

Original Article

Impact of Drug-Eluting Stent Expansion on Saphenous Vein Graft Percutaneous Intervention

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ABSTRACT

Background: We intended to evaluate the effects of stent expansion in percutaneous coronary intervention (PCI) on saphenous vein graft (SVG) lesions and compare over-expansion and under-expansion between SVG stents in the case of major adverse cardiac events (MACE).

Methods: Totally, 196 SVG lesions were treated with drug-eluting stents. The ratio of the stent diameter to the diameter of the normal part of the SVG (which was without lesions and considered the reference part of the SVG) was measured by quantitative coronary angiography. Subsequently, the patients were divided into 3 groups: Group I (<0.90 expansion: undersized stents), Group II (0.90–1.0 expansion: normal-sized stents), and Group III (>1.0: oversized stents). MACE rates during PCI, hospitalization length, and follow-up findings were compared between the groups. Additionally, the effects of the embolic protection device (EPD) on MACE were assessed.

Results: Oversized stenting was associated with increased cardiac enzymes ($P=0.035$) during hospitalization but was not associated with more MACE or restenosis on follow-up. Statistical analysis demonstrated nonsignificant more revascularization in the oversized group mainly due to unknown vessel revascularization and non-target vessel revascularization ($P=0.167$ and $P=0.108$, respectively). There were no differences in other MACE outcomes. The EPD was used in 25% of the patients. By comparison with the group without the EPD, there was no decrease in MACE components except a higher incidence of heart failure in the EPD group ($P=0.03$).

Conclusions: Aggressive stent expansion in SVG lesions resulted in higher myocardial injury; and unlike native arteries, there was no improvement in target vessel revascularization rates at follow-up. (*Iranian Heart Journal 2022; 23(1): 85-94*)

KEYWORDS: Saphenous vein graft, PCI, Stent expansion

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Saphenous vein grafts (SVGs) are typically used for multivessel and complex coronary artery disease during coronary artery bypass graft surgery (CABG). SVGs are, however, susceptible to both degeneration and occlusion, resulting in low long-term patency compared with arterial grafts.¹ The effectiveness of CABG relies on the long-term patency of arterial and venous grafts.² Atherosclerotic changes in aortocoronary SVGs constitute one of the most common drawbacks after CABG, with over 50% of SVGs becoming occluded at 10 years after the surgery and 25% showing significant stenosis at angiographic follow-ups.³ Late graft failure may result from early localized stenosis, most likely associated with improper surgical techniques; nonetheless, atherosclerosis appears to be the most common cause.⁴ SVG failure is far more problematic than the progression of native coronary artery disease in these patients and is also the main rationale for repeat interventions either by redo surgery or percutaneous coronary intervention (PCI). Compared with native vessel coronary artery disease, vein graft lesions are more frequently plagued with intracoronary thrombotic lesions, and there is a substantial plaque burden, which can increase the risk of periprocedural adverse events.⁵ Because of the friable, degenerated atheromatous and thrombotic debris, SVG PCI is associated with higher rates of periprocedural myocardial infarction, in-hospital mortality, no-reflow phenomenon,⁶⁻⁸ restenosis, and distal embolization than PCI on native coronary arteries.⁹⁻¹¹ Distal embolization is one of the most frequent complications of PCI on SVGs, which leads to enzyme elevation and in some circumstances myocardial infarction.^{10, 12, 13} PCI on native coronary arteries is often preferable to PCI on SVGs and is accompanied by higher complications.¹⁴

In some circumstances, it is not feasible to perform PCI on native arteries, and PCI on SVGs is the last resort for the heart team. Several strategies are used to minimize distal embolization and the no-reflow phenomenon in PCI on SVGs. They include distal embolic protection device (EPD) use, vasodilator administration, direct stenting, and recently debated undersized stents.¹⁵⁻¹⁸ Larger postprocedural lumen dimensions proved by intravascular ultrasound^{19, 20} or quantitative coronary angiography^{21, 22} are associated with less late restenosis in native coronary arteries. Nevertheless, in the case of PCI on SVGs, previous studies have reported that oversizing the stent is associated with a higher incidence of myocardial infarction and higher levels of myocardial damage.^{18, 23} In this study, we sought to assess major adverse cardiac events (MACE), defined as cardiovascular mortality, nonfatal myocardial infarction, cerebrovascular accident (CVA), and target lesion revascularization, during the PCI procedure, hospitalization length, and follow-up in 3 groups of patients with SVG oversized, undersized, and normal-sized stent deployment.

