

# Late Spontaneous Rupture of a Saphenous Vein Graft

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**ABSTRACT:** Aortocoronary graft ruptures are a complication of coronary artery bypass grafting. The majority of graft ruptures are iatrogenic and occur after interventional procedures or surgery, whereas graft ruptures are significantly less common. Our case report highlights a clinical presentation of a late spontaneous saphenous vein graft rupture that developed approximately 16 years after initial bypass surgery and captures some of the ensuing complications. Several different imaging modalities were used to diagnose and characterize the lesion, and it was ultimately treated with percutaneous coronary intervention and minimally invasive surgery.

## INTRODUCTION

Coronary artery ruptures of native vessels and aortocoronary bypass grafts are well-documented complications during interventional procedures.<sup>1</sup> However, the occurrence of spontaneous aortocoronary graft ruptures is less common, with an incidence of < 1%.<sup>2-5</sup> We observed such a case in a 65-year-old male 16 years after initial coronary artery bypass grafting (CABG). Despite graft rupture being an uncommon phenomenon, this diagnosis should be included in the differential in patients presenting with chest pain and a history of CABG.

## CLINICAL COURSE

A 65-year-old male presented to an outlying facility with chest pain and a history of coronary artery disease (CAD). He had undergone two-vessel bypass 16 years prior, with left internal mammary artery grafting to the left anterior descending (LIMA-LAD) and saphenous vein grafting to the right posterior descending artery (SVG-rPDA), which required two drug-eluting stents after 7 years. His medical history also included congestive heart failure with reduced ejection fraction (EF) of 20% and an implantable cardiac defibrillator, essential hypertension, mixed hyperlipidemia, and non-oxygen-dependent chronic obstructive pulmonary disease. He reported that his chest pain had been occurring for 3 days prior to presentation and described it as pressure that worsened with exertion, improved with rest and nitroglycerin, and was associated with shortness of breath. He denied any history of trauma to his chest.

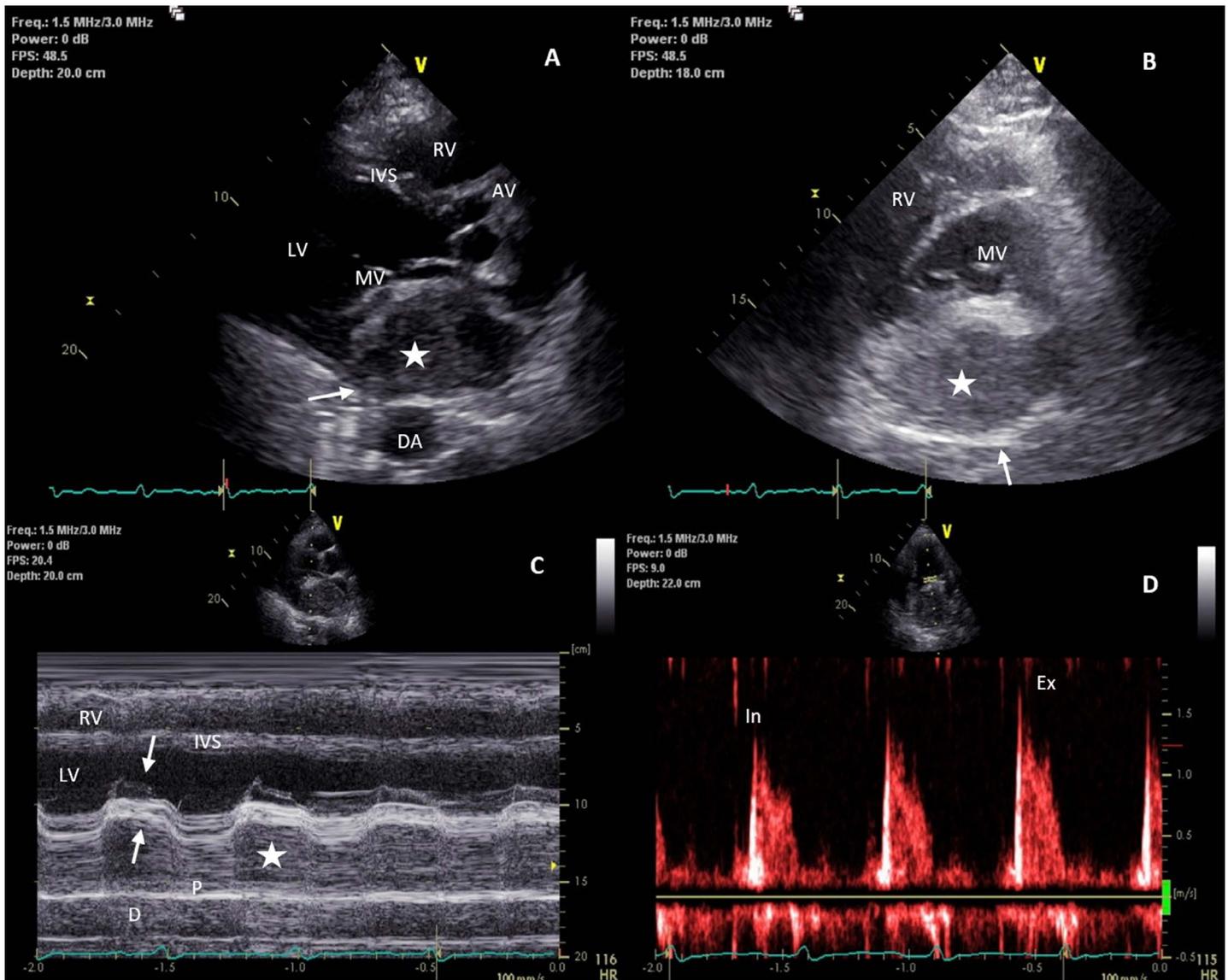
At the outlying facility, his vital signs included a heart rate of 115 beats per minute, respiratory rate of 26 breaths per minute, SpO<sub>2</sub> < 90% on room air, and blood pressure of 117/71 mm Hg. Lab work revealed hemoglobin of 14.6 g/dL, troponin I of 0.26 ng/mL, and BNP of 944 pg/mL. Electrocardiogram (EKG)

revealed sinus tachycardia with inferolateral ST depressions and a nonspecific intraventricular conduction delay that appeared similar to an EKG performed in 2015. The patient received aspirin 81 mg, clopidogrel 75 mg, and a heparin bolus followed by a heparin drip. There was concern of pulmonary embolism due to persisting tachycardia and developing hypoxia with SpO<sub>2</sub> 87%, for which the patient underwent computed tomography angiography (CTA) of the chest. The CTA ruled out pulmonary embolism but revealed a 6.1 × 9.5 × 8.7-cm retrocardiac mass facing the left atrium and pulmonary veins and concerning for hematoma. A CT of the chest performed a year prior showed no evidence of a mass. The heparin drip was discontinued due to concern for a developing hematoma, and the patient was transferred to our facility for evaluation.



*Figure 1.*

Computer tomographic angiography image of the chest revealing a retrocardiac mass (white dotted line) measuring 6.1 × 9.5 × 8.7 cm effacing the left atrium and pulmonary veins. LV: left ventricle; RV: right ventricle; LA: left atrium; RA: right atrium; DA: descending thoracic aorta; AV: aortic valve

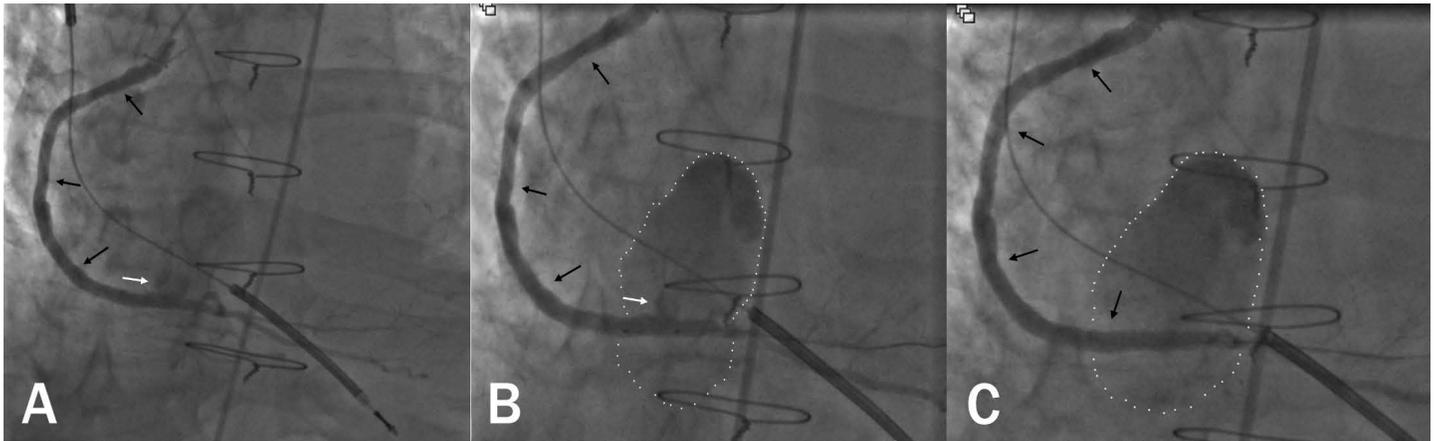


**Figure 2.**

Echocardiographic images of a large posteriorly located intrapericardial hematoma. (A) Parasternal long-access view showing severe compression of the left atrium by a posteriorly located echolucent mass (white star) contained within the pericardium (white arrow). The posterior leaflet of the mitral valve has been severely anteriorly displaced. (B) Parasternal short-access view depicