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Saphenous Vein Graft Stenosis

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ABSTRACT

Key clinical message: When used as conduits for coronary atery bypass surgery, saphenous vein grafts (SVG) develop atherosclerotic disease that may result in stenosis or occlusion in 50% of patients by 10 years. SVG intervention has become an attractive alternative to re operation in these patients, but also it is favourable with less acute and long term outcomes compared with percutaneous coronary intervention (PCI) of native vessels. The potentional of Drug Eluting Stents (DES) in improving long term results of SVG interventrtion is still debated and up to date there is no clear evidence of benefit in relevant clinical end point, However a retrospective study conducted in italy commented that the study they conduced supports the growing use of DES for SVG PCI by documenting safety and reduced rates of restenosis and adverse cardiovascular events.

Keywords: Saphenous vein graft; Percutaneous coronary intervention; Drug eluting stents

INTRODUCTION

Coronary artery disease (CAD) is a highly prevalent and initial consensus disease afflicting many. The treatment of CAD ranges from medical management and life style modification to invasive coronary revascularization. Using of SVG has been ongoing since 1967 because of an easy accessibility and reliability conduit with significant length [1].

In most SVG the Great Saphenous vein (GSV) is utilized, however in select circumstances the Short saphenous vein (SSV) may be a suitable option [2].

SVG lesions are associated with a higher restenosis rate, particularly in the proximal anastomotic (58%) and body (52%) portions of the graft. Distal anastomotic narrowing responds to angioplasty well, especially in patients with recurrent CABG surgery [3]. The clinical patency of SVG In arterial circulation can be divided into: Early (0 days-30 days); Short Term (30 days-24 month) and Long Term (More than 24 months).

In The United states, >300,000 patients undergo CABG each year, Graft occlusion before discharge has been reported to occur in approximately 10% of the Venous grafts. During the 1st year after CABG approximately 15%-30% occlusion has been recorded and After 1 year Post CABG annual occlusion rate 2% and it rises approximately 4% annually between post OP and years 4 and 6 [4].

The Main causes of graft diseases are: During the Early Period-Stenosis due to Technical issues occurring at graft anastomoses eg. Position of graft, Kinking of graft, Poor distal runoff which occurs in as many as 10% of total vein graft failure [5]. During the short Term period the possible causes of graft diseases are perivascular myofibroblast remodeling, Platelet derived growth factor, Smooth muscle Proliferation, decreased local nitrogen oxide release, Decreased endothelial relaxation and Initimal vein thickening. The Causes for Diseased graft at

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the long term phase are formation of atherosclerosis with plaque formation to adjacent areas of lipid deposition intimal and hyperplasia [6].

CASE REPORT

We report a case of a 68 year old male patient who was referred for coronary angiography because of clinical presentation of unstable angina for 5 days (CCS?) and a Positive Trop leak of 1.613 ng/mL. He had a history of arterial hypertension, hyperlipidemia, DM type 2, Cigarette smoking, parkinsonism and coronary artery diseases post CABG in 2014 (8 years ago) with LIMA to LAD, SVG to PDA and OM [7].

Electrocardiography showed a Premature Ventricular Complex (PVC), T wave inversion in lead I and aVL (High Lateral Leads), Q wave in lead III (**Figure 1**), Whereas Echocardiography revealed No Regional wall motion abnormality with a EF of 60%, Mild to Moderate Mitral Regurgitation, Moderate Aortic Regurgitation, Mild Tricuspid Regurgitation, Mild Pulmonary Hypertension, Concentric LVH [8].

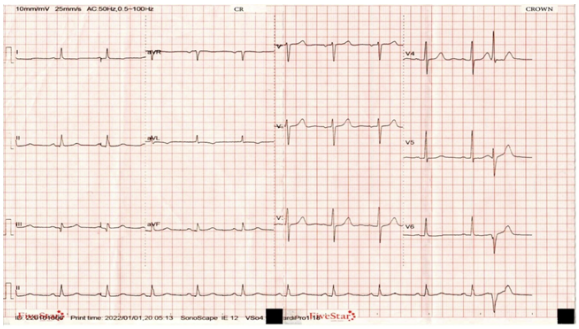

Figure 1: Electrocardiography

Coronary and Graft angiography showed a Normal Left Main Coronary artery with a Patent LIMA with good distal runoff into mid LAD artery and a retrograde flow into proximal LAD artery and LCX artery (**Figures 2 and 3**), Occlusion of the mid SVG to a second OM branch and a patent to the PD branch of proximally (**Figure 4**) [9].