METHODS

For the enrollment of eligible patients, the PCI reports of patients who underwent PCI on SVGs were reviewed. Primarily, 263 patients with a history of CABG and PCI with drug-eluting stents on their SVGs were recruited as the study population, and they were followed up at Rajaie Cardiovascular Medical and Research Center for between 6 and 75 months from March 2013 through April 2019.

For the purposes of level data measurement and report of genuine results, patients with PCI on anastomosis sites (n=26); PCI on native vessels via SVGs (n=25); PCI on 1 SVG lesion with multiple stents, which was

defined as at least 2 stents in 1 SVG (n=4); and more than 1 SVG PCI procedure such as patients with simultaneous PCI on native coronaries and SVGs in 1 session (n=2) were excluded. Additionally, at the final stage, 10 more patients were excluded because of their inaccessibility for follow-up contact. The patients' selection process was based on the following inclusion criteria: patients not amenable for redo CABG, patients not amenable for PCI on native vessels, and patients for whom SVG PCI was considered more beneficial than native vessel PCI.²⁴

The ratio of the stent diameter (after expansion) to the diameter of the normal part of the SVG (the part without any lesion and considered the reference part of the SVG) measured by quantitative coronary angiography was regarded as a scale to compare the stent size and its expansion ratio in each patient (the stent expansion size/normal reference part of SVGs). The size of the reference part of each SVG was recorded before stent expansion. Afterward, the study population was divided into 3 groups according to previously defined ratios to compare the size and expansion of stents: Group I (<0.90 expansion: undersized stents, n=45 [22.9%]), Group II (0.90–1.0 expansion: normal-sized stents, n=43 [1.9%]), and Group III (>1.0 expansion: oversized stents, n=108 [55.1%]).

The thrombolysis in myocardial infarction (TIMI) grade flow and MACE were assessed in 3 separate phases: during PCI, during hospitalization, and during follow-up. The SPSS software for Windows, version 22, (IBM Corp) was used for all the statistical analyses. Continuous variables were presented as the mean \pm the standard

deviation (SD) and compared via 1-way ANOVA models. Categorical variables were presented as counts (%) and compared between the groups by using the Pearson χ^2 (or the Fisher exact) test. A *P* value of less than 0.05 was considered statistically significant.

The study protocol and conduct were approved by the institutional ethics committee.

RESULTS

The baseline characteristics and frequencies of cardiovascular risk factors are summarized in Table 1. Totally, 196 patients were eligible to be followed up. The mean age of the patients was 64.4 ± 8.4 years. Forty-five patients (22.9%) were in the undersized group (Group I), 43 (21.9%) in the normal-sized group (Group II), and 113 (57.6%) in the oversized group (Group III). Systemic hypertension was reported more frequently in Group III, with 88 patients (81.4%) ($P=0.001$).

As is demonstrated in Table 1, more patients presented with non-ST-segment-elevation myocardial infarction (NSTEMI), which was diagnosed in 67 patients (34.1%). The mean graft age was 12.09 ± 5.2 years. No significant differences were found in terms of mean age, clinical presentation, graft age, or the use of the distal EPD between the 3 groups. Out of the 196 patients, 3 patients presented with stent thrombosis (1 patient in Group I and 2 patients in Group II), and in-stent restenosis was reported in 10 patients (2 patients in Group I and 8 patients in Group II), which was statistically meaningful ($P<0.001$).

Table 1. Baseline clinical characteristics and cardiac risk factors

Ratio of the Stent Diameter to the Average QCA Reference Lumen Diameter		Total	P value			
Groups	<0.89, Group I (n=45 pts, 22.8%)	0.9–1.0, Group II (n=43 pts, 22.3%)	>1.0, Group III (n=108 pts, 54.9%)	196 pts		
Age (y)	66.7 ± 9.12	66.91 ± 8.95	67.98 ± 7.9	67.47 ± 8.4	0.385	
Sex	Male	39 (86.6%)	39 (90.6%)	95 (87.9%)	173 (88.2%)	0.833
Clinical Presentation	SIHD	11 (24.4%)	13 (30.2%)	35 (32.4%)	59 (30.1%)	0.753
	UA	14 (31.1%)	15 (34.8%)	27 (25%)	56 (28.5%)	
	NSTEMI	17 (37.7%)	11 (25.5%)	39 (36.1%)	67 (34.1%)	
	STEMI	3 (6.6%)	4 (9.3%)	7 (6.4%)	14 (7.1%)	
Systemic hypertension	35 (77.7%)	23 (53.4%)	88 (81.4%)	146 (74.4%)	0.001	
Diabetes mellitus	21 (46.6%)	21 (48.8%)	60 (55.5%)	102 (52.0%)	0.540	
Hypercholesterolemia	24 (53.3%)	23 (53.4%)	74 (68.5%)	121 (61.7%)	0.096	
Smoking	17 (37.7%)	13 (30.2%)	35 (32.4%)	65 (33.1%)	0.731	
Family history of CAD	9 (20%)	12 (27.9%)	25 (23.1%)	46 (23.4%)	0.677	
Graft age (y)	11.59 ± 5.49	12.84 ± 4.48	11.99 ± 5.1	(12.09 ± 5.2)	0.341	
In-stent restenosis	2 (4.4%)	8 (9.3%)	0	10 (5.1%)	<0.001	
Ejection fraction (%)	36.8 ± 10.35	34.9 ± 10.12	38.14 ± 10.22	(37.12 ± 10.22)	0.672	
Distal embolic protection device	14 (31.1%)	10 (23.2%)	30 (27.7%)	54 (27.5%)	0.710	
Follow-up duration (mon)	23.35 ± 14.74	34.6 ± 13.36	29.53 ± 15.95	29.16 ± 14.81	0.902	

CAD, Coronary artery disease; CVA, Cerebrovascular accident; HF, Heart failure; NSTEMI, Non-ST-segment-elevation myocardial infarction; SIHD, Stable ischemic heart disease; UA, Unstable angina, QCA, Quantitative coronary angiography

Table 2. Coronary angiographic findings and procedural results

Groups	<0.89, Group I	0.9–1.0, Group II	>1.0, Group III	Total	P value
Stent diameter (mm)	3.37 ± 0.38	3.29 ± 0.53	3.59 ± 0.36	3.41 ± 0.42	0.347
Reference vessel diameter	4.33 ± 0.85	3.40 ± 0.78	2.93 ± 0.93	3.55 ± 0.8	0.759
Stent length (mm)	28.46 ± 0.41	26.78 ± 0.39	27.72 ± 0.46	27.65 ± 0.44	0.822
Predilation (n.)	15 (33.3%)	14 (32.5%)	32 (29.6%)	61 (31.1%)	0.921
Postdilation (n.)	14 (31.1%)	9 (20.9%)	16 (14.8%)	39 (19.8%)	0.735

As was previously explained, we assessed the study results in 3 different chronological phases, as follows:

1- During the Procedure

In the evaluation of the TIMI flow grade just after the procedure, there was no change in this variable in 75.2% of the study population. Still, deterioration and improvement in the TIMI flow grade were detected in 13.1% and 11.7% of the patients after PCI, respectively. There was no significant difference between these groups ($P=0.116$). No significant differences were observed in the types of stents and filters, as

well as the diameter and length of the stents, between the 3 groups (Table 2).

Predilation was done in 61 patients (31.1%) and postdilation in 39 (19.8%), without any meaningful difference between the groups ($P=0.921$ and $P=0.735$, respectively). During the procedure, no MACE occurred in any patient.

2- Hospitalization

MACE during hospitalization is summarized in Table 3. The rise in the plasma cardiac troponin level (an increase in the troponin level ≥ 5 times from the baseline according to the fourth definition of myocardial

infarction)²⁴ was significantly higher in the oversized group ($P=0.035$).

There was only 1 case of in-hospital stent thrombosis in the undersized group, in which the patient presented to the hospital with STEMI several hours after PCI. Unfortunately, the patient developed cardiopulmonary arrest immediately after presentation, and despite resuscitation, he passed away.

There were also 2 cases of in-hospital cardiovascular deaths: 1 in the undersized group and 1 in the normal-sized group. One of them presented with stent thrombosis, and the other one with STEMI and cardiogenic shock due to left ventricular failure, which resulted in multiorgan failure and cardiogenic shock.

