WEBINAR: VEIN GRAFT INTERVEN-TIONS

# SASCI-Mayo Clinic Fellows webinar: Vein graft interventions

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## ABSTRACT

This publication is the third instalment in a series of webinars conducted jointly by the South African Society of Cardiovascular Intervention (SASCI) and Mayo Clinic. Hosted by the regular faculty, the webinar began with an in-depth analysis of the pathogenesis of vein graft disease by Dr Barsness. It included a critical appraisal of published data supporting various interventional strategies in this patient population. His presentation was followed by a clinical case study by Dr Engelbrecht, focusing on a patient with recurrent acute coronary syndrome (ACS) events due to vein graft disease. Cardiology fellows from various South African universities participated as discussants.

**Objective:** This manuscript, derived from the webinar series, summarises a multidisciplinary discussion of vein graft intervention complexities. It addresses the underlying pathophysiology, technical considerations, and current evidence-based management strategies.

**Case summary:** A male patient with prior coronary artery bypass grafting (CABG) presented with recurrent episodes of ACS secondary to progressive vein graft disease. The discussion explored the pathogenesis of vein graft disease and the technical challenges of intervention, with a specific focus on the evidence and clinical considerations when deciding between vein graft and native vessel revascularisation. Following recurrent ACS events and percutaneous coronary intervention (PCI) attempts in the diseased vein graft, the patient eventually achieved successful revascularisation through a chronic total occlusion (CTO) procedure in the native left anterior descending artery (LAD).

**Key messages**

- Pathophysiology of vein graft failure: Understand the pathogenesis and underlying mechanisms contributing to the long-term failure of saphenous vein grafts (SVG).
- Procedural complexities: Recognise the technical challenges and complications associated with vein graft interventions, with a focus on the no-reflow phenomenon.
- Mitigation strategies: Evaluate clinical strategies and pharmacological interventions to prevent and manage the no-reflow phenomenon.
- Comparative evidence: Review contemporary evidence guiding the choice between native vessel and vein graft PCI, including an evaluation of the recent PROCTOR trial.
- Clinical considerations: Outline key considerations for native vessel revascularisation in patients with previous CABG.

**Online resource:** Recorded SASCI fellows webinars (restricted to verified healthcare professionals) are available from: https://www.sasci.co.za/content/page/sasci-educational-videos1.

**Keywords:** CABG, vein graft interventions, ACS in CABG, vein graft vs native vessel PCI, pathogenesis of vein graft disease, cardiology education, SASCI fellows webinar.

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## INTRODUCTION

**SASCI convenor:** We are privileged to discuss the important topic of vein graft interventions. There is comparatively little evidence guiding our practice, but the recent publication of new data has renewed dialogue on what constitutes optimal management in patients with degenerated vein grafts. In keeping with our established format, we will begin with a general overview of the topic, followed by an interesting case presentation.

### Overview of vein graft interventions (Barsness)

Despite the risk and clinical effort put forth for open-heart surgery, vein grafts themselves are, at best, palliative. They have a high failure rate over time. Its mechanism can be divided into 3 timeframes:

1. Early (weeks to months): Technical issues or conduit/flow problems.
2. Intermediate (1–5 years): Fibromuscular proliferation and intimal hyperplasia.
3. Delayed (> 5 years): Accelerated atherosclerosis with accumulation of friable, necrotic "gruel"-like plaque.

At 10 years, only about 50% of vein grafts remain patent. The VEST trial randomised patients undergoing CABG to standard vein graft harvest and implantation techniques or use of an investigational external support device to improve graft patency. In this contemporary trial, the rate of vein graft failure in the

control group treated in the usual way was 25–30% at 1 year.[1] On a per-patient basis (because patients usually get more than 1 vein graft), about 40–50% of patients who underwent bypass surgery suffered from a failed vein graft within a year. So, whether on a per-graft or per-patient basis, the failure rate is very high, even in a contemporary series. Thus, if you are caring for patients with vein grafts, you will see disease accumulate, and you'll have to deal with it in some way.

Management selection for this progressive vein graft disease, whether it is repeat surgical intervention or PCI, depends on clinical and anatomic realities in this patient population. We know that surgical risk increases with low ejection fraction, age, female sex, long perfusion times, and triple-vessel disease. Moreover, many of these risk factors describe patients presenting post-bypass with recurrent disease. Hence, people with recurrent vein graft disease are at high surgical risk.

