Giant Saphenous Vein Graft Aneurysm Causing Left Atrial Compression and Cardiogenic Shock

Chee Yuan Ng, MD, Steven Stevens, MD, Mamta Buch, MD, PhD, Suhail Dohad, MD, Prediman K. Shah, MD, FACC, FACP, FCCP, Bojan Cercek, MD, PhD, Kuang-Yuh Chyu, MD, PhD

Division of Cardiology and Cedars Sinai Heart Institute, Cedars Sinai Medical Center, Los Angeles, CA

Saphenous vein graft aneurysm is a rare complication of coronary artery bypass graft (CABG) surgery that is challenging to manage and is associated with catastrophic consequences. We present the case of a 72-year-old woman with prior CABG surgery who presented with chest pain and was found to have a giant saphenous vein graft pseudoaneurysm. Further evaluation revealed that a vein graft pseudoaneurysm was causing significant compression of the left atrium. The pseudoaneurysm was successfully excluded from the blood flow with a covered stent; however, despite intra-aortic balloon pump and supportive therapy, the patient succumbed to cardiogenic shock and sepsis.

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Key words: Saphenous vein graft aneurysm • Pseudoaneurysm • Acute coronary syndrome • Percutaneous intervention • CT angiography

A 72-year-old woman with a history of coronary artery bypass grafting (CABG) presented with symptoms of new-onset chest pain. The sudden onset of chest pain began at rest; it was sharp and retrosternal, it radiated to the back, and was associated with diaphoresis, nausea, vomiting, and shortness of breath. The patient had been asymptomatic since her CABG procedure 18 years prior. Her past medical history included hypertension, dyslipidemia, and endovascular repair of an abdominal aortic aneurysm. She had undergone CABG with 4 vessel bypasses: left internal mammary artery (LIMA) to the left anterior descending artery (LAD), and the saphenous vein graft (SVG)

was a triple jump graft to 2 circumflex marginal branches and to the posterior descending artery. She was a former smoker and had a family history of coronary artery disease.

On presentation to the emergency room, she was hemodynamically stable. On physical examination, she was noted to have a pulse of 80 beats/min and a blood pressure (BP) of 100/54 mm Hg. The lungs were clear to auscultation. There was no jugular venous distention or lower extremity edema. Cardiac examination revealed no palpable precordial impulse, a normal s1 and s2 and a grade 2/6 early peaking systolic ejection murmur at the upper left sternal border, without radiation to the carotid arteries. Initial electrocardiogram revealed ST depressions in leads II, III, AVF, V4, V5, and V6. Initial troponin I level was 0.02 ng/mL, which subsequently increased to 3.30 ng/mL.

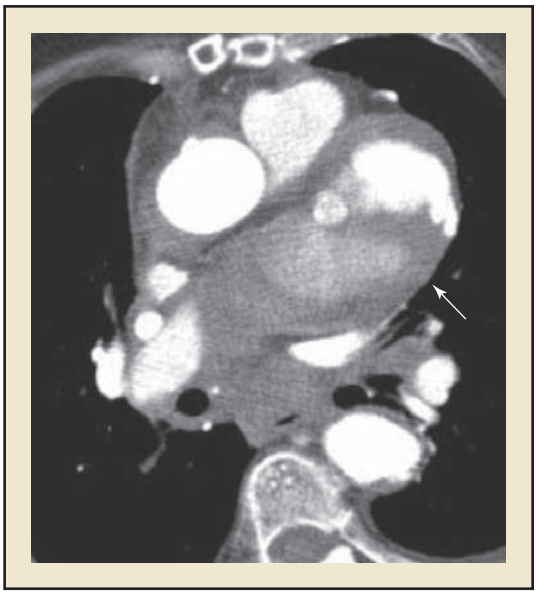

Figure 2. *Contrast-enhanced computed tomography angiogram of chest showing pseudoaneurysm* (arrow) *emanating from the saphenous vein graft measuring 8.3 × 5.2 cm. It contains a thrombosed component as well as a contrast enhancing component.*

Contrast-enhanced computed tomography (CT) angiogram of the chest (Figure 2) was remarkable for a pseudoaneurysm emanating from the SVG. The pseudoaneurysm measured 8.3 × 5.2 cm and contained a thrombus. Coronary angiography confirmed the findings of the CT scan and also showed occlusion of the posterior descending artery (Figure 3), likely accounting for her acute coronary syndrome (ACS). The other 2 marginal arteries distal to the triple jump graft were patent. Intravascular ultrasound (IVUS) demonstrated the presence of a pseudoaneurysm involving the media and adventitia in the midbody of the SVG. Of note, the LIMA to LAD graft was patent but the distal artery was diffusely diseased. No intervention was done given the complexity of the lesions.