Heart failure was reported in 7 patients (3.4%): 1 in the undersized group, 3 in the normal-sized group, and 3 in the oversized group; there was no significant difference between the 3 groups ($P=0.733$). There was no report of non-cardiovascular death or CVA in any group.

3- Follow-up

MACE after discharge is summarized in Table 4. No significant differences concerning MACE were found between the 3 groups during the follow-up period. The difference regarding the rate of composite MACE (cardiovascular death, myocardial infarction, heart failure, and CVA) was statistically insignificant between the 3 groups ($P=0.653$): 12 patients (26.6%) in Group I, 14 (32.5%) in Group II, and 26 (24.0%) in Group III.

During the follow-up, 11 patients (5.6%) died of cardiovascular causes: 3 (6.7%) in the undersized group, 3 (7%) in the normal-sized group, and 5 (4.6%) in the oversized group. The difference between the groups in this regard was not statistically significant ($P=0.802$).

After PCI, 162 patients (82.7%) reported an improvement in their functional class according to the New York Heart Association (NYHA) functional class scale: 35 patients in the undersized group (77.8%), 38 in the normal-sized group (88.4%), and 89 in the oversized category (82.4%). These differences were not statistically significant ($P=0.421$) (Table 4).

Twenty-seven (13.8%) events of myocardial infarction (a composite of STEMI and NSTEMI) were reported in the patients' follow-up. Although myocardial infarction was reported more frequently in the normal-sized group, as was expected, the differences were statistically insignificant ($P=0.417$). Five patients (2.6%) suffered CVA, but the difference between the groups in this regard was nonsignificant ($P=0.455$).

Seventy-eight (39.8%) out of the 196 patients, who participated in the follow-up, were rehospitalized between 1 and 4 times (mean =1.37 times): 16 patients (35.5%) in the undersized group, 18 (41.8%) in the normal-sized group, and 44 (40.7%) in the oversized group. This difference failed to constitute statistical significance between the 3 groups ($P=0.797$). The most common cause of admission was unstable angina (43.6%) and NSTEMI (30.8%), and the difference between the groups was not meaningful ($P=0.751$).

During the follow-up period, 58 patients (29.6%) underwent coronary angiography. The undersized group had more angiographic procedures (35.5%), but this difference was not statistically significant ($P=0.567$) (Table 5). The statistical analyses demonstrated more revascularization procedures in the oversized group (8 [26.7%] vs 9 [30%] in the non-target vessel revascularization (non-TVR) group and 9 [30%] in the unknown vessel revascularization group), mainly due to unknown vessel revascularization and non-TVR ($P=0.167$ and $P=0.108$, respectively).

Stented segment analysis showed in-stent restenosis in 11 patients (19%): 3 (3.3%) in the undersized group, 2 (4.6%) in the normal-sized group, and 6 (5.5%) in the oversized group, with a nonsignificant difference ($P=0.6$).

For the evaluation of the role of the EPD, the patients were divided into 2 subgroups: with the EPD and without the EPD, and MACE was compared between these 2

subgroups. The MACE endpoints were reported more frequently in the EPD group, but these differences were statistically insignificant, except for heart failure, which significantly increased after EPD use ($P=0.03$) (Table 6). Target vessel and lesion revascularization procedures were not statistically significantly different between these 2 subgroups ($P=0.182$).

Table 3. Hospitalization for major adverse cardiac events

Groups	<0.89, Group I	0.9–1.0, Group II	>1.0, Group III	Total	P value
Myocardial infarction	1 (2.2%)	0	0	1 (0.5%)	0.449
Cardiac death	1 (2.2%)	1 (2.3%)	0	2 (1.0%)	0.200
Noncardiac Death	0	0	0	0	-
Heart failure	1 (2.2%)	3 (6.9%)	3 (2.7%)	7 (3.5%)	0.733
CVA	0	0	0	-	-
Stent thrombosis	1 (2.2%)	0	0	1 (0.5%)	0.449
troponin rise >5 ×	9 (20%)	11 (25.5%)	29 (26.8%)	51 (26.0%)	0.035