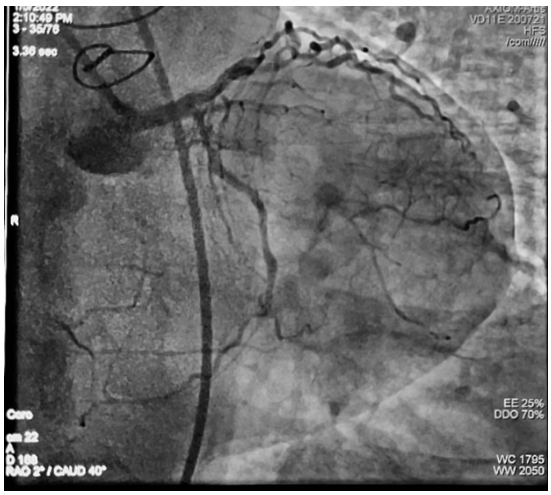

Figure 2: Coronary Angiography of Left Coronary Artery.

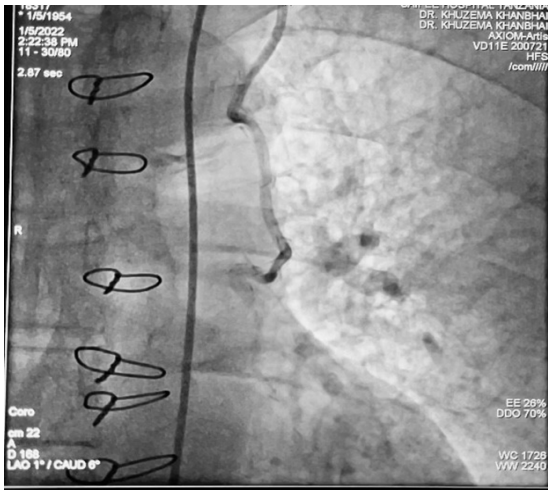

Figure 3: Angiography of LIMA

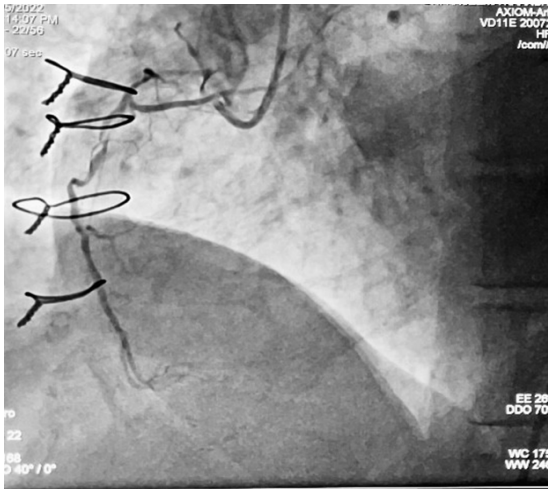

Figure 4: Coronary Angiography Right Coronary Artery

We proceeded with stent angioplasty of the Mid SVG lesion. After low pressure predilation with BrosMed 2.5 x 15 mm Semi complaint balloon (**Figure 5**), Treatment of the critical stenosis of SVG-PDA and OM was successfully done using a 3.0 × 29 mm Angiolite sirolimus and excellent angiographic result was obtained (**Figures 6 and 7**). No reflow phenomenon was not observed and final angipgraphy showed good dilation and patency of the SVG with normal distal runoff into the Dg artery and retrograde flow distribute to a large area of the anterior-lateral wall [10] (**Figures 8 and 9**).

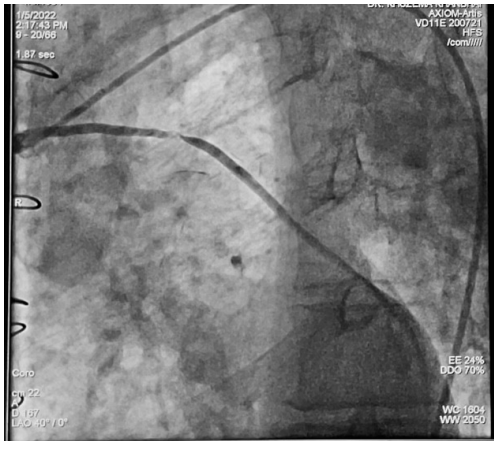

Figure 5: Angiography of SVG – Showing Mid SVG Stenosis

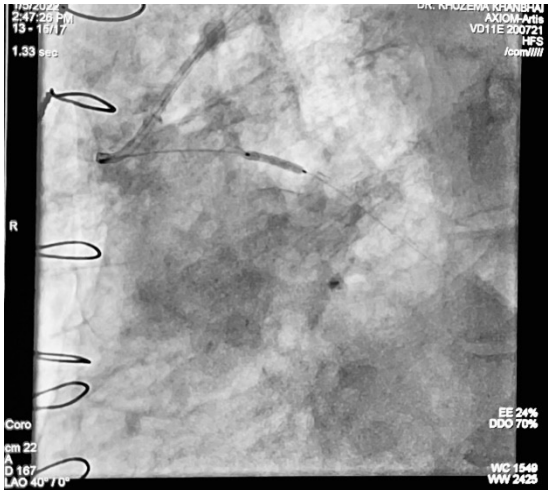

Figure 6: (Angio) Showing Ballooning (Pre Dilation) of Mid SVG

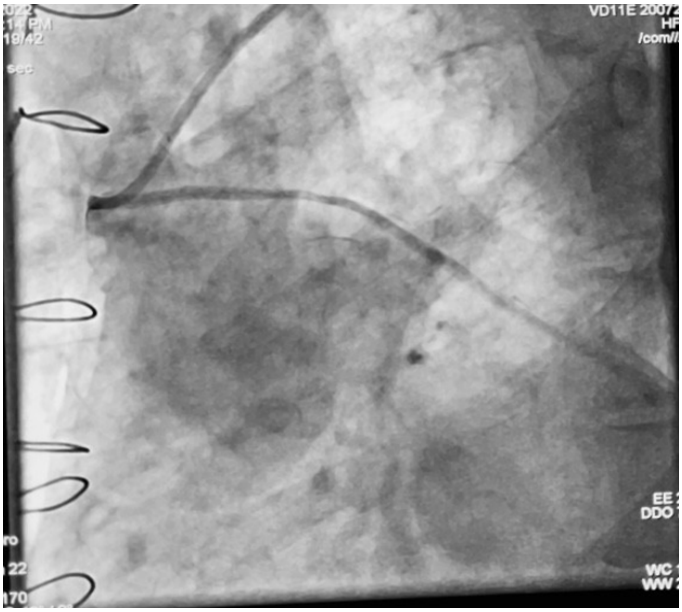

Figure 7: Post Ballooning (PRE Dilation) Flow

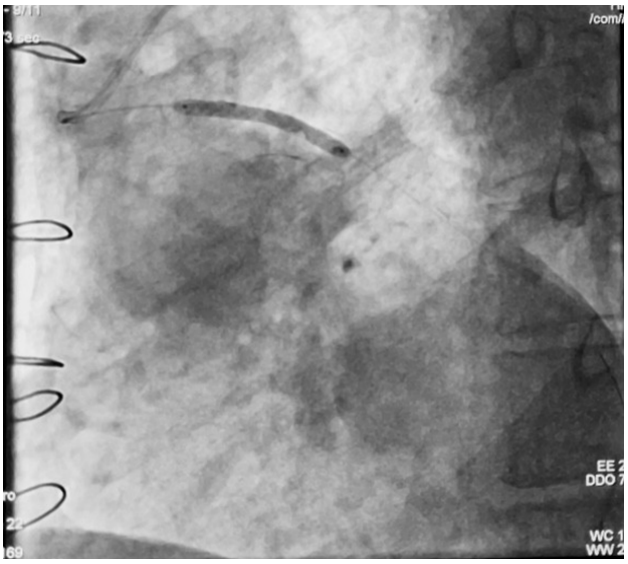

Figure 8: DES Placement at Mid SVG

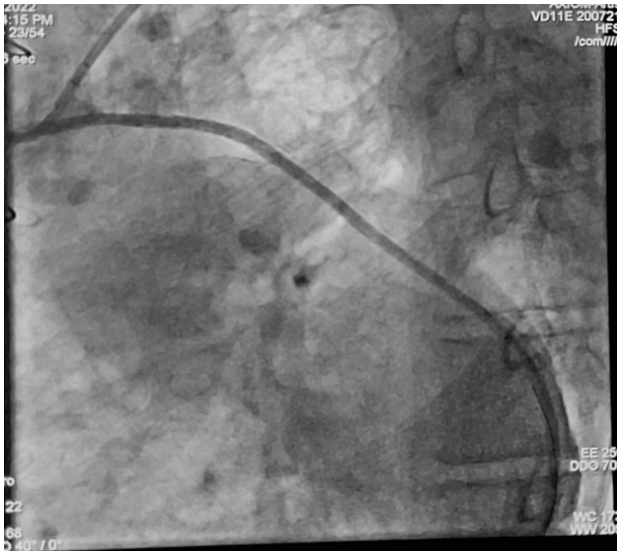

Figure 9: Post Stenting Flow of SVG

Post procedural patient was kept in ICU for observation and then discharged 2 days later on dual antiplatelet therapy with aspirin and clopidogrel, an Angiotensin converting enzyme inhibitor, a beta blocker and a statin [11] (**Table 1**).

Table 1: Segment values

Vessel/ segment %	Ostial	Proximal	Mid	Distal	Timi flow
RCA	90%	95%	90%	50%	Ii
LM	Normal	Normal	Normal	Normal	Iii
LAD	ECTASIA	ECTASIA	90%	Total	II
D1	10%	10%	MADINA 1,1,1		II
LCX	ECTASIA	20%	40%	ECTASIA	II
OM2	90%	70%	95%	TIMI II	I
RAMUS		**SMALL BRANCH**			
LIMA	Patent	Patent	Patent	Patent	Iii
SVG	Patent	Patent	90%	Patent	Iii
LIMA	Patent	Patent	Patent	Patent	Iii
SVG	Patent	Patent	90%	Patent	Iii

DISCUSSION

Dynamic segmental compression of SVG rare, yet clinically relevant angiographic finding [12]. Compared with native coronary artery disease (CAD), vein graft lesions are frequently associated with considerable plaque burden and intracoronary thrombus, which increases the risk of peri-interventional adverse events. In addition, a unique pathophysiology and histomorphology of SVG lesions with typically friable degenerated plaques result in a different vessel response to endoluminal interventions and a high risk of debris embolisation. Finally, patients with diseased SVGs frequently have additional conditions including advanced age, more extensive atherosclerosis and reduced left ventricular function, which predicts a worse outcome after peri-interventional complications compared with 'healthier' patients; thus, optimised approaches that minimise complications and maximise the efficacy in patients undergoing vein graft PCI are

of specific importance. Saphenous venous bypass grafts are often used as conduits for CABG. However, ten years after CABG only 60% of venous grafts remain patent [13].

CONCLUSION

Saphenous vein graft patency continues to be a significant problem after CABG because only half of them are free of significant stenosis. For revascularization after CABG, both surgical and percutaneous methods have limitations. Percutaneous coronary intervention (PCI) is recommended only if feasible, mainly in patients with co-morbidities, left ventricular dysfunction, lack of available saphenous veins, and in the elderly. SVG stenting is an attractive alternative in patients without PCI option in the native vessels. A greater interval between surgery and SVG PCI reduces the technical success and is associated with a higher rate of early complications and reduced long term patency.

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