Beyond clinical status, we consider anatomy when deciding between repeat bypass and percutaneous revascularisation. Repeat surgical revascularisation can be an attractive option in some young patients with multivessel disease and multiple failed grafts, especially given the accelerated proximal native vessel disease progression often seen in patients following CABG. Therefore, we would consider repeat bypass in patients with ischaemia in the LAD territory, especially if an unused arterial conduit (the internal mammary artery) is still available. However, we consider vein graft PCI in most patients for clinical and anatomic reasons. In ACS, we think about it with unfavourable native target anatomy within 1–3 years with focal vein graft disease, or especially when there is patency of a prior arterial graft, usually to the LAD.

Until recently, I had been confident that the suitability of a native vessel as a target for PCI was a good reason to proceed with native vessel PCI rather than high-risk, repeat CABG or vein graft PCI. Despite a lack of data, I was intuitively certain that treating native vessels was the right approach in patients with vein graft disease. I'm going to talk about that for a second and why that may or may not still be true.

Long-term outcomes in a Mayo Clinic propensity-matched model suggest worse outcomes in patients who undergo PCI of the diseased vein graft compared with PCI of the native vessel, with a persistent, clinically and statistically significant effect for 10–15 years. This long-term outcome, at least in our series, suggests a benefit of either native vessel intervention or even repeat bypass surgery in these patients.[2] A large pooled analysis of 22 observational studies of 40 000 patients with 2-year follow-up reported consistent findings, suggesting that, compared with vein graft PCI, native vessel PCI was associated with reduced major adverse cardiovascular events (MACE), all-cause death, myocardial infarction (MI), and target vessel revascularisation – all largely statistically significant and with a large risk reduction across all those endpoints, with no difference in acute bleeding or stroke.[3]

Consequently, it seemed like native vessel PCI was the clear winner and should be the preferred option when feasible in patients with recurrent vein graft disease. Then, more recently, many of you will be aware of the PROCTOR trial, which prospectively randomised post-bypass individuals to either native vessel intervention or vein graft intervention. They found that native vessel PCI had a 34% MACE rate at 1 year, whereas vein graft PCI had an event rate of about half that, resulting in significantly reduced adverse events and a clear, statistically and clinically meaningful benefit favouring vein graft intervention.[4] All-cause mortality was not much different, yet there was a significant difference regarding PCI-related MI and repeat revascularisation.

There are a few noteworthy limitations to the study. The trial was stopped early due to funding and enrolment issues, but the investigators were still able to conduct a statistically meaningful endpoint analysis. However, stopping a study early always has its concerns. It was also an open-label design, which may have influenced clinical decision-making, particularly regarding revascularisation options. There was also a short 1-year follow-up, and I'm going to emphasise why that's important. Furthermore, this was a select population that excluded early graft failure, occluded vein grafts, and high-risk vein grafts. We'll talk about what that entails. Moreover, 72% of the treated native lesions were actually CTOs, mostly treated with a retrograde approach, and the stent length was almost 9 cm in patients with treated CTOs, compared with 2 cm in the vein graft patients. So, it speaks to the complexity of the native vessel treatment in this particular trial, as well as its applicability to the treatment of our own patients.

Conversely, a vein graft intervention study from SCAAR (Swedish Coronary Angiography and Angioplasty Registry) shows that at 3 years, all-cause mortality was 20%, MIs occurred in 20%, and any revascularisation in nearly 40%.[5] These population-based event rates are really an important sort of caveat when thinking about vein graft interventions.

Let's look at a case. Here, we see a patient with relatively focal, mid-vessel disease (at least the tightest portion is quite focal). It is easy to wire, but after a single balloon inflation, there is no-reflow, with a tenfold increase in infarction and mortality. So how do we identify this risk and subsequently manage it?

## Anticipating no-reflow

It turns out that plaque volume plays a big role. Plaque volume is directly related to the risk for no-reflow and adverse events associated with vein graft intervention, which can be substantial even early after CABG. Plaque volume can be assessed either by an integrated assessment all along the graft or by using a scoring system (semi-quantitative vein graft degeneration score).[6]