CVA, Cerebrovascular accident

Table 4. MACE at the 1-year follow-up

Groups	<0.89, Group I	0.9–1.0, Group II	>1.0, Group III	Total	P value
Admission	16 (35.5%)	18 (41.9%)	44 (40.7%)	78 (39.8%)	0.797
FC Improvement	35 (77.8%)	38 (88.4%)	89 (82.4%)	162 (82.7%)	0.421
Myocardial infarction	4 (8.9%)	8 (18.6%)	15 (13.9%)	27 (13.8%)	0.417
Heart failure	3 (6.7%)	2 (4.7%)	4 (3.7%)	9 (4.6%)	0.727
CVA	2 (4.4%)	1 (2.3%)	2 (1.9%)	5 (2.6%)	0.455
Cardiac death	3 (6.7%)	3 (7%)	5 (4.6%)	11 (5.6%)	0.802
PCI -to-death time (mon)	12 ± 9.68	35 ± 26.53	19 ± 17.95	22 ± 20.66	0.390
Noncardiac death	0	0	1 (0.9%)	1 (0.5%)	>0.999

MACE, Major adverse cardiac events; CVA, Cerebrovascular accident; FC, Functional class

Table 5. Follow-up angiography

Groups	Follow-up Angiography			Total (58 pts, 29.5%)	P value
	<0.89, Group I (16 pts, 35.5%)	0.9–1.0, Group II (12 pts, 27.9%)	>1.0, Group III (30 pts, 27.7%)		
TVR	1 (6.3%)	1 (8.3%)	1 (3.3%)	3 (5.2%)	0.589
Non-TVTR	0	2 (16.7%)	9 (30%)	11 (18.9%)	0.108
CABG	0	0	1 (3.3%)	1 (1.7%)	>0.999
No revascularization	9 (56.3%)	5 (41.7%)	5 (16.7%)	19 (32.8%)	0.109
Unknown vessel revascularization	0	2 (16.7%)	8 (26.7%)	10 (17.2%)	0.167
TLR	6 (37.5%)	2 (16.7%)	6 (20%)	14 (24.1%)	0.182

CABG, Coronary artery bypass grafting; TLR, Target lesion revascularization; TVR, Target vessel revascularization

Table 6. Major adverse cardiac events with or without the EPD

Groups	With the EPD (49 pts) 25%	Without the EPD (147 pts) 75%	Total (196 pts)	P value
Re-admission	23 (46.9%)	55 (37.4%)	78 (39.8%)	0.238
FC improvement	40 (81.6%)	122 (83%)	162 (82.7%)	0.828
Myocardial infarction	10 (20.4%)	17(11.6%)	27 (13.8%)	0.120
Cardiac death	3 (6.1%)	8 (5.4%)	11(5.6%)	0.885
Heart failure	5 (10.2%)	4 (2.7%)	9 (4.6%)	0.030
CVA	2 (4.1%)	3 (2%)	5 (2.6%)	0.433
PCI-to-death time (mon)	12	35	23.5	0.390
Noncardiac death	1 (2.0%)	0	1 (0.5%)	0.698

CVA, Cerebrovascular accident; EPD, Embolic protection device; FC, Functional class

DISCUSSION

Aortocoronary interventions on SVGs are affected by the chronic degenerative process, regardless of the bypassed arterial territory. Indeed, about 10% of SVGs are occluded before discharge or within the first 30 days, and in 10 years approximately 50% are occluded.²⁴⁻²⁷ In the first year after CABG, 25% of patients experience stable angina pectoris.²⁸ The best predictors of 30-day MACE of SVG interventions are SVG degeneration and plaque volume.²⁹

Previous studies on native coronary arteries have demonstrated that larger post-PCI lumen dimensions are associated with lower rates of restenosis.^{22, 29-33} A previous investigation reported that oversizing was of benefit with less future TVR on native coronary arteries, which is in contrast to our finding as regards SVG interventions. However, a study concluded that aggressive stent expansion in native coronary arteries could lead to increased rates of in-hospital myocardial infarction.²³

According to our findings, the oversizing of the stent in SVG interventions was associated with a higher troponin rise, indicating more myocardial injury ($P=0.035$); still, we found no significant difference concerning MACE during hospitalization or follow-up. A prior investigation reported the outcomes of SVG interventions with drug-eluting stents in 3

groups based on intravascular ultrasound reference lumen diameters, as was the case with our patient group allocations. Additionally, an elevation in CK-MB of greater than 3 times the normal level was reported in 6%, 9%, and 19%, of the 3 groups, respectively ($P=0.03$), without any difference in clinical events.¹⁸

