e-CliniC 2024; Vol. 12, No. 2: 150-156
DOI: https://doi.org/10.35790/ecl.v12i2.52514
URL Homepage: https://ejournal.unsrat.ac.id/index.php/eclinic

# Percutaneous Coronary Intervention in a Saphenous Vein Graft Failure: A Case Report

**Edmond L. Jim**

Department of Cardiology and Vascular Medicine, Universitas Sam Ratulangi, Manado, Indonesia
Email: edmondleonard@ymail.com
*Received: November 13, 2023; Accepted: December 24, 2023; Published online: December 27, 2023*

**Abstract:** Recent published data from the Society of Thoracic Surgery (STS) national database displayed that among patients aged >65 years old who survived following an initial coronary artery bypass graft (CABG) operation, rates of repeat revascularization at 1, 5, 10, and 18 years were 2%, 8%, 16%, and 25%, respectively. The most preferred mode of revascularization was percutaneous coronary intervention (PCI). While remain the most frequently used conduits beside left internal mammary artery (LIMA), saphenous vein grafts (SVGs) have high failure rates. Percutaneous coronary intervention in SVGs is associated with an increased risk of distal coronary embolization, commonly resulting in periprocedural MI. We reported a 79-year-old male patient admitted due to progressive symptoms of chronic coronary syndrome. The patient had a history of hypertension and a CABG surgery in 2012. Electrocardiogram showed ischemia and old myocardial infarction (OMI) in inferior leads as well as poor R wave progression in the precordial leads. Transthoracic echocardiogram displayed a normal ejection fraction but impaired left ventricular (LV) diastolic relaxation. Diagnostic coronary angiography revealed a 70% tubular stenosis in proximal part of SVG supplying posterior descending artery, patent LIMA graft to left anterior descending artery, and complex lesion on native vessel (chronic total occlusion in mid right coronary artery). Implantation of drug eluting stent in SVG-posterior descending artery was performed. Direct-stenting PCI technique using undersized-stent was done to prevent any complications. In conclusion, despite the high risk of complications, vein graft intervention was successfully performed without any complications. Since the high rates of in-stent restenosis following PCI in SVGs, long-term dual antiplatelet therapy should be commenced in this patient.
**Keywords**: saphenous vein graft; vein graft intervention; percutaneous coronary intervention

## INTRODUCTION

Coronary artery bypass grafting (CABG) is a commonly performed surgical procedure whose efficacy has been enhanced using arterial grafts, off-bypass procedures, and minimally invasive surgical techniques.[1] Various methods have been developed to increase graft patency from CABG procedures, specifically by prescribing antiplatelet agents and lipid-lowering drugs. However, myocardial ischemic syndrome occurs in 3-5% of patients immediately after surgery, and recurrent ischemic symptoms occur in 4-8% of patients following CABG procedures annually. Saphenous vein graft (SVG) failure is the most common cause, up to 40% during the first year, with 1-2% of grafts experiencing occlusion every year, in 1-5 years after surgery and 5% every year after 5 years of surgery. Advances in surgical techniques and pharmacotherapies have improved the mid- and long-term effects of SVG patency, with recent studies reporting arterial graft patency at 5 years and SVG graft at 8 years as high as 91%. Nevertheless, 13% of patients undergoing CABG require repeat revascularization within 10 years, 18% of all PCI procedures are performed in patients with a history of CABG, and 6% of all PCIs are performed in SVG.[2] With the increased periprocedural risk of myocardial infarction associated with SVG intervention, PCI of the native vessel is usually preferred over a degenerating SVG.[3,4] Whenever native vessel intervention is not possible, the clinical benefit of a high-risk SVG intervention must be weighed against the risk of morbidity and mortality if surgical revascularization is performed again (risk increases 2 to 4 times compared with primary intervention).[1,5]

## CASE REPORT

A 79-year-old male patient came to the cardiac outpatient clinic at Prof. Dr. R. D. Kandou Hospital Manado due to progressive anginal pain and chest discomfort. The symptoms had been felt since the last one year. Chest pain sometimes felt like it was full in the chest, uncomfortable, sometimes it felt like being crushed by a heavy object. Chest pain appeared to come and go, appearing especially during activities such as walking long distances or climbing stairs. The duration of the pain lasted for 5-10 minutes, the pain was reduced by resting or sitting or by taking medication under the tongue. The patient denied complaints of shortness of breath, fatigue, palpitations, fainting, nausea, vomiting, and cold sweat.

