of evidence in favor of usefulness/efficacy" (5).

SEE PAGE 1954

In this issue of *JACC: Cardiovascular Interventions*, Gruberg et al. (6) report from the National Cardiovascular Data Registry (NCDR) in-hospital outcomes of 15,628 STEMI patients treated from

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2009 through 2011 in 297 U.S. hospitals comparing patients with previous revascularization (CABG or PCI) to those without previous revascularization. They excluded patients with shock, cardiac arrest, and between-hospital transfers. Patients with previous surgical revascularization were significantly older than those with a previous PCI or no previous revascularization (66 vs. 60 vs. 59 years; $p = 0.001$) and had significantly more diabetes, peripheral vascular disease, dyslipidemia, heart failure, previous myocardial infarction, and long-term dialysis. The unadjusted mortality rate of post-CABG patients was significantly higher than that of patients with no previous revascularization (3.3% vs. 1.8%; $p = 0.0089$), with a numerically higher mortality rate in patients with culprit lesions in a graft (4.3% vs. 1.8%; $p = 0.065$). Unadjusted major adverse cardiac and cerebrovascular events and major bleeding were similar in the 3 groups of patients.

There were significant between-group differences in angiographic and procedural characteristics. Lesion complexity was greater in post-CABG patients, and left ventricular function was poorer. The culprit vessel was the left anterior descending coronary artery in ~40% of patients with a previous PCI or no previous revascularization and in only 14.5% of post-CABG patients. In previous CABG patients, the target vessel was the right coronary artery in 56% of patients. These findings suggest that less myocardium was at risk in post-CABG patients, which may have offset to a degree the advanced age and comorbidity of post-CABG patients. An important and potentially modifiable factor in post-CABG patients was the lower percentage of patients achieving door-to-balloon time ≤90 min compared with patients with a previous PCI or no previous revascularization (76.4% vs. 88.5% vs. 88%). A door-to-balloon time ≤90 min was achieved in 90.1% of previous PCI patients with in-stent restenosis, but in only 75.9% when the culprit lesion was in a bypass graft. This delay to reperfusion has important implications for myocardial salvage. However, door-to-cath lab and cath lab-to-balloon times were not provided, so it is not clear where the delay occurred. Increased awareness of "time is muscle" may lead to shortened door-to-cath lab times, and timely performance of the more complex diagnostic study in post-CABG patients may shorten cath lab-to-balloon times in these patients. It is noteworthy that, at Abbott Northwestern Hospital, a center with a special focus on primary PCI, door-to-balloon times for post-CABG patients were not different from those of patients without previous surgery and that in-hospital and 1-year mortality rates were similar (4).

In the patients with a previous CABG in this NCDR report, 54.4% underwent PCI of a graft lesion, and there was a trend among these patients for increased unadjusted and adjusted mortality compared with native vessel PCI. The frequency of use of embolic protection devices (EPDs) was not stated. There has been no dedicated trial to evaluate their use in STEMI with an SVG culprit vessel, and results of studies in native vessels have not been encouraging. In a recent report from the NCDR for the 2005 to 2009 period, EPDs were used in only 21% of SVG PCIs and even less frequently in STEMIs (7). Given the complexity of the infarct vessel with increased thrombus burden, impaired visualization of the distal vessel and the need to cross the culprit lesion with a bulky filter, it seems unlikely that more frequent use of EPDs would have a positive impact on outcomes. In the observational NCDR report by Brennan et al. (7), use of EPDs was associated with a slightly higher risk of procedural complications, calling into question the value of routine use of EPDs. The increased cost and complexity of EPDs and potential complications have negatively affected their use in contemporary SVG PCI where direct stenting, conservative stent sizing, and avoidance of post-deployment balloon dilation yield a low rate of periprocedural myocardial infarction and no reflow in selected patients without bulky SVG target lesions. Notably, in the ISAR-CABG (Intracoronary Stenting and Antithrombotic Research-CABG) study, in which 610 patients underwent stent implantation in SVGs, the occurrence rate of major adverse cardiac events at 30 days was only 4% despite the use of EPDs in <5% of patients (8). It seems clear that practice patterns in place at the time that the SAFER (Saphenous vein graft Angioplasty Free of Emboli Randomized) trial was conducted were quite different from those used today. In the SAFER trial, which is the only randomized trial in which embolic protection was compared with no embolic protection, further balloon dilation after stent deployment, a practice usually avoided currently, was used in 40% of control patients and 27% of EPD-treated patients (9). Perhaps of even more significance, the balloon size used was larger than the reference vessel diameter (4.2 mm vs. 3.4 mm). Post-dilation with a large balloon, which was performed more in the control group, may have increased periprocedural myocardial infarction.

Perhaps the most important take-home message from the work of Gruberg et al. (6) is that patients with a previous CABG presenting to NCDR hospitals with STEMI experienced delays to reperfusion compared with other patients and had a higher unadjusted rate of in-hospital major adverse cardiac and cerebrovascular events. This contrasts with the

experience reported by Kohl et al. (4), who, by using standardized protocols in a regional STEMI system, were able to achieve equally prompt reperfusion and similar outcomes in post-CABG patients compared with other STEMI patients. The observations of Gruberg et al. (6) should be interpreted as a "call to action," with the goal of earliest possible reperfusion in all patients with STEMI, including those who have had a previous CABG.

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