Some studies have concluded that greater postprocedural lumen dimensions might be associated with increased myocardial infarction without decreasing the rate of restenosis.^{23, 34} Previous research has also indicated that increased rates of cardiac enzymes after successful PCI on SVGs could be related to increased mortality.¹²

We had a higher rate of revascularization in the oversized group, mainly due to unknown vessel revascularization and non-TVR ($P=0.167$ and $P=0.108$, respectively). A prior study concluded that oversizing was of no benefit in the case of TVR.³⁵ It is now prudent to size balloons and stents no more than the vessel size with a view to preventing perforation, especially in older vein grafts.³⁶ These findings are in favor of performing less aggressive PCI on SVG lesions in order to diminish the rate of target vessel and target lesion revascularization.

The other unexpected finding of the present study was that EPD use during SVG interventions was not associated with a reduction in MACE, myocardial infarction, or re-hospitalization, although known

studies such as SAFER have proven this benefit.³⁷⁻³⁹ In our study, 25% of the graft procedures were performed with the aid of the EPD. Only 5% of the patients in the ISAR-CABG trial underwent interventions with the aid of the EPD and their MACE rate was low (4%).⁴⁰ We believe that our rate is in consequence of our small study population and the number of the EPD used. We opted to choose drug-eluting stents in our patients' PCI procedures since they are workhorse stents in any catheterization laboratory. In contrast, the latest meta-analysis in this regard showed no significant differences between drug-eluting stents and bare-metal stents in the long-term incidence of target lesion revascularization, TVR, stent thrombosis, MACE, and all-cause mortality.⁴¹ Future trials are needed to prove the benefits of the underexpansion of graft stenting.

CONCLUSIONS

SVG interventions are gaining more favor by the year, and to diminish their undesirable complications, and in light of the findings of the present study, we recommend the use of stents and balloons the same size as the reference vessel or smaller.

Disclosures

The authors hereby declare that they received no other contributions or funding.

REFERENCES

1. Widimsky P, Straka Z, Stros P, Jirasek K, Dvorak J, Votava J, et al. One-year coronary bypass graft patency: a randomized comparison between off-pump and on-pump surgery angiographic results of the PRAGUE-4 trial. *Circulation*. 2004; 110(22):3418-23.
2. Goldman S, Zadina K, Moritz T, Ovitt T, Sethi G, Copeland JG, et al. Long-term patency of saphenous vein and left internal mammary artery grafts after coronary artery bypass surgery: results from a Department of Veterans Affairs Cooperative Study. *Journal of the American College of Cardiology*. 2004; 44(11):2149-56.
3. Beijk M, Harskamp R. Treatment of coronary artery bypass graft failure. *Artery Bypass: IntechOpen*; 2013.
4. Campeau L, Enjalbert M, Lespérance J, Vaislic C, Grondin C, Bourassa M. Atherosclerosis and late closure of aortocoronary saphenous vein grafts: sequential angiographic studies at 2 weeks, 1 year, 5 to 7 years, and 10 to 12 years after surgery. *Circulation*. 1983;68(3 Pt 2):III-7.
5. McKavanagh P, Yanagawa B, Zawadowski G, Cheema A. Management and prevention of saphenous vein graft failure: a review. *Cardiology and therapy*. 2017; 6(2):203-23.
6. Kaplan BM, Benzuly KH, Kinn JW, Bowers TR, Tilli FV, Grines CL, et al. Treatment of no-reflow in degenerated saphenous vein graft interventions: comparison of intracoronary verapamil and nitroglycerin. *Catheterization and cardiovascular diagnosis*. 1996; 39(2):113-8.
7. Baim DS, Carrozza Jr JP. Editorial comment: Understanding the "no-reflow" problem. *Catheterization and cardiovascular diagnosis*. 1996; 39(1):7-8.
8. Morishima I, Sone T, Mokuno S, Taga S, Shimauchi A, Oki Y, et al. Clinical significance of no-reflow phenomenon observed on angiography after successful treatment of acute myocardial infarction with percutaneous transluminal coronary angioplasty. *American heart journal*. 1995; 130(2):239-43.
9. Lee MS, Park S-J, Kandzari DE, Kirtane AJ, Fearon WF, Brilakis ES, et al. Saphenous vein graft intervention. *JACC: Cardiovascular Interventions*. 2011; 4(8):831-43.
10. Lefkovits J, Holmes DR, Califf RM, Safian RD, Pieper K, Keeler G, et al. Predictors and sequelae of distal embolization during saphenous vein graft intervention from the CAVEAT-II trial. *Circulation*. 1995; 92(4):734-40.