The patient had a history of previous heart disease and hypertension for a long time. The patient regularly took medication (acetyl salicylic acid 80 mg once a day [quaque die, qd], amlodipine 10 mg qd, atorvastatin 20 mg qd, telmisartan 80 mg qd, glyceryl trinitrate 2.5 mg twice a day [bis in die, bid], bisoprolol 2.5 mg qd.). The patient had previously undergone coronary angiography in December 2011 with normal left main (LM), left anterior descending artery (LAD) had multiple critical proximal stenosis 95%, left circumflex (LCx) had a diffuse stenosis from proximal to distal, there were 90% proximal stenosis to OM1, right coronary artery (RCA) had total occlusion in the mid after the RV branch, distally filled from LAD. Then the patient underwent CABG surgery in January 2012 in Jakarta with 5 conduits, namely, left internal mammary artery (LIMA)– LAD, SVG–posterior descending artery (PDA), SVG- posterolateral branch artery (PLB), SVG-LCx, SVG-diagonal artery 1 (D1). Echocardiogram evaluation in January 2012, was carried out with the results of normal global LV systolic function, ejection fraction (EF) 71%, global normokinetics, LV diastolic dysfunction, normal right ventricular (RV) contractility, and mild aortic regurgitation (AR). There was no family history of the disease with the same complaints.

General appearance of the patient was mildly ill and level of consciousness compos mentis. The patient's vital signs showed blood pressure 106/60 mmHg, pulse rate 67 beats/minute, respiratory rate 18 breaths/minute, oxygen saturation 98% room air, and body temperature 36.2ºC. From physical examination of the head and neck, neither the conjunctiva was anemic, nor the sclera was icteric, and the jugular venous pressure was 5+1 cm $H_2O$. Cardiac examination revealed normal heart border, single heart sounds, regular, no murmurs or gallops. Normal examination of the lungs was found. The abdomen looked flat, no organ enlargement was visible. The extremities felt warm, and there was no edema.

Laboratory examination in August 2023 showed hemoglobin level 14.4 g/dL, hematocrit 31.9%, leukocytes 6,600/mm3, platelets 166,000/mm3, urea 34 mg/dL, creatinine 0.9 mg/dL, estimated rate renal filtration (eGFR) 86.9, random blood sugar 119 mg/dL, SGOT 20 U/L, SGPT 12 U/L, sodium serum 141 mmol/L, potassium serum 3.8 mmol/L, chloride serum 108 mmol/L, INR 1.09 seconds. Fasting laboratory examination in July 2023 showed fasting blood sugar 98 mg/dL, uric acid 6 mg/dL, total cholesterol 123 mg/dL, HDL 54 mg/dL, triglycerides 90 mg/dL, HbA1c 5.6%. Chest X-ray examination in July 2023 showed no significant abnormalities. Electrocardiogram in August 2023 displayed sinus bradycardia 48x/minute, normoaxis, inferior old myocardial infarction (OMI), with T inversion of the inferior leads, poor R wave progression in the precordial leads. Follow-up echocardiography revealed concentric LV hypertrophy, LVEF 72%, global normokinetic LV, LV diastolic dysfunction, mild aortic and mitral valve regurgitation, and normal RV contractility. Cardiac calcium examination exhibited a total Agatston score of 2934 (LM 0, LAD 1526, LCx 314, RCA 1094).

Based on the history taking, physical examination, laboratory, ECG, chest x-ray, echocardiography, and cardiac calcium score results, the patient was diagnosed with chronic coronary syndrome with Canadian Cardiovascular Society (CCS) class II, history of CABG 2012, hypertension on therapy, and hypertensive heart disease (HHD). The patient was given acetyl salicylic acid 80 mg qd, amlodipine 10 mg qd, atorvastatin 20 mg qd, telmisartan 80 mg qd, glyceryl trinitrate 2.5 mg bid, bisoprolol 2.5 mg qd. The patient was subsequently scheduled for coronary angiography and percutaneous coronary intervention.

Coronary angiography in August 2023 (on the third day of treatment) disclosed that the LIMA-LAD graft was patent, SVG-LCx was patent, SVG-PDA had 70% tubular stenosis proximally, SVG-RPL and D1 could not be cannulated (Figure 1). Percutaneous coronary intervention was accomplished by deployment of a stent on the SVG-PDA graft with the result being CAD 3VD post CABG post PCI 1 SVG-PDA stent, patent graft at LIMA-LAD and SVG-LCx. Subsequently the patient was observed at ward, and treated with double antiplatelet therapy (DAPT), adequate hydration, and left distal radial compressor release. On the following day, an evaluation of laboratory was taken with results of ureum 27 mg/dL, creatinine 0.9 mg/dL, sodium serum 139 mmol/L, potassium serum 3.2 mmol/L, and chloride serum 104 mmol/L. The patient's complaints improved, and he was planned for outpatient treatment with addition of clopidogrel 75mg qd, lansoprazole 30 mg bid, sucralfate cth II three times a day [ter in die, tid], and slow-release potassium 600 mg tid. The patient was suggested to avoid foods and drinks that might lead to poorly controlled risk factors and was educated to take medications regularly. The patient was planned to receive a long-term DAPT to minimize in-stent restenosis of stent in SVG. Apart from that, the patient was told to remain active by doing light exercise according to his abilities regularly and gradually.

## DISCUSSION

Coronary artery bypass operation is repeatedly selected in patients with diabetes mellitus, SYNTAX score >22, diffuse coronary lesions, involving bifurcation or trifurcation branches, relatively low ejection fraction, chronic total occlusion (CTO), severe calcification, multivessel disease (MVD), or involving the LM artery, tortuous blood vessels, valve abnormalities that require valve replacement surgery or the need for other concomitant heart surgeries.[1,6]

David C. Sabiston, Jr, MD was the first physician to use a saphenous vein graft (SVG) to revascularize RCA in 1962 at Johns Hopkins University.[7] Only 4 conduits proved to be effective: the SVG; the left and right internal mammary artery (LIMA and RIMA); the radial artery (RA); and the gastroepiploic artery (GEA). The left IMA had not been the graft of choice for LAD but became the preferred graft for the LAD during the 1980s, because its caliber was a good fit for the LAD diameter. In general, arterial conduits are athero-resistant, highly patent, have an endothelium that less susceptible to damage, and higher production of nitric oxide, and a low rate of vascular smooth muscle cells proliferation, differentiation, and migration.[8]

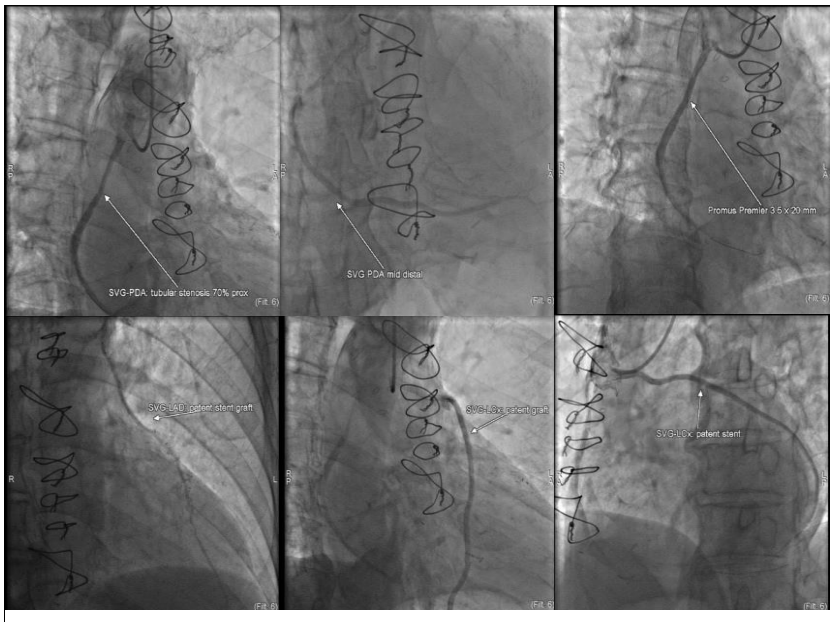


**Figure 1**. Coronary Angiography and Percutaneous Coronary Intervention in SVG-PDA graft on the third day of treatment. There was a tubular stenosis 70% in proximal SVG-PDA (coronary angiography shown in upper left-middle figure). The other grafts demonstrated patency in LIMA-LAD (lower left figure) and SVG-LCx (lower middle-right figure). Promus premier 3.5x20 mm stent was eventually deployed in SVG-PDA (upper right figure).

Despite the limitations of SVGs, they remain the most frequently used conduits in conjunction with the LIMA. Thrombosis and technical failure are the predominant mechanism of SVG failure within the first week and during the first month after CABG. The failure of SVG grafts have been shown to occlude (up to 50%) as early as 10 years after implantation as a result from intimal hyperplasia.[7,8] Intimal hyperplasia forms the ground for atherosclerosis development. Atherosclerosis of SVG progresses at fast pace and is often concentric and diffuse, with a less well-defined or even absent fibrous cap compared with native vessel atherosclerosis, and thus, is more likely to rupture.[9]

Management of therapy commences by considering whether the patient is a candidate for repeat CABG and there is a need for additional coronary revascularization. In patients who are not surgical candidates or who have only one SVG lesion, PCI is the preferred option when the SVG lesion is relatively simple (a short lesion without cellular debris and without diffuse SVG degeneration) and the coronary artery supplying the same portion is complex. If the native artery lesion is simple and the SVG lesion is complex, PCI of the native coronary artery is preferred. When both the SVG lesion and the native coronary artery lesion are complex, PCI of the native coronary artery is generally preferred first if possible. Operator experience in complex PCI is essential in selecting target vessel. Based on data from the National Cardiovascular Data Registry, target PCI of blood vessels in patients with a history of previous CABG at 0-1 year was 68% performed on natural coronary vessels, 26% on SVG grafts. At 1-5 years after CABG, 76.5% of patients underwent PCI on natural coronary vessels and 20.4% on SVG grafts. As time goes by, the trend of targeted PCI revascularization on SVG grafts is increasing, 53% underwent PCI on natural blood vessels and 44.2% underwent PCI on SVG grafts.[10]

Although a systematic review of mostly observational studies showed similar success rates between radial and femoral access, in the Radial CABG trial, radial access was associated with greater contrast use, longer duration time, and higher radiation exposure compared to femoral access. However, the advantages of radial access are better patient comfort, fewer vascular complications, and shorter hospital stays. If radial access is chosen, the left radial artery should

be used to facilitate access to the LIMA and other grafts.[11]

SVG lesions that undergo PCI have two main limitations, namely distal embolization and no-reflow phenomenon in the acute phase, and high levels of restenosis and progression of SVG lesions during control. Percutaneous intervention is preferred in patients with a history of CABG who require revascularization. Factors favoring PCI include poor graft targeting, old age, ACS, diabetes, dementia, malignancy, and collagen disorders. Meanwhile, repeat CABG is preferred in conditions of decreased ejection fraction, multiple SVG failures, old SVG stenosis (>5 years). In cases of SVG failure, PCI can be performed either in the culprit SVG or the native vessels in the relevant territory. Although there are no RCTs comparing these two strategies, observational studies have shown better short- and long-term effects of native vessel PCI compared to graft PCI.[11]

Choosing the right guiding catheter is an important first step. In the location of the vein graft to the RCA, the multipurpose catheter has the best alignment and support when performing the procedure. A vein graft originating from the left anterior surface (usually to LAD/D1 or LCx/obstuse marginal, OM), is usually preferred to a right Judkins catheter (JR) in cases of femoral access. If additional support is required, a left Amplatz catheter (AL) can be used. The 7-Fr catheter size is preferred because of more optimal visualization, facilitating stent deployment, and accommodating balloon sizes to large stents.[12]

Lesions of SVG have a high rate of restenosis, which often presents as an acute coronary syndrome.[12] The use of drug eluting stents (DES) in reducing the rate of restenosis in native coronary artery lesions significantly compared with bare metal stents (BMS). The Veterans Affairs CART study explored during long-term follow-up (>2 years), DES use was associated with lower mortality than BMS and a similar incidence of myocardial infarction.[13,14] However for long-term follow-up, two large studies (DIVA and ISAR-CABG) showed no difference between DES and BMS.[11] In this patient, PCI direct stenting with DES was performed.

