Totally Occlusive Diffuse In-stent Restenosis in Saphenous Vein Graft to Right Coronary Artery and Acute Coronary Syndrome

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CASE REPORT

A 78-year-old gentleman presented with Canadian Cardiological Society (CCS) class III angina and had been getting excruciating substernal pain while walking 50 to 100 m on the flat over the last 15 days despite optimal medical therapy. From his past medical history the patient underwent CABG in 1997 (LIMA to LAD and SVG to dominant RCA); in 2006 he had an angioplasty done in his SVG and stents were implanted (no medical data regarding the angioplasty were found). Cardiac enzymes and troponin were negative; a mild increase of creatinine was noted on admission. The echocardiogram revealed severe basal inferior wall hypokinesia with overall reasonably preserved left ventricular and right ventricular systolic function.

The patient was catheterized and patent LIMA graft to LAD was found; the SVG

Abbreviations
BMS = bare metal stent(s)
CCS = Canadian Cardiological Society
DES = drug-eluting stent(s)
LAD = left anterior descending
LIMA = left internal mammary artery
PCI = percutaneous coronary intervention
RCA = right coronary artery
SVG = saphenous vein graft
TLR / TVR = target lesion/vessel revascularization

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FIGURE 1. ECG on admission.
a BMW 0.014-inch guidewire was used. The following balloons were selected: Sprinter 1.25 x 6 mm, NC Durastar 4.0 x 10 mm, NC Trek 5.0 x 15 mm, Cutting balloon (Boston Scientific) 4.0 x 15 mm. A manual thromboaspiration catheter (Export AP 6 Fr) was employed. Finally 2 long drug-eluting stents (DES) were implanted (Endeavor Resolute 3.5x 38 mm and Endeavor Resolute 3.5 x 30 mm).

**PROCEDURE**

The guidewire supported by a small balloon 1.25 x 6mm passed through the occlusion with some difficulty and multiple passages with an Export thromboaspiration catheter were carried out immediately after without predilatation. Due to high thrombotic burden 2 mg of tenecteplase and 500 \( \mu \text{g} \) of adenosine to protect the distal microvasculature were administered through the distal lumen of the thromboaspiration catheter. Beyond the high thrombus grade there was diffuse in stent restenosis and multiple cutting balloon dilatations were utilized. Two long minimally overlapping stents (DES) were deployed accompanied by multiple very high pressure post dilatations with 4.0 and 5.0 mm NC balloons up to 30 bar. The final angiographic result although not perfect was acceptable with 20-30\% residual stenosis, TIMI III flow and blush grade III.

**DISCUSSION**

Percutaneous SVG intervention is a feasible treatment strategy despite the fact that the procedure is extremely chal-
lenging due to high rates of periprocedural adverse events (slow-flow, no-reflow, myocardial infarction), increased restenosis rate and accelerated atheromatous SVG disease beyond the treated segments. The decision was made to treat the SVG instead of recanalization of the native RCA because of many unfavourable anatomic characteristics of this 14-year old chronic total occlusion. A number of studies showed sobering results in SVG intervention and probably one may opt for native vessel PCI if that is feasible.

In our case we had to solve two pathophysiologically distinct issues. The high thrombotic burden on one hand and also the diffuse in stent restenotic process on the other hand. Acute or subacute totally occlusive with high thrombus grade diffuse in stent restenosis in SVGs deserves a special mention.
since there is currently little scientific data applicable to this subset of lesions.

We performed, right from the outset, manual thromboaspiration with a 6F Export AP catheter (without pre-dilation) with multiple passages and also via the central lumen of the aspiration catheter we administered 2 mg of tenecteplase and repeated bolus doses of adenosine (up to 500 μg) as a pharmacological distal protection regimen. Although prophylactic adenosine injections in small boluses (>5 boluses of 24 μg) through the guiding catheter prior to SVG intervention was not found to be protective for the no-reflow phenomenon, we administered it locally through the Export AP catheter in high doses with excellent microvasculature protective effect and post procedure blush grade III.

Brodie et al found a higher in-hospital mortality (21.1% vs 8%, p=0.0004) in patients treated for acute thrombotic SVG occlusion as compared to thrombotic occlusion of native coronary arteries. There are no randomized trials regarding the long term effectiveness and long term prognosis in percutaneous SVG re-interventions due to diffuse in stent restenosis. Although this subset of lesions are infrequent in daily practice we need more data in order to be able to offer our patients the best treatment strategy.

We did not utilize embolic protection device in our case despite the class I ACC/AHA guideline recommendation for a number of reasons. Firstly, because we did not have one immediately available on the shelf and the patient was unstable. Secondly, the vein graft was totally occluded with severe and diffuse in stent hyperplasia and high amount of thrombotic material which could have made distal protection devices extremely difficult to place with a disease free landing zone. Probably, a proximal protection device such as Proxis might have been useful. Thirdly, it is known that protection devices are underused worldwide approximately in 23% of SVG interventions.

Although we did not know the type of stents used in the previous intervention we made up our minds to implant second generation DES (zotarolimus). The safety and efficacy of DES vs BMS remains uncertain due to contradictory reports of either TLR and TVR with DES or a probable excess of clinical events with DES. Nonetheless some available evidence supports treatment with DES in this high risk lesion subset.

REFERENCES