## How to prevent no-reflow

Embolic protection devices (EPD) have some benefits. A pooled analysis of the trials demonstrated the general benefits of EPDs, as well as a relative risk reduction in each quartile of degeneration score.[6,7] This just means you can't really predict who won't

benefit from embolic protection. However, you know that the higher the degeneration score, the higher their absolute risk, and that relative risk reduction translates to a greater absolute risk reduction in these patients. EPDs, at least in this study, showed a clear benefit compared with standard care in the early years. However, not everyone uses an EPD, and it's not universally used in most labs. The reason might be ascribed to a meta-analysis of multiple observational studies and 2 randomised trials, which did not show a statistically significant difference in MACEs with EPDs, leading to a downgrade in United States guidelines from class I to class IIA. There are also technical challenges with EPDs, like how to use them in a distal vein graft lesion or in tortuous anatomy.[8]

Other interventions for management or prevention of no-reflow and distal embolisation include good peri-procedural anticoagulation, which should go without saying. However, GP2B3A inhibitors have a class III indication and should not be routinely used. Other agents such as nicardipine, verapamil, nitroprusside, and adenosine can be considered. It's possible to use these agents to pre-treat a vein graft. In most cases, you can give the agent through the guide. However, when treating no-reflow, it is important to give these agents distally (e.g. using a microcatheter). Other considerations include direct stenting with non-aggressive stent sizing, avoiding pre-dilation and other vessel manipulation as much as possible. In essence, when intervening on a vein graft, we are slightly limited in the tools available with proven efficacy. Regarding PCI, a DES is preferred to POBA in most instances.

### Summary

- The new data from the PROCTOR trial at least gives us pause to consider the benefit of treating focal vein graft lesions, even if the native vessel is amenable to revascularisation, especially if it's a CTO.
- If the vein graft is heavily diseased and there is native vessel disease, even a CTO that can be addressed, treating the native vessel disease in an antegrade fashion, or using the vein graft as a conduit for retrograde CTO treatment, is certainly a reasonable option.
- Optimal medical therapy, as always, is essential.

## CASE PRESENTATION

**Fellow 1:** I'll be jumping straight into our case. A 77-year-old gentleman has cardiovascular risk factors of diabetes, hypertension, and dyslipidaemia. He had CABG in 2008 with left internal mammary artery (LIMA) to LAD, a vein graft to his circumflex, and a vein graft to his first diagonal. In December 2017, he presented with life-limiting angina and a stress echo, which showed inducible ischaemia in his posterior lateral wall. Angiography showed a CTO of his proximal LAD with a dominant left circumflex artery (LCx) system with severe stenosis in the mid-circumflex. Regarding his right system, he had a CTO of his distal right coronary artery (RCA). On injection of his grafts, he had an occluded SVG to the LCx. The SVG to the diagonal was patent and filled the LAD retrogradely. The LIMA was small and atretic, likely due to competitive flow from the SVG to D1. The decision was made to treat the lesion in the LCx with a stent, as it was thought to be responsible for the inducible ischaemia.