The only strategy that has been tested in randomized controlled trials is the use of embolic protection devices (EPDs).[15,16] Despite vast evidence, EPDs remain underutilized (<25% of SVG treated with PCI) in daily practice. The main reasons are the high cost, length of action, and lack of expertise in using the device. It is also important to note that the use of an EPD is difficult for distal graft and tortuous lesions because it increases the risk of distal dissection and the device becoming trapped.[17] Other strategies that can be used to prevent embolism and no-reflow are administering vasodilators, direct stent placement, and undersized stents. Nicardipine is often preferred due to its long duration of action and less hypotensive effects and is often given before and after PCI. Other agents such as adenosine, nitroprusside, and verapamil have also been shown to prevent and improve no-reflow upon intragraft administration. In contrast, glycoprotein IIb/IIIa receptor inhibitors are harmful in SVG interventions and should not be used routinely in SVG PCI.[18]

Direct stenting has the same effectiveness as the use of distal embolic protection in patients undergoing SVG intervention. An undersized stent approach is defined as a stent diameter <10% of the average reference lumen diameter, was used in SVG lesions with good initial results. The "undersized stenting" strategy is a very attractive technique to use in SVG lesions when embolic protection devices are not available or cannot be used. Hong et al[4] have reported better results using slightly smaller stents in SVG lesions. In choosing a PCI strategy for the SVG graft, it was decided to use direct stenting techniques and undersized stent due to limited amenities, which have been proven to be good in preventing embolism and no-reflow events.

Optimal dosing and duration of DAPT following SVG PCI have not been well studied. Most operators recommend a bolus of 600 mg of clopidogrel prior to SVG PCI, followed by a dose of 75 mg daily for at least a year. A recently published cohort study of 411 post-SVG PCI patients who were event free at the time of clopidogrel cessation revealed a clustering of death or MI within 90 days. The cumulative 5-year event rate was lowest in patients treated with clopidogrel for more than 2 years (p<.001), which supports long-term DAPT (at least 2 years) following SVG PCI.[19]

In this case, a 79-year-old patient came with progressive chronic coronary syndrome with a history of CABG surgery in 2012 and he regularly took his medicine. He had a severe native

coronary anatomy, patent LIMA-LAD graft, stenosed SVG-PDA with relatively good eGFR and left ventricular ejection fraction function ≥50%, had a fairly low risk of death within one year, namely <5% if PCI was performed. Due to his condition, it was decided for this patient to undergo PCI on the stenosed SVG. The access location chosen was left distal radial with the advantages of better patient comfort, fewer vascular complications, shorter hospital stay and left side to facilitate access to the LIMA and other grafts. Judkins Right 3.5/6Fr catheter was used as a guiding catheter. PCI in SVG-PDA was successfully performed by implanting promus premier 3.5 x 20 mm stent using direct stenting technique and undersized stent to avoid any complications. Patient was given DAPT following PCI, planned for long term DAPT to prevent any complications in the future, particularly in-stent restenosis on the target vessel.

## CONCLUSION

In a case of graft failure, PCI is the first choice over redo CABG for late graft failure. Strategy of PCI is based on operator experience in complex PCI. Native vessel intervention is preferred to PCI of the failure graft whenever possible. Usage of distal embolic protection devices for PCI of vein graft lesions with diffuse degeneration is recommended. If considered safe, PCI with DES is preferred to BMS implantation. Administration of long DAPT should be considered in patients treated with PCI in SVGs.

### Ethics Approval

This case report has been approved by the Ethical Commission of Faculty of Medicine, Universitas Sam Ratulangi. The patient has also given informed consent to join and permit to publish the data.

### Conflict of Interest

The author declares no conflict of interest to publish this case report.