11. de Feyter PJ, van Suylen R-J, de Jaegere PP, Topol EJ, Serruys PW. Balloon angioplasty for the treatment of lesions in saphenous vein bypass grafts. *Journal of the American College of Cardiology*. 1993;21(7):1539-49.
12. Hong MK, Mehran R, Dangas G, Mintz GS, Lansky AJ, Pichard AD, et al. Creatine kinase-MB enzyme elevation following successful saphenous vein graft intervention is associated with late mortality. *Circulation*. 1999; 100(24):2400-5.
13. Hong MK, Mehran R, Dangas G, Mintz GS, Lansky A, Kent KM, et al. Are we making progress with percutaneous saphenous vein graft treatment?: A comparison of 1990 to 1994 and 1995 to 1998 results. *Journal of the American College of Cardiology*. 2001; 38(1):150-4.
14. Nguyen T, Pham L, Cheem TH, Douglas JS, Hermiller J, Grines C. Approach to the patient with prior bypass surgery. *Journal of interventional cardiology*. 2004;17(5):339-46.
15. Baim DS, Wahr D, George B, Leon MB, Greenberg J, Cutlip DE, et al. Randomized trial of a distal embolic protection device during percutaneous intervention of saphenous vein aorto-coronary bypass grafts. *Circulation*. 2002; 105(11):1285-90.
16. Grygier M, Araszkiwicz A, Lesiak M, Grajek S. Intracoronary adenosine administered during aortocoronary vein graft interventions may reduce the incidence of no-reflow phenomenon. A pilot randomised trial. *Kardiol Pol*. 2014; 72(2):126-33.
17. Mauri L, Cox D, Hermiller J, Massaro J, Wahr J, Tay SW, et al. The PROXIMAL trial: proximal protection during saphenous vein graft intervention using the Proxis Embolic Protection System: a randomized, prospective, multicenter clinical trial. *Journal of the American College of Cardiology*. 2007; 50(15):1442-9.
18. Hong YJ, Pichard AD, Mintz GS, Kim SW, Lee SY, Kim SY, et al. Outcome of undersized drug-eluting stents for percutaneous coronary intervention of saphenous vein graft lesions. *The American journal of cardiology*. 2010; 105(2):179-85.
19. Verbin C, White R, Donayre C, Kopchok G. The ideal guidance imaging system for endovascular interventions. *The Journal of cardiovascular surgery*. 1996; 37(3 Suppl 1):5-9.
20. Elezi S, Kastrati A, Neumann F-J, Hadamitzky M, Dirschinger J, Schömig A. Vessel size and long-term outcome after coronary stent placement. *Circulation*. 1998; 98(18):1875-80.
21. Akiyama T, Moussa I, Reimers B, Ferraro M, Kobayashi Y, Blengino S, et al. Angiographic and clinical outcome following coronary stenting of small vessels: a comparison with coronary stenting of large vessels. *Journal of the American College of Cardiology*. 1998; 32(6):1610-8.
22. Hsieh IC, Chien CC, Chang HJ, Chern MS, Hung KC, Lin FC, et al. Acute and long-term outcomes of stenting in coronary vessel > 3.0 mm, 3.0–2.5 mm, and < 2.5 mm. *Catheterization and cardiovascular interventions*. 2001; 53(3):314-22.
23. Iakovou I, Dangas G, Mintz GS, Mehran R, Kobayashi Y, Aymong ED, et al. Relation of final lumen dimensions in saphenous vein grafts after stent implantation to outcome. *The American journal of cardiology*. 2004; 93(8):963-8.
24. Xenogiannis I, Tajti P, Hall AB, Alaswad K, Rinfret S, Nicholson W, Karpaliotis D, Mashayekhi K, Furkalo S, Cavalcante JL, Burke MN. Update on cardiac catheterization in patients with prior coronary artery bypass graft surgery. *JACC: Cardiovascular Interventions*. 2019 Aug 14.
25. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). *Journal of the American College of Cardiology*. 2018; 72(18):2231-64.
26. Fitzgibbon GM, Kafka HP, Leach AJ, Keon WJ, Hooper GD, Burton JR. Coronary bypass graft fate and patient outcome: angiographic follow-up of 5,065 grafts related to survival and reoperation in 1,388 patients during 25 years. *J Am Coll Cardiol*. 1996; 28:616-26.