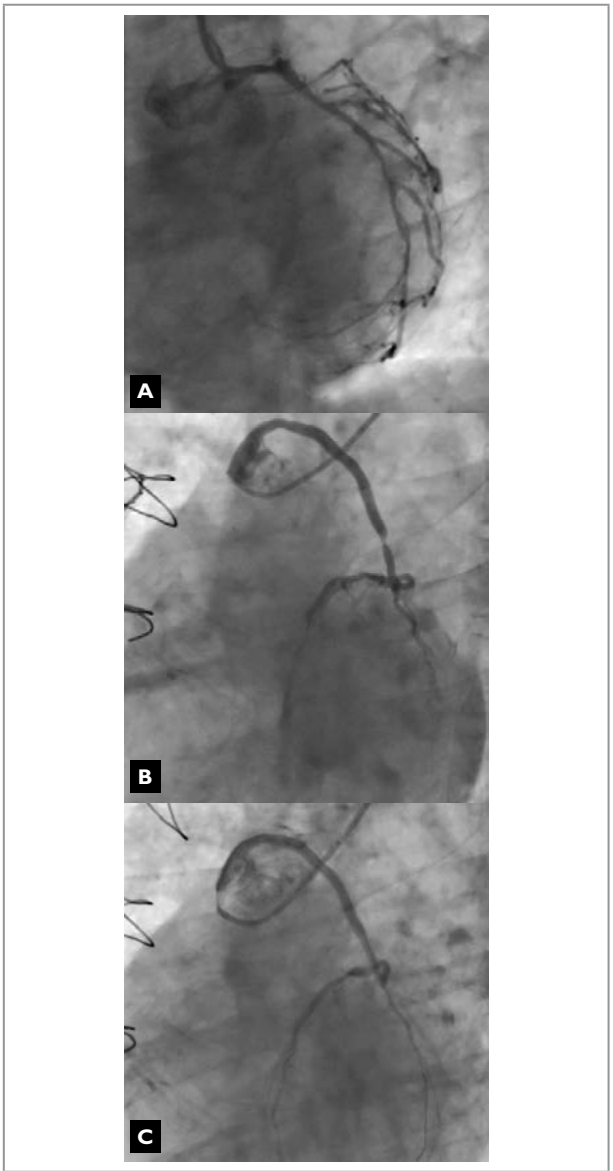


**FIGURE 1: (A) LAO caudal view demonstrating native vessels with CTO of the proximal LAD, (B) vein graft injection demonstrating severe distal lesion in the SVG to diagonal (note concomitant filling of LAD), (C) SVG after PCI with stent to lesion.**
*CTO: chronic total occlusion, LAD: left anterior descending artery, LAO: left anterior oblique, PCI: percutaneous coronary intervention, SVG: saphenous vein graft.*

The same patient presented to us 7 years later in 2024, with non-ST-segment elevation myocardial infarction (NSTEMI) and a troponin leak of 146. Another angiogram was performed, showing findings similar to those before, except that SVG to D1 now had a focal, discrete lesion in the distal vein graft, which was the probable culprit (Figure 1). So, what do we do next?

**Fellow 2:** If you consider that the LAD is effectively being supplied via the diseased graft due to the atretic LIMA, then the diseased SVG becomes a very important vessel. Additionally, it appears anatomically favourable for PCI; a focal lesion in a vein graft that appears otherwise healthy and of good calibre. Alternatively, you could consider a CTO procedure of the native vessel. However, considering the lesion's focal nature and the recent PROCTOR trial, it is probably worthwhile to address the vein graft lesion with PCI.

**Mayo faculty:** This is a fantastic case because it brings up everything we don't know about vein graft intervention. Firstly, this is an ACS case, and it is clearly a high-risk setting in which the patient is truly dependent on this vein graft. We don't have any data about treating vein graft lesions in ACS. There's not a single trial that we can rely on. There are anecdotes, and that's all. But, I will say that apart from the fact that this is ACS, which was not included in the PROCTOR trial, this is the kind of lesion that would have been included in the trial – a very focal vein graft lesion and complex CTO of the LAD. At this point, the data suggest that performing vein graft intervention would yield the best 1-year outcome for this patient. Unfortunately, it is just distal enough to preclude you from using an EPD, especially for the retrograde LAD territory.

A few more technical considerations (remember we are trying to avoid no-reflow):

- Ensure adequate anticoagulation and consider pre-treating with some antithrombotic agent to good levels.
- We don't have any data on GP2B3A inhibition in this case; in fact, we have negative data for GP2B3A inhibition in this patient cohort.
- Remember, it is crucial you do not putter around with this lesion. You wouldn't pre-dilate it because every time you touch it, you increase the potential for problems. You don't want to do much post-dilatation either.
- Regarding stents, you don't necessarily want to oversize or undersize them. You want to treat it with the appropriately sized balloon and ensure the procedure goes well. However, in this particular case, perfection is the enemy of good.

**SASCI convenor:** Ideally, you would like to perform direct stenting (sized 1:1) and provide sufficient inflation to achieve good initial expansion. This vein graft is more than a decade old, so you must anticipate fibrosis and gruel throughout the graft. Even though we don't see irregularities, we know the whole graft is lined with atherosclerotic "junk".

**Fellow 1:** We proceeded with PCI to the SVG lesion. We pre-dilated with a 2.5 × 15 mm semi-compliant (SC) balloon (considering our recent discussion, this might not have been the best strategy), then deployed a 3.5 × 15 mm drug-eluting stent (DES). Our result was good with maintained flow (Figure 1C).

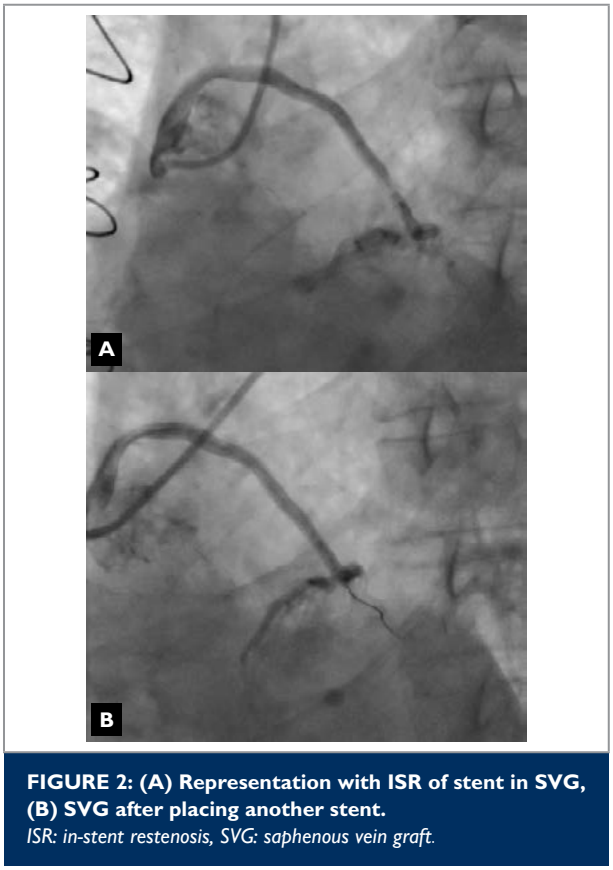


**FIGURE 2: (A) Representation with ISR of stent in SVG, (B) SVG after placing another stent.**
*ISR: in-stent restenosis, SVG: saphenous vein graft.*

Unfortunately, we saw the patient again 5 months later. He presented with another NSTEMI and troponin leak of 625. We performed a new angiogram; the native vessels were unchanged, but there appeared to be in-stent restenosis (ISR) or thrombosis at the distal end of the previously placed stent. We decided to re-intervene on this vein graft. We pre-dilated once more and deployed another 3.0 × 15 mm stent with distal overlap of the old stent. We were quite happy with our result (Figure 2). The patient did well for 9 months. Unfortunately, he presented again with NSTEMI and troponin leak of 922. A repeat angiogram showed recurrent ISR in the distal vein graft. The patient adhered to his medication, and his diabetes was under control, but he did have worsening chronic kidney disease (CKD), which is a risk factor for ISR

**Fellow 2:** I would like to optimise all the patient's risk factors and consider prolonged dual antiplatelet therapy (DAPT) in this case, depending on the bleeding risk. I am reluctant to place another stent; instead, I would consider a drug-coated balloon (DCB) as an option. Also, given that this is his third presentation, it may be time to adopt a different strategy, like native vessel intervention. Another option would be repeat CABG with right internal mammary artery (RIMA) grafting, if feasible.

**Mayo faculty:** Regarding the DCB strategy, the pathology here is different from that in native vessel disease. You have this "peanut butter" inside the vessel, and coating it with a DCB probably won't make much difference.

**Fellow 1:** We were reluctant to intervene a third time, so we opted for medical therapy and intensified low-density lipoprotein (LDL)-lowering. Unfortunately, our plan was short-lived, as we saw him again 1 month later with another NSTEMI. The repeat angiogram showed the same filling defects in the stent portion of the SVG. We thought that the LAD territory was still viable. To summarise, this is a patient with recurrent ACS episodes who had repeated interventions on the SVG and complex CTO of the LAD. We think that our only option here is native vessel PCI and opening the LAD CTO. Any thoughts on this before we continue?

**Mayo faculty:** This is going to be a challenging intervention. There is a retrograde conduit, which was how most of the CTOs were treated in the PROCTOR trial. But the anatomic complexity and calcium burden are high, making intervention potentially difficult. Nonetheless, it is the only real option at this point. I would likely take a dual-injection approach with a guide in the left main stem (LMS) and another guide in the graft, and try to work via a retrograde approach, as getting into the LMS from the LAD will be easier than the reverse.

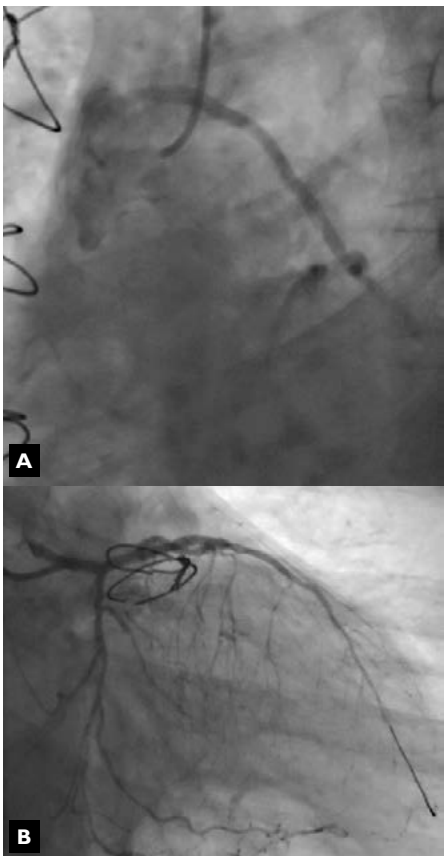


**FIGURE 3: (A) Patient represented with ACS and recurring disease in the vein graft, (B) native vessel treated with CTO procedure of LMS into LAD.**
*ACS: acute coronary syndrome, CTO: chronic total occlusion, LAD: left anterior descending artery, LMS: left main stem.*

**Fellow 1:** We proceeded with the CTO procedure, opting for an initial anterograde approach. We used the right radial access, with an EBU guide. We employed anterograde wire escalation, using a PROGRESS 40 guide wire after failing with a BMW and Fielder XT-A. We were able to deliver the PROGRESS wire into the distal LAD using a FineCross microcatheter. We were unable to deliver balloons over the PROGRESS, so we changed to a Grand Slam wire via the microcatheter for extra support. We could then pass a MINI TREK balloon, progressively escalate to a bigger balloon, and pre-dilate all the way back to the LMS. We proceeded to deploy a 3.0 × 48 mm stent from the LMS into the LAD and used a provisional strategy with proximal optimisation technique (POT) and kissing inflation to optimise the LAD-LCx bifurcation. We were quite satisfied with the result (Figure 3).

**SASCI convenor:** This case highlights that vein grafts are not without problems. We probably faffed around with the vein graft disease for too long. Once they start closing, they don't want to be opened. Whenever you face a vein graft problem, consider tackling the native vessel. That brings our discussion to an end. It has been a terrific session. Thank you to our faculty, Prof Barsness, for the great talk, and Prof Holmes, for the great discussion.

## REFERENCES

1. Goldstein DJ, Puskas JD, Alexander JH, et al. External support for saphenous vein grafts in coronary artery bypass surgery: A randomized clinical trial. JAMA Cardiol. 2022;7(8):808-16. https://doi.org/10.1001/jamacardio.2022.1437.
2. Locker C, Greiten LE, Bell MR, et al. Repeat coronary bypass surgery or percutaneous coronary intervention after previous surgical revascularization. Mayo Clin Proc. 2019;94(9):1743-52. https://doi.org/10.1016/j.mayocp.2019.01.048.
3. Farag M, Gue YX, Brilakis ES, Egred M. Meta-analysis comparing outcomes of percutaneous coronary intervention of native artery versus bypass graft in patients with prior coronary artery bypass grafting. Am J Cardiol. 2021;140:47-54. https://doi.org/10.1016/j.amjcard.2020.10.062.
4. de Winter RW, Hoek R, Walsh SJ, et al. PCI of native coronary artery vs saphenous vein graft after prior bypass surgery: A multicenter, randomized trial. J Am Coll Cardiol. 2026;87(3):269-82. https://doi.org/10.1016/j.jacc.2025.09.1577.
5. Saidi-Seresht S, James S, Erlinge D, et al. Outcome of saphenous vein graft percutaneous coronary intervention using contemporary drug-eluting stents: A SCAAR report. J Soc Cardiovasc Angiogr Interv. 2024;3(10):102232. https://doi.org/10.1016/j.jscai.2024.102232.
6. Coolong A, Baim DS, Kuntz RE, et al. Saphenous vein graft stenting and major adverse cardiac events: A predictive model derived from a pooled analysis of 3958 patients. Circulation. 2008;117(6):790-7. https://doi.org/10.1161/CIRCULATIONAHA.106.651232.
7. Naidu SS, Turco MA, Mauri L, et al. Contemporary incidence and predictors of major adverse cardiac events after saphenous vein graft intervention with embolic protection (an AMEthyst trial substudy). Am J Cardiol. 2010;105(8):1060-4. https://doi.org/10.1016/j.amjcard.2009.12.004.
8. Paul TK, Bhatheja S, Panchal HB, et al. Outcomes of saphenous vein graft intervention with and without embolic protection device: a comprehensive review and meta-analysis. Circ Cardiovasc Interv. 2017;10(12):e005538. https://doi.org/10.1161/CIRCINTERVENTIONS.117.005538.