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A chest radiograph demonstrated a prominent rounded soft tissue density near the left hilar area (Figure 1).

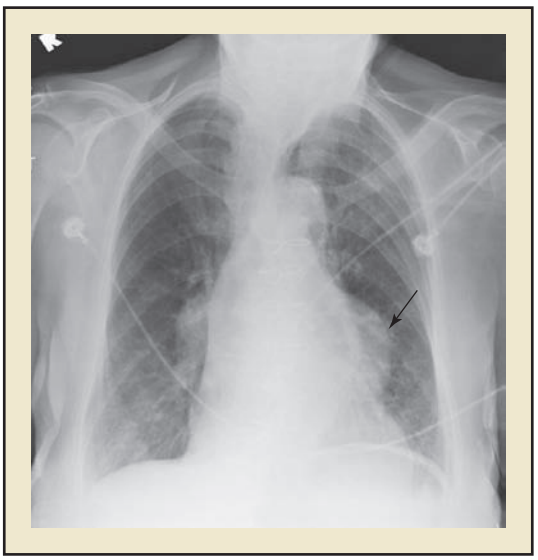

Figure 1. *Anteroposterior view of chest radiograph demonstrating prominent left hilar structure* (arrow).

After consultation with the cardiothoracic surgical team, it was recommended that percutaneous isolation of the vein graft pseudoaneurysm be considered because surgery would be too high risk due to the patient's comorbidities, as well as because of the proximity of the pseudoaneurysm to both the pulmonary artery and left atrium. The age-adjusted Charlson comorbidity index score was calculated to be 5, which correlated to a predicted 1-year mortality rate of 85%.[1,2] Therefore, the patient was initially managed conservatively with plans for subsequent percutaneous intervention.

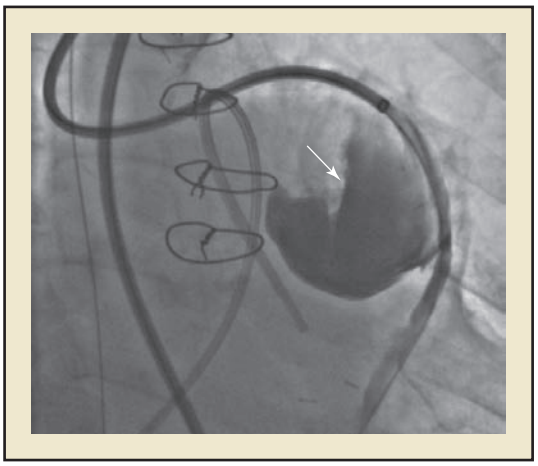

Figure 3. *Contrast can be seen entering the pseudoaneurysm* (arrow) *as it is being injected into the catheter.*

She continued to have intermittent episodes of chest pain; her troponin I level rose to 24.82 ng/mL and remained elevated with fluctuations from 6.46 to 21.37 ng/mL during her 11-day hospital course, indicating ongoing ischemia from unrevascularized myocardium. On hospital day 5, she developed cardiogenic shock with respiratory distress. She was intubated urgently. A pulmonary artery catheter was placed for hemodynamic monitoring. The initial cardiac index was 1.7 L/m^2 with a BP of 105/41 mm Hg, a pulmonary capillary wedge pressure of 25 mm Hg, a mean pulmonary artery pressure of 28 mm Hg, and a pulmonary vascular resistance of 115 dyn·s·cm^{-5}; her systemic vascular resistance was 1836 dyn·s·cm^{-5}. An intra-aortic balloon pump (IABP) was deployed for circulatory support and nitroprusside infusion was administered because of concern for ischemic mitral regurgitation. However, her hemodynamic profile continued to deteriorate with cardiac index falling to 1.4 L/m^2.

Included in the differential diagnosis of possible causes of her acute decompensation was ischemic mitral regurgitation associated with her posterior myocardial infarction (MI), as well as left ventricular failure. However, on transthoracic echocardiogram her ejection fraction was 65% with hyperdynamic function, and the mitral regurgitation was only mild. For more comprehensive assessment, a transesophageal echocardiogram (TEE) was obtained to better assess the cause of the cardiogenic shock. The mitral valve structure was normal and there was only mild to moderate mitral regurgitation. However, the left atrium was noted to be severely compressed to a crescent shape (< 50% of the normal size) from the pseudoaneurysm, which was measured at 8 cm in diameter (Figure 4).

Based on the TEE findings, it was felt that the nitroprusside infusion could be decreasing the preload and afterload in an already underfilled ventricle and therefore it was discontinued. In addition, the IABP was also considered to be contributing to a worsening clinical status possibly by augmenting diastolic graft flow contributing to expansion of the SVG aneurysm. Furthermore, the IABP catheter was compromising arterial circulation of her lower extremities; therefore, the IABP catheter was removed. Doppler ultrasound of her lower extremities revealed thrombus in her right common femoral artery and an emergent left to right femoral bypass surgery was performed to restore blood flow to the ischemic limb.

