

# Surgical Treatment of Saphenous Vein Graft Aneurysms After Coronary Artery Revascularization

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**Background.** Saphenous vein graft (SVG) aneurysms (SVGAs) after coronary artery bypass grafting (CABG) occur rarely. Most reports are anecdotal. To determine early and late outcomes of surgical treatment, we reviewed our experience with management of this rare complication of surgical revascularization.

**Methods.** From July 1975 to October 2007, 16 patients (15 men), mean age,  $60.9 \pm 14.6$  years, underwent repair of aortocoronary SVGAs.

**Results.** Chest pain was present in 11 of 16 patients. The rest were asymptomatic. The average maximum diameter of the SVGA was  $64 \pm 30$  mm. The concern of SVGA rupture was the primary indication for operation in 9 patients (56%). Repair in the remaining patients occurred during other cardiac operations. A pseudoaneurysm (75%) at the body or anastomotic sites of the SVG was the most common cause of SVGA. In 8 patients

(50%), the aneurysm involved SVG anastomotic sites. Thirteen patients (81%) had intraluminal thrombi. Vein grafts with aneurysm were patent in 9 patients (56%). Surgical procedures included excision of the aneurysm and direct distal coronary target vessel revascularization in 10 (63%), excision and interposition vein graft in 5 (31%), and exclusion by ligation in 1 (6%). Median follow-up was 7 years (maximum, 20 years). Survival was 83% at 5 years and 72% at 10 years after SVGA repair.

**Conclusions.** Ischemic symptoms often accompany SVGA, and operation is indicated to prevent rupture. Ligation or excision of SVGA with simultaneous revascularization appears to be the optimal therapy, with satisfactory midterm and long-term results.

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Most operations for coronary artery bypass grafting (CABG) involve the use of reversed saphenous vein grafts (SVGs). The late failure of a reversed SVG is due primarily to graft atherosclerosis that produces stenosis of the conduit. Aneurysmal dilatation and pseudoaneurysms of aortocoronary SVGs (SVGAs) are rare but potentially dangerous late complications [1, 2]. These usually occur at the proximal or distal anastomosis of the SVG, but they may affect the body of the graft. Pseudoaneurysms may cause serious complications, including rupture, compression of surrounding structures, fistula formation, or myocardial infarction [3–5]. Most previous publications have reported single patients [6, 7]. We investigated and analyzed the presentation, operative techniques, and long-term outcome of surgical repair for SVGA in a series of patients.

## Patients and Methods

Between July 1975 and October 2007, 28,603 patients underwent CABG at Mayo Clinic Rochester. During this interval, 16 patients had repair of SVGA and 9 had a prior operation elsewhere. The median patient age was 65 years (range, 23 to 81 years), and 15 were men (94%).

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Institutional approval for this study was obtained from the Mayo Clinic Institutional Review Board, and each patient provided informed consent.

Medical records were reviewed for demographic and clinical information including age, gender, indication for coronary angiography, cardiac risk factors, and cardiac events, such as previous myocardial infarction and percutaneous coronary intervention. Follow-up forms were sent to the patients asking about their previous condition, general health, limitations, and subsequent surgery. Graft patency was defined as maintaining aortocoronary circulation through the aneurysmal SVG.

## Statistical Analysis

Continuous variables were expressed as mean  $\pm$  standard deviation or median (range) where appropriate. Categorical variables were expressed as frequency and percentage. Overall survival rates at 5 and 10 years were estimated by using the Kaplan-Meier technique, and 95% confidence intervals (CI) were calculated for each of these estimates.

## Results

Most of the patients underwent CABG during the earlier era when SVGs were the most commonly used conduits. Among the patients with SVGA, a left internal thoracic artery (LITA) was used at initial operation in only 2

Table 1. Patient Demographic Characteristics and Preoperative Data

Variables	No (%) or Mean ± SD (Range)
Age, y	62.1 ± 14.6 (22-81)
Gender	
Female	1 (6)
Male	15 (94)
Hypertension	8 (50)
Diabetes mellitus	3 (19)
Hyperlipidemia	10 (71)
Smoking	9 (64)
Previous MI	13 (93)
Ejection fraction	0.475 ± 0.167 (0.20-0.65)
Ejection fraction < 0.40	4 (23)
NYHA functional class	1.8 ± 0.5
CCS angina class	2.4 ± 1.0

CCS = Canadian Cardiovascular Society; MI = myocardial infarction; NYHA = New York Heart Association; SD = standard deviation.

patients. Other concomitant cardiac procedures at the time of initial CABG included aortic valve replacement (13%), left ventricle aneurysm repair (13%), and mitral valve replacement (6%).

Our patients with SVGA underwent surgical repair at an average of 13 ± 7 years (range, 6 to 28 years) after their first operation. The clinical characteristics of all patients at the time SVGA repair are presented in Table 1. Chest pain was present in 11 patients (69%), and the SVGA in 5 patients was discovered incidentally during graft angiography, echocardiography, and chest roentgenogram or during an operation. In the remaining 5 patients without chest pain, the SVGA was identified during evaluation of aortic valve stenosis, at the time of drainage of a mediastinal abscess, during evaluation of dyspnea on echocardiogram, and accidentally on chest roentgenogram.

The presence of the SVGA and the attendant risk of rupture was the primary indication for repair in 9 patients (56%), and mean maximum diameter of the SVGA in these patients was 78 ± 23 mm (range, 50 to 120 mm). In the remaining 7 patients, the SVGA was diagnosed and treated incidentally at the time of reoperation for other indications, and the mean maximum diameter in these patients was 29 ± 11 mm (range, 18 to 40 mm).

The locations of the SVGA are presented in Table 2. The aneurysm in 6 patients (38%) involved a graft to the left anterior descending (LAD) coronary artery, and 4

Table 2. Aneurysmatic Lesion Location According to Target Anastomosis

Aneurysm Location	No. (%)
Left anterior descending graft	6 (38)
Right coronary artery graft	4 (25)
Circumflex coronary artery graft	3 (19)
Diagonal coronary artery graft	1 (6)
Multiple coronary artery grafts	2 (13)

Table 3. Patient Operative Characteristics

Variables	No. (%) or Mean ± SD (Range)
Aortic cross-clamp time, min	74 ± 41 (37-137)
CPB time, min	143 ± 73 (46-262)
Excision	
+ CABG	10 (63)
+ Vein interposition	5 (31)
Ligation alone	1 (6)
Early death	1 (6)
Follow-up, mon	86 ± 78 (1-240)
5-year survival, %	83
10-year survival, %	72
NYHA functional class	8
I	5 (63)
II	2 (25)
III	1 (13)
CCS angina class	
No angina	4 (50)
I	3 (38)
IV	1 (13)

CABG = coronary artery bypass grafting; CCS = Canadian Cardiovascular Society; CPB = cardiopulmonary bypass; NYHA = New York Heart Association; SD = standard deviation.

patients (25%) had right coronary artery graft aneurysms. Graft aneurysms developed in circumflex bypass conduits in 3 patients (19%), and 1 patient (6%) had a diagonal coronary graft aneurysm. One patient (6%) had 2 aneurysms that involved grafts to the LAD and right coronary arteries; another patient had 3 separate vein graft aneurysms involving the right, circumflex, and LAD coronary grafts.

Redo sternotomy was the approach in 94% of patients. In 2 patients with bypass graft occlusion distal to the aneurysms, a lateral thoracotomy was used for ligation and excision of SVGA without additional revascularization. One of these patients was converted to redo sternotomy due to difficulty in obtaining full access to the aneurysm. Arterial cannulation was performed through ascending aorta in 10 patients (63%) and through the femoral artery in 4 (25%). The operation in 2 patients (13%) was performed with off-pump technique. Procedures are detailed in Tables 3 and 4. Vein grafts were used to replace SVGA or to bypass the same target vessels in 88%. Two patients (13%) underwent CABG with LITA to LAD after excision of the SVGA.

Intraoperative findings and details of the specific locations of SVGAs are summarized in Table 4. Half of the SVGAs involved the anastomotic site, most likely representing a pseudoaneurysm (Fig 1). In the remaining 8 patients, the SVGA was within the body of the SVG, without involving the suture line (Fig 2). Histologic study of the resected SVGAs revealed pseudoaneurysm in 12 patients (75%) and true aneurysms in 2 (13%). No pathologic classification was given in 2 patients. Thirteen patients (81%) had intraluminal thrombus (Fig 2). Aneurysmal vein grafts were occluded in 7 patients (44%).

Table 4. Intraoperative Approach to Saphenous Vein Graft Aneurysms

Pt	Diameter, mm	SVGA Localization	Replaced Graft	Outcome and Survival
1	40	Proximal RCA graft	SVG	Alive, stable
2	30	Proximal LAD graft	SVG int	Died 13 years after SVGA repair
3	80	Midportion CX graft	No	Alive, stable
4	60	Distal RCA graft	SVG	Alive, stable
5	UK	Proximal diagonal graft	SVG	Died 4 years after SVGA repair
6	UK	Distal RCA graft	SVG	Early death
7	80	Midportion RCA graft	SVG int	Alive, stable
8	UK	Midportion LAD graft	LITA	Died 14 years after SVGA repair
9	UK	Proximal LAD graft	SVG	Lost to follow-up
10	18	Proximal LAD graft	SVG	Died 12 years after SVGA repair
11	UK	Proximal LAD, CX, RCA grafts	SVG int	Died 6 years after SVGA repair
12	70	Midportion CX graft	SVG int	Died
13	120	Midportion LAD graft	SVG	Alive, stable
14	100	Midportion LAD graft	LITA	Alive, stable
15	60	Midportion CX graft	SVG	Alive, stable
16	50	Midportion RCA graft	SVG int	Alive, stable

CX = circumflex; int = interposition; LAD = left anterior descending; LITA = left internal thoracic artery; Pt = patient; RCA = right coronary artery; SVG = saphenous vein graft; SVGA = saphenous vein graft aneurysm.

One patient (6%) presented with an infected SVGA and purulent pericarditis and subsequently died on postoperative day 19 due to ongoing sepsis and multiorgan failure. Median follow-up was  $7 \pm 6$  years (range, 19 days to 20 years). Survival of the patients after surgical SVGA repair was 83% (95% CI, 63.1% to 100%) at 5 years and 72% (95% CI, 49.6% to 100%) and 10 years. No SVGAs were known to have developed subsequently in any of the patients. Follow-up functional and anginal status is presented in Table 3.

**Comment**

Late failure of saphenous vein aortocoronary bypass grafts is predominantly due to vein graft atherosclerotic disease. Although SVGs have been used in a large number of patients undergoing CABG, only a few case

reports have described aneurysmal dilatation of these conduits [8-10]. The pathogenesis of SVGA formation is still unclear.

Indeed, SVGAs are rare, and we encountered only 16 patients who underwent surgical repair related to SVGAs during a 30-year interval in which more than 28,603 patients underwent CABG at our Mayo Clinic. The true prevalence is almost certainly higher because many patients will be asymptomatic. As reported by others [11, 12], chest pain was the most common presenting symptom, although we could not determine whether SVGA was the cause. There could be a number of reasons for chest pain, such as progression of native coronary artery disease, vein graft occlusion, and distal embolization from intraluminal thrombus or compression from a large mass of SVGA in these patients. Initial presentation could be rupture of the aneurysm leading to cardiac tamponade and sudden death.

Surgical treatment was recommended in most of our patients to prevent rupture, and the SVGAs were an average size of 8 cm. The exact risk of rupture is unknown, and only 1 patient in this series was symptomatic from acute expansion/rupture. This patient was monitored with echocardiogram and computed tomography (CT) imaging for 3 years until the SVGA became symptomatic. The rest of the patients underwent operation once the SVGA was diagnosed. However, gradual enlargement does lead to compression of mediastinal structures, including other bypass grafts, and fistula formation has been reported. As others have pointed out, the triad of chest pain, mediastinal mass, and previous CABG should raise concern of a SVGA [12, 13].

Management of SVGA depends on recognition, subsequent evaluation with CT or magnetic resonance imaging of the chest to delineate its location and patency of the graft lumen [12]. Evaluation of any suspected SVGA

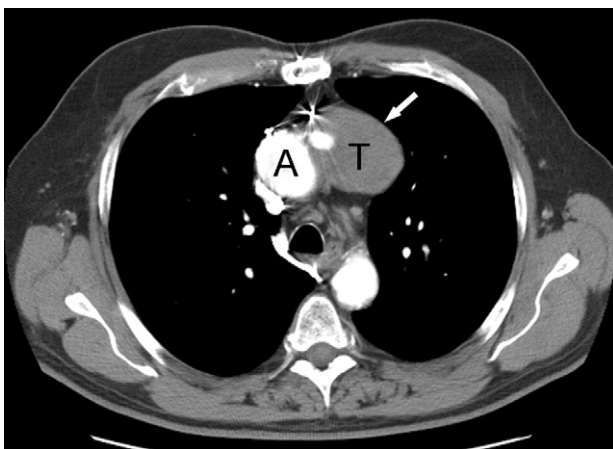


Fig 1. Computed tomography image shows aneurysmatic saphenous vein graft aneurysm (arrow) full of thrombus (T). (A = aorta).

Fig 2. A patent aneurysmatic vein graft with contained rupture is shown by (A) angiography and (B) computed tomography imaging. (C) Gross specimen of an aneurysmatic vein graft. (D) Gross segment shows contained rupture and atherosclerotic vein graft.

should include graft and coronary angiography to determine patency and the presence of other coronary stenosis that should be addressed at the time of operation. Graft angiography can appear relatively normal if the aneurysm sac contains a large amount of clot but the aneurysmal graft is patent [2]. As a complication of these aneurysms, a fistula to one chamber of the heart may develop [14, 15]. Most often, the interval between intervention and diagnosis of the aneurysm exceeds 5 years [1, 15]. In a review article that included 50 cases of SVGA from various centers [16], the aneurysms were diagnosed at a mean of 10 years (range, 7 days to 21 years) after CABG. The interval between the previous intervention and the diagnosis of the aneurysm in our study was 13 years, and none of our patients had a fistula.

The site of the SVGA provides some information on pathogenesis: Half of the vein graft aneurysms in this study involved the proximal or distal anastomotic sites, suggesting pseudoaneurysm formation caused by suturing techniques. The other half were contained within the vein graft itself, which we might consider a true aneurysm in nature. Two patients had multiple aneurysms on the same aneurysmatic graft or on different target grafts; however, pathologic examination of the resected specimens identified a true aneurysm in only 2 patients.

We speculate a true aneurysm with a leak or contained rupture by the time of repair may lead to a pathologic diagnosis of pseudoaneurysm, which might explain the fewer pathologic diagnoses of true aneurysms in our series compared with current reports [17]. True SVGAs are typically fusiform and involve the body of the graft. One could speculate that true SVGAs form in thin-walled

areas that are intrinsically weak. Benchimol and associates [18] suggested that the vicinity of the venous valves may be intrinsically weak because of a lack of circular muscle; veins may also be weakened at branching sites or in areas damaged at the initial surgical procedure [15].

Alternatively, graft atherosclerosis may be involved. Hypertension and hyperlipidemia could accelerate arteriosclerosis and formation of a true aneurysm [19]. Other mechanisms include injury incurred during vein harvesting [20]. It is interesting to speculate on whether new methods of saphenous vein harvesting with the endoscope or with limited skin incisions may lead to more thermal or traction injury and possibly an increased incidence of SVGA over time.

The use of arterial grafts may prevent problems unrelated to suturing technique because arterial grafts are less prone to trauma during harvesting and have stronger vessel walls. Pseudoaneurysms, in contrast, are sacular. They usually develop at the anastomotic site and are often related to technical issues, infections, or partial tears [11, 16]. In the current series, the common location of the SVGA was at the anastomotic suture line.

Treatment includes excision of the aneurysm and further revascularization if necessary. Special care should be taken to prevent perioperative myocardial infarction by atheroembolism. Traction and manipulation of aneurysmal vein grafts should be avoided before cross-clamping, cardiac arrest, and ligation of diseased veins. In one patient in our series, perioperative myocardial infarction was identified at autopsy; this patient presented with purulent pericarditis and had cardiogenic shock postoperatively. We believe antegrade cardioplegia is not a

direct cause of embolic events, but direct cannulation of diseased vein grafts should be avoided when possible. LITA grafts were used in 2 patients with occluded SVGAs to the LAD. Because of concern of inadequate flow when the LITA is used as a replacement of a patent SVGA to LAD graft, SVG were the preferred conduit of choice in this setting.

The appropriate timing of surgical intervention for patients with a SVGA is unknown. Dieter and associates [7] suggested conservative management of SVGA, but they had only 2 surgically treated patients in their group and the mean aneurysm size in their conservative group was approximately 3 cm. We advocate prompt repair and surgical revascularization of symptomatic patients with aneurysms to prevent potential complications [1, 15, 21]. A more conservative, observational approach for asymptomatic patients seems reasonable for SVGAs of less than 1 cm in diameter and with brisk graft flow. Longitudinal observation can be performed noninvasively by using magnetic resonance imaging or CT. For those patients with an SVGA diameter exceeding 1 cm or with diminished graft flow, prompt surgical revascularization should be recommended [16].

The small sample size limits the more general applicability of our findings; however, important concepts and pitfalls of SVGA were presented. Unfortunately, we did not have a control group, which ideally would include patients diagnosed with this rare pathology but followed up medically. One patient was lost to follow-up.

In conclusion, patients with SVGA often present with symptoms of angina. Surgical repair is recommended for asymptomatic patients with larger aneurysms because of the risk of rupture. A pseudoaneurysm at the anastomotic site was the most common finding, and this may be preventable with meticulous surgical technique. Ligation or excision of the SVGA with simultaneous revascularization appears to be the optimal therapy, with satisfactory midterm and long-term results.

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