27. Tatoulis J, Buxton BF, Fuller JA. Patencies of 2127 arterial to coronary conduits over 15 years. *Ann Thorac Surg.* 2004; 77:93-101.
28. Cameron AA, Davis KB, Rogers WJ. Recurrence of angina after coronary artery bypass surgery: predictors and prognosis (CASS Registry). *Coronary Artery Surgery Study. Journal of the American College of Cardiology.* 1995; 26:895-9.
29. Hsieh IC, Chien CC, Chang HJ, Chern MS, Hung KC, Lin FC, et al. Acute and long-term outcomes of stenting in coronary vessel > 3.0 mm, 3.0–2.5 mm, and < 2.5 mm. *Catheterization and cardiovascular interventions.* 2001; 53(3):314-22.
30. Coolong A, Baim DS, Kuntz RE, O'Malley AJ, Marulkar S, Cutlip DE, Popma JJ, Mauri L. CLINICAL PERSPECTIVE. *Circulation.* 2008 Feb 12; 117(6):790-7.
31. Iakovou I, Dangas G, Mintz GS, Mehran R, Kobayashi Y, Aymong ED, et al. Relation of final lumen dimensions in saphenous vein grafts after stent implantation to outcome. *The American journal of cardiology.* 2004; 93(8):963-8.
32. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). *Journal of the American College of Cardiology.* 2018; 72(18):2231-64.
33. Pepine C, Holmes Jr D. Coronary artery stents. *American College of Cardiology. Journal of the American College of Cardiology.* 1996; 28(3):782.
34. Keeley EC, Velez CA, O'Neill WW, Safian RD. Long-term clinical outcome and predictors of major adverse cardiac events after percutaneous interventions on saphenous vein grafts. *Journal of the American College of Cardiology.* 2001; 38(3):659-65.
35. Iavouvau I, Dangas G, Mintz GS, et al. Relation of final lumen dimensions in saphenous vein grafts after stent implantation to outcome. *Am I Cardiol.* 2004; 93:963-968.
36. Topol EJ, Teirstein PS. *Bypass Graft Intervention. Textbook of interventional cardiology, 8th edition.* Philadelphia, Elsevier, 2020, 447-460.
37. Paul TK, Bhatheja S, Panchal HB, Zheng S, Banerjee S, Rao SV, et al. Outcomes of saphenous vein graft intervention with and without embolic protection device: a comprehensive review and meta-analysis. *Circulation: Cardiovascular Interventions.* 2017; 10(12):e005538.
38. Brennan JM, Al-Hejily W, Dai D, Shaw RE, Triletskaya M, Rao SV, et al. Three-year outcomes associated with embolic protection in saphenous vein graft intervention: results in 49 325 senior patients in the Medicare-linked National Cardiovascular Data Registry CathPCI Registry. *Circulation: Cardiovascular Interventions.* 2015; 8(3):e001403.
39. Baim DS, Wahr D, George B, et al. Randomized Trial of a Distal Embolic Protection Device During Percutaneous Intervention of Saphenous Vein Aorto-Coronary Bypass Grafts. *Circulation.* 2002; 105:1285–1290
40. Mehilli J, Pache J, Abdel-Wahab M, Schulz S, Byrne RA, Tiroch K, Hausleiter J, Seyfarth M, Ott I, Ibrahim T, Fusaro M. Drug-eluting versus bare-metal stents in saphenous vein graft lesions (ISAR-CABG): a randomised controlled superiority trial. *The Lancet.* 2011 Sep 17; 378(9796):1071-8.
41. Kheiri B, Osman M, Abdalla A, Ahmed S, Bachuwa G, Hassan M. The short- and long-term outcomes of percutaneous intervention with drug-eluting stent vs bare-metal stent in saphenous vein graft disease: An updated meta-analysis of all randomized clinical trials. *Clin Cardiol.* 2018; 41(5):685-692. doi:10.1002/clc.22908