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LETTERS TO THE EDITOR

Saphenous Vein Graft Intervention

Discussion on Acute Vein Graft Occlusion Intervention

We read with interest the State-of-the-Art paper on saphenous vein graft (SVG) intervention by Lee et al. (1), and commend the authors for a comprehensive review.

We would like to highlight that the review failed to mention the management of ST-segment elevation myocardial infarction due to acute SVG occlusion. Retrospective data (2,3) have confirmed this area remains a particularly high-risk subset of SVG intervention, with 30-day mortality at 14.3% and major adverse cardiac events rate of 36.8% at 1 year. These are significantly worse than contemporary outcomes of acute coronary syndrome from coronary artery culprit lesions. We previously presented (4) a case of an 84-year-old patient with an ST-segment elevation myocardial infarction of a thrombotic occlusion in the SVG to the posterior descending artery, during which difficulty was encountered in restoring and maintaining flow in the culprit vessel. Significant effort was focused on preventing no-reflow with: 1) continuation of the upstream tirofiban infusion; and 2) manual (Export catheter, Medtronic Inc., Minneapolis, Minnesota) and mechanical thrombectomy (Angiojet rheolytic thrombectomy catheter, Medrad Inc., Warrendale, Philadelphia). A distal embolic protection device (Emboshield Abbott Vascular, Santa Clara, California) was delivered distal to the culprit lesion before the placement of a bare-metal stent. The stent nonetheless led to no-reflow, necessitating treatment with adenosine, verapamil, and nitrate intracoronary infusion through a local delivery balloon (ClearWay RX, Atrium Medical, Hudson, New Hampshire). The final result was TIMI (Thrombolysis In Myocardial Infarction) flow grade 3 with no local complication. A 12-month clinical follow-up revealed no reintervention or rehospitalization.

We feel that acute SVG occlusion deserves a special mention. There is currently little data applicable to this group of patients, as virtually all trials on the aforementioned pharmacotherapy (i.e., IIb/IIIa inhibitors, adenosine, verapamil) and devices (thrombus aspiration and distal embolic protection) excluded patients with acute SVG occlusion. The significant thrombus burden in the culprit vessel makes a satisfactory procedural outcome difficult and the resulting compromised epicardial flow impairs long-term outcome (5). It is not foreseeable that robust evidence-based guidelines will ever become available given this relatively infrequent occurrence. Interventional cardiologists may be resigned to extrapolate data from research on SVG and acute coronary syndrome interventions and be resourceful in approaching these lesions using all the tools that are available in interventional cardiology.

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Reply

We are grateful to Drs. Poon, Roati, and Walters for their interest in our State-of-the-Art paper (1). Their case highlights the complexities encountered during treatment of ST-segment elevation myocardial infarction (STEMI) due to saphenous vein graft (SVG) thrombosis.

Acute occlusion of SVGs is commonly associated with extensive atherosclerotic and thrombotic burden, which increases the risk of distal embolization and no-reflow and, thus, death and myocardial infarction (2). Aged SVGs develop a form of accelerated atherosclerosis in which the plaque composition is highly friable, often described as having the consistency of "gruel" or "cottage cheese." As a result, glycoprotein IIb/IIIa inhibitors are ineffective, and embolic protection devices remain the default therapy to prevent periprocedural complications. Randomized trials have demonstrated that, although these devices are helpful, they are incapable of preventing all instances of distal embolization (3). These considerations are likely to be even more relevant in the case of an occluded SVG with STEMI, and once the SVG occludes distally—given the lack of side branches—the SVG often backfills extensively with thrombus. Fortunately, SVG occlusion is responsible for <5% of STEMI (34 of 3,602 infarctions in the HORIZONS-AMI [Harmonizing Outcomes with Revas-

cularization and Stents in Acute Myocardial Infarction] trial) (4), and, hopefully, this number will further decrease with the increasing emphasis on pan-arterial revascularization.

We agree that, given its infrequent occurrence, it will be difficult to accrue meaningful data on best treatments for STEMI from adequately powered randomized trials. Absent such data, we empirically recommend potent antiplatelet and antithrombotic agents (in patients at low risk of bleeding) and liberal use of intragraft vasodilators to prevent and treat no-reflow. Either proximal or distal embolic protection devices should also routinely be used, with consideration of aspiration or thrombectomy for further debulking. Drug-eluting stents have now been shown to be safe in SVGs and might decrease restenosis (5). Preferential intervention of the native coronary arterial circulation (rather than the occluded SVG) should always be considered as an alternative route to reperfusing the myocardium and, if possible, will result in higher acute success and late patency rates (6).

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Percutaneous Coronary Interventions Following Coronary Artery Bypass Graft

In-Hospital Mortality and Long-Term Follow-Up

We have read with great interest the paper from Brilakis et al. (1) concerning percutaneous coronary interventions (PCI) following coronary artery bypass graft (CABG). Patients with a previous CABG constitute an extremely high-risk group with an increased frequency of comorbidities and multivessel disease. Brilakis et al. notice very well that patients undergoing bypass graft PCI have higher-risk clinical characteristics. Our attention was especially attracted to the very important and didactic conclusion that compared with native coronary PCI, bypass graft PCI is independently associated with higher in-hospital mortality, which relates to our research that soon will be published by Xanthopoulou et al. (2). Interestingly, the 2 papers supplement each other, as at this retrospective analysis of 190 patients, we found that post-CABG patients, undergoing graft compared with native vessel PCI have worse long-term clinical outcome (follow-up with a median duration of 28 months) in terms of major adverse cardiac events, death, and repeat revascularization. Brilakis et al. also found that saphenous vein graft PCI becomes more prevalent with longer time intervals from CABG, a finding in accordance with our results.

By contrast, although the efficacy of embolic protection devices is proven, the investigators do not mention the frequency of their use in the saphenous vein graft PCI population (3). Additionally, it would be important to assess the amount of myocardium in jeopardy following PCI in the 2 groups. According to our opinion, it would be helpful to incorporate those parameters in the multivariate analysis, as they could have influenced the clinical outcome of those patients (4).

Disease progression to the native vessels (NV) or failure of grafts due to similar mechanisms with NV atherosclerosis usually lead to repeat coronary revascularizations (5). Furthermore, the NV is recommended as the ideal target for PCI when diseased grafts are >3 years old (6).

In conclusion, it is clear that regardless of the selection criteria for the PCI target vessel, patients subjected to graft PCI have worse in-hospital mortality than patients who had NV-PCI, and these data could be enhanced with our results adding details about long-term clinical outcome. Physicians should prefer a NV if PCI is to be performed in a patient following CABG, if the anatomy of the vessels of the heart is suitable for it.

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