## REFERENCES

1. Fosbol EL, Zhao Y, Shahian DM, Grover FL, Edwards FH, Peterson ED. Repeat coronary revascularization after coronary artery bypass surgery in older adults. The Society of Thoracic Surgeons' National Experience, 1991–2007. Circulation. 2013;127(16):1656–63. Doi: 10.1161/CIRCULATIONAHA.113.001882.
2. Xenogiannis I, Zenati M, Bhatt DL, Rao SV, Cabau JR, Goldman S, et al. Saphenous vein graft failure: from pathophysiology to prevention and treatment strategies. Circulation 2021;144(9):728-45. Doi: 10.1161/CIRCULATIONAHA.120.052163.
3. 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. Am Coll Cardiol. 2001;38(3):659-65. Doi: 10.1016/s0735-1097(01)01420-6.
4. 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. Available from: https://doi.org/10.1161/01.CIR.100.24.2400.
5. Yap CH, Sposato L, Akowuah E, Theodore S, Dinh DT, Shardey GC, et al. Contemporary results show repeat coronary artery bypass grafting remains a risk factor for operative mortality. Ann Thorac Surg. 2009;87(5):1386-91. Doi: 10.1016/j.athoracsur.2009.02.006.
6. Stefanini GG, Alfonso F, Barbato E, Byrne RA, Capodanno D, Colleran R, et al. Management of myocardial revascularisation failure: an expert consensus document of the EAPCI. Eurointervention. 2020;16(11):e875-e890. Doi: 10.4244/eij-d-20-00487.
7. Mack MJ, Squiers JJ, Lytle BW, DiMaio JM, Mohr FWM. Myocardial revascularization surgery: JACC historical breakthroughs in perspective, J Am Coll Cardiol. 2021;78(4):365-383. Available from: https://doi.org/10.1016/j.jacc.2021.04.099.
8. Gharibeh L, Ferrari G, Ouimet M, Grau JB. Conduits' biology regulates the outcomes of coronary artery bypass grafting. JACC State of the art review. JACC Basic Transl Sci. 2021;6(4):388–96. Available from: https://doi.org/10.1016/j.jacbts.2020.11.015.

9. Yazdani SK, Farb A, Nakano M, Vorpahl M, Ladich E, Finn AV, et al. Pathology of drug-eluting versus bare-metal stents in saphenous vein bypass graft lesions. JACC Cardiovasc Interv. 2012;5(6):666–74. Doi: 10.1016/j.jcin.2011.12.017.
10. Brilakis E, Banerjee S, Burke M. A new treatment strategy for saphenous vein graft lesions? Letting it go. J Am Coll Cardiol. 2018;71(18):1983–5. Available from: https: //doi.org/10.1016/j.jacc.2018.03.457.
11. Xenogiannis I, Tajti P, Hall AB, Alaswad K, Rinfret S, Nicholson W, et al. Update on cardiac catheterization in patients with prior coronary artery bypass graft surgery. JACC: Cardiovasc Interv. 2019;12(17):1635-49. Doi: 10.1016/j.jcin.2019.04.051.
12. Topol EJ, Teirstein PS. Textbook of Interventional Cardiology E-Book: Elsevier Health Sciences; 2018.
13. Eeckhout E, et al. PCR–EAPCI textbook of percutaneous interventional cardiovascular medicine. Europa Publications; 2019.
14. Aggarwal V, Stanislawski MA, Maddox TM Nallamothu BK, Grunwald G, Adams JC, et al. Safety and effectiveness of drug-eluting versus bare-metal stents in saphenous vein bypass graft percutaneous coronary interventions: insights from the Veterans Affairs CART program. J Am Coll Cardiol. 2014;64(17):1825-36. Doi: 10.1016/j.jacc.2014.06.1207.
15. Lansky AJ, Yang YM, Khan Y, Costa RA, Pietras C, Tsuchiya Y, et al. Treatment of coronary artery perforations complicating percutaneous coronary intervention with a polytetrafluoroethylene-covered stent graft. Am J Cardiol. 2006;98(3):370-4. Doi: 10.1016/j.amjcard.2006.02.041.
16. 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. Am J Cardiol. 2010;105(2):179-85. Doi: 10.1016/j.amjcard.2009.09.006.
17. Brennan JM, Al-Hejily W, Dai D, Shaw RE, Trilesskaya 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. Circ Cardiovasc Interv. 2015;8(3):e001403. Doi: 10.1161/CIRCINTERVENTIONS.114.001403.
18. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, et al. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. Circulation. 2011;124(23):e574-651. Doi: 10.1161/CIR.0b013e31823ba622.
19. Sachdeva A, Bavisetty S, Beckham G, Shen AY, Aharonian V, Mansukhani P, et al. Discontinuation of long-term clopidogrel therapy is associated with death and myocardial infarction after saphenous vein graft percutaneous coronary intervention. J Am Coll Cardiol. 2012;60(23):2357-63. Doi: 10.1016/j.jacc.2012.09.014.