She continued to have ischemic changes on electrocardiogram and troponin elevation; therefore, it was decided to place a covered stent in the SVG to exclude the aneurysm and eliminate the possibility of thrombi from the aneurysm traveling distally and occluding the coronary circulation. An iCast™ (Atrium Medical Corp., Hudson, NH) 6 mm × 59 mm covered stent was placed in the SVG and it excluded the aneurysm from blood flow, confirmed by angiography (Figure 5). No additional coronary lesions were identified.

The patient continued to deteriorate clinically with hypotension and

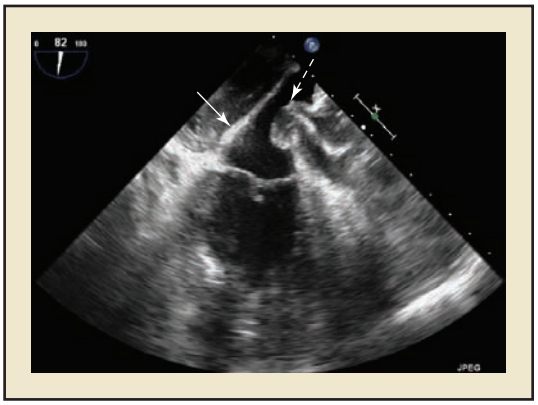

Figure 4. *Transesophageal echocardiogram. Crescent-shaped left atrium* (white arrow) *as a result of severe compression by the giant pseudoaneurysm. A large nonmobile thrombus* (broken arrow) *in the left atrial appendage formed as a result of severe left atrial compression and paroxysmal atrial fibrillation.*

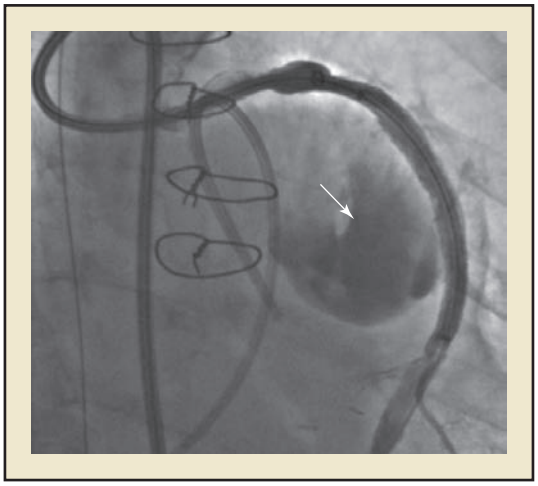

Figure 5. *This image demonstrates closure of the pseudoaneurysm after the stent was deployed; contrast material no longer flowed into the pseudoaneurysm* (arrow).

eventually developed multiorgan failure, likely related to *Clostridium difficile* infection, and she expired after cardiac arrest from ventricular fibrillation.

Discussion

SVG aneurysm is an uncommon complication of CABG that is usually diagnosed 10 to 20 years after surgery.[3-5] Based on a single institution study of 1658 patients with 5579 grafts from 1975 to 1991, the incidence was estimated to be 0.07%.[6] However, this is likely an underestimation because many cases are asymptomatic. Furthermore, SVG aneurysm may be masked on coronary angiography if there is significant thrombus within the aneurysm.[3]

SVG aneurysm can be classified as either true or false aneurysm (pseudoaneurysm). True aneurysm involves all 3 layers of the vessel wall, whereas pseudoaneurysm involves 1 or more layers of vessel wall with a well-defined collection of blood or thrombus outside the endothelium. True aneurysms are typically fusiform and occur at the body of the graft, whereas pseudoaneurysms are saccular and occur at the anastomosis of the graft and typically occur as a result of suture dehiscence or due to technical problems during anastomosis.[7] This patient developed a pseudoaneurysm in the body of the graft, most likely from atheroma formation on the wall leading to vessel wall injury and eventual dilatation with pseudoaneurysm formation.[8,9]

