Orbital Atherectomy and Heavily Calcified Saphenous Vein Graft Intervention

Percutaneous coronary intervention in the diseased saphenous vein graft differs significantly from that in the diseased native coronary artery. After being exposed to arterial pressures over time, vein grafts have substantially different plaque characteristics, with more inflammatory cells, more diffuse disease, and less calcification. Severe calcification of saphenous vein grafts, although uncommon, poses a high risk of stent underexpansion. Orbital atherectomy for treatment of de novo calcified coronary lesions has been associated with better outcomes at 5-year follow-up. However, there are no published data on the use of orbital atherectomy to treat severely calcified saphenous vein graft lesions. We present the case of a 77-year-old woman with non-ST-segment-elevation myocardial infarction who underwent successful orbital atherectomy to prepare a severely calcified saphenous vein graft lesion for stent implantation. (Tex Heart Inst J 2020;47(1):41-3)

Saphenous vein graft (SVG) failure is one of the main contributors to poor long-term ischemic outcomes and death after coronary artery bypass grafting (CABG). Ten percent to 15% of SVGs occlude within one year, and nearly half fail within 10 years after CABG, presumably because of thrombosis, neointimal hyperplasia, or atherosclerotic degeneration.¹ Performing percutaneous coronary intervention (PCI) in the diseased SVG differs significantly from performing it in the diseased native coronary artery. Vein grafts exposed to arterial pressures over time have substantially different plaque characteristics, including more inflammatory cells, more diffuse disease, and less calcification.² Percutaneous intervention in SVG plaques is technically challenging, and it is associated with a high risk of distal embolization, no-reflow phenomenon, and periprocedural myocardial infarction (MI).³ Although severe calcification of SVGs is uncommon, its presence can lead to underexpansion of stents during deployment. We present the case of a patient with non-ST-segment-elevation MI (NSTEMI) in whom we used the DIAMONDBACK 360® Coronary Orbital Atherectomy System (OAS) (Cardiovascular Systems, Inc.) to prepare a severely calcified SVG lesion for stent implantation.

Case Report

In January 2017, a 77-year-old woman presented after a 15-minute episode of dull midsternal nonradiating chest pain (3 on a pain scale of 0–10) the previous night. The patient’s medical history included quadruple CABG 25 years previously, transcatheter aortic valve replacement 2 years previously, stage IV chronic kidney disease, type 2 diabetes mellitus, hypertension, and hyperlipidemia. Physical examination revealed nothing of note and included normal vital signs. An electrocardiogram showed an old left bundle branch block with no new ischemic changes. Results of laboratory evaluation included an elevated troponin I level peaking at 1.19 ng/mL (normal, ≤0.03 ng/mL) and a creatinine level of 2.06 mg/dL (baseline, 1.9–2 mg/dL; normal, 0.5–1.3 mg/dL). The patient was diagnosed as having NSTEMI. An echocardiogram revealed normal systolic function with a left ventricular ejection fraction of 0.70 and a well-seated prosthetic aortic valve with normal gradients.

A coronary angiogram revealed a patent left internal mammary artery graft to the left anterior descending coronary artery (LAD), a patent SVG to the first diagonal branch (D1), and high-grade stenosis of the SVG to the obtuse marginal branch of
the left circumflex coronary artery (Fig. 1). We decided to attempt PCI in the culprit SVG, which was severely calcified. A 6F Amplatz left 1.0 guide catheter was used to engage the SVG, and then a 6F GuideLiner® catheter (Vascular Solutions, Inc.) was used to provide additional support. Although we could introduce a Balance Middleweight (BMW) guidewire (Abbott Vascular) through the lesion, we could not cross it with even the smallest balloon or a Corsair catheter (Asahi Intecc).

Therefore, we decided to prepare the SVG for PCI by performing orbital atherectomy at the lesion site with the Diamondback 360® Coronary OAS. However, we waited 3 days before performing the atherectomy because of the patient’s poor renal function. At that time, a ViperWire® system (Cardiovascular Systems, Inc.) was introduced, and 3 atherectomy runs were performed without issues. Flow improved beyond the graft lesion (Fig. 2). The ViperWire was then exchanged with a BMW wire using a FineCross® MG Coronary Micro-Guide Catheter (Terumo Interventional Systems). The SVG was predilated using a 2.5 × 20-mm NC TREK Coronary Dilatation Catheter (Abbott Vascular), after which a 3.25 × 12-mm drug-eluting stent was easily implanted. The final angiogram showed good flow through the vein graft (Fig. 3). Although we could not use a distal embolic protection device with the OAS because of the severely stenotic segment of the SVG, the patient had no procedure-related MI or other complications. The patient’s postprocedural course was uneventful, and she was discharged from the hospital the next day in stable condition. At her one-month follow-up visit, she had no angina, and her creatinine level was stable at 1.9 mg/dL.

Discussion

When compared with PCI in de novo coronary artery lesions, coronary angioplasty in SVG stenosis is a highly complex procedure that is associated with major adverse cardiac events, including higher rates of MI, in-stent restenosis, target-vessel revascularization, and death. Moreover, distal embolization may occur during SVG intervention and lead to slow or no-reflow phenomena, especially in heavily calcified and fibrotic lesions.

Fig. 1 Coronary angiogram (right anterior oblique caudal view) shows high-grade stenosis (arrow) of the saphenous vein graft to the obtuse marginal branch of the left circumflex coronary artery.

Fig. 2 Coronary angiogram (right anterior oblique caudal view) shows improved flow (arrow) beyond the saphenous vein graft lesion after orbital atherectomy.

Fig. 3 Coronary angiogram (right anterior oblique caudal view) shows good results from the stenting of the saphenous vein graft to the obtuse marginal branch, after orbital atherectomy.
The Diamondback 360 Coronary OAS has been associated with favorable outcomes of PCI in severely calcified native coronary lesions, including stenosis of the unprotected, heavily calcified left main coronary artery. In the first-in-human ORBIT I trial, 5-year follow-up data from one of the 2 trial centers showed that applying OAS before stenting in de novo calcified coronary lesions was safe and effective. In the pivotal ORBIT II trial, atherectomy with the OAS followed by stent placement resulted in low ischemic complication rates in patients with severely calcified coronary lesions and durable outcomes for up to 3 years, including low target-lesion revascularization. In a recent retrospective subanalysis of 458 consecutive patients who underwent orbital atherectomy of heavily calcified coronary lesions, 30-day angiographic and clinical outcomes were compared in those with (n=77) and without (n=381) a history of CABG; outcomes did not differ between the 2 groups. However, no patient with a history of CAGB underwent orbital atherectomy of the bypass graft.

Use of the Rotablator® Rotational Atherectomy System (Boston Scientific Corporation) in nondilatable calcified SVG lesions has been described in a few case reports. Don and colleagues reported successful rotational atherectomy for plaque modification and PCI in a sequential SVG to the LAD–D1 in a patient who presented with acute coronary syndrome.

Meanwhile, to our knowledge, there have been no studies or case reports of orbital atherectomy in heavily calcified SVG lesions. Use of the OAS has been associated with higher rates of successful PCI when compared with traditional methods, without any change in future restenosis rates or other complications. Despite being a more challenging procedure, orbital atherectomy might be the only option for revascularization in select cases. In our case, the patient had a 25-year-old SVG that was severely calcified. Given our inability to pass even the smallest balloon or Corsair through the SVG, we considered atherectomy with the OAS to be the best option for PCI. We think that, once the ViperWire was in the proper position, the continuous movement of the orbital atherectomy device’s crown across the severely calcified lesion was able to fracture the calcium and facilitate the crossing. Moreover, as already noted, even though we could not use a distal embolic protection device with the OAS because of the severely stenotic segment of the SVG, the patient had no procedure-related MI or other complications.

In conclusion, the present case shows that even though orbital atherectomy of an SVG carries slightly higher risks than do traditional balloon angioplasty and PCI, it is a viable option for some patients.

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