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Clinical presentation differs depending on the type of aneurysm. The majority of true aneurysm are asymptomatic (47%) and often present with a hilar mass on chest radiograph.[10] On the other hand, only a minority (< 15%) of the patients with pseudoaneurysm are asymptomatic. Chest pain from ACS is the most common initial clinical presentation, followed by bleeding, congestive heart failure, hemoptysis, and infection.[3] Symptomatic patients usually present with cardiac events due to graft thrombosis and distal embolization, such as MI, as was true in this case. Compression of surrounding structure, as described, is an atypical manifestation. However, other case reports describe compression of vital structures, including the pulmonary arteries, cardiac chambers, and coronary arteries.[3,11] The aneurysm can fistulize into adjacent structures, and rarely can rupture.[5,7] The natural history of SVG aneurysms is not well defined because they are so rare. The University of Wisconsin School of Medicine and Public Health (Madison, WI) reviewed 15 consecutive cases of SVG aneurysm and compared results of surgical versus conservative therapy. The average time from CABG to diagnosis was similar: 15 and 12.6 years, respectively. Likewise, survival after diagnosis was similar, 1.5 versus 2.3 years, respectively.[6]

Chest pain from ACS is the most common initial clinical presentation, followed by bleeding, congestive heart failure, hemoptysis, and infection.

This report is the first described case of a giant SVG pseudoaneurysm causing a mass-like effect on the left atrium leading to heart failure and cardiogenic shock. The patient had a chest radiograph suspicious of a hilar mass and the subsequent CT angiogram revealed the SVG pseudoaneurysm with mass effect on the left atrium. CT angiography is a good tool for identifying SVG aneurysm and it provided us with information such as continuity of the mass with the SVG, presence of thrombus, differentiation of solid and cystic masses, and mass effect on adjacent structures.[12] The use of coronary angiography is limited in identifying thrombus within the aneurysm but

paramount for identifying coronary artery disease with which most symptomatic patients present. TEE, on the other hand, provided us with information on the anatomic and physiologic effect of the aneurysm on the surrounding structure. It showed us the extent of the mass effect of the SVG pseudoaneurysm on the left atrium. It has also been demonstrated to be useful in other entities[13,14]; in one scenario, contrast imaging was used to identify a left atrial mass as actually an SVG aneurysm.[15]

The optimal management for SVG aneurysm is not well defined. Basically, 3 approaches have been used in the past: medical therapy with surveillance, surgical therapy, and a percutaneous approach.[6] In our case, we achieved total isolation of the pseudoaneurysm percutaneously using a polytetrafluoroethylene (PTFE)-covered stent.[16,17] Coil embolization, vascular plugs,[18,19] and vein graft-covered stents[20] are other percutaneous interventions that have been used successfully to achieve closure of the aneurysm. The 2 percutaneous interventions available to us, coil embolization and PTFE-covered stents, both have their advantages and disadvantages. The coils are technically easier to place, and have been described as successfully thrombosing the aneurysm in a number of case reports. However, they do not exclude thrombus from traveling distally to the native coronary circulation and may result in MI. On the other hand, PTFE stents are difficult to place, particularly because it may be difficult to size and find landing zones proximal and distal to the aneurysm. They are often large and may be sized for other anatomic purposes. For example, the stent used in this case was actually a biliary stent and was not readily available in our catheterization laboratory. Furthermore, pseudoaneurysms may form at sites of SVG-coronary anastomosis, which would make stent placement impossible. In the case just described, the pseudoaneurysm formed in the body of the SVG graft and there was enough vein graft both proximal and distal to the site of the aneurysm, confirmed by IVUS, to allow for stent placement. Additionally, given that the patient had an ACS, the alternative of coil embolization would not have been a good choice, risking further thrombus dislodging downstream. Therefore, the PTFE stent was thought to be the ideal choice. Nevertheless, despite successful stent placement, the combination of sepsis from *C difficile* and low cardiac output from compression of the left atrium made this patient's prognosis poor. Surgery was considered and would have been very high risk because the aneurysm can adhere to vital structures that are thin walled, like the pulmonary artery and atria. Furthermore, adhesions would have made redo-sternotomy technically challenging.

Conclusions

The first case of a giant SVG pseudoaneurysm causing significant compression of the left atrium in a patient presenting with ACS was described. TEE was essential in revealing the cause of cardiogenic shock in this case. However, managing an SVG pseudoaneurysm at such a late stage with concomitant ACS is challenging. Earlier identification and treatment may have prevented the demise of this patient. However, there is no screening practice for SVG graft aneurysm and it is probably not very cost effective because it is a relatively rare complication. Furthermore, given the typical quiescent course of an SVG aneurysm, it is difficult to select patients appropriate for intervention. A percutaneous approach using a covered stent is a viable option to achieve isolation of SVG aneurysm. ■

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Main Points

- Saphenous vein graft (SVG) aneurysm is a rare complication of coronary artery bypass graft surgery that is challenging to manage and is associated with catastrophic consequences.
- Although optimal management for SVG aneurysm is not well defined, 3 approaches have been used in the past: medical therapy with surveillance, surgical therapy, and percutaneous intervention.
- Computed tomography angiography and transesophageal echocardiography are necessary tools for identifying SVG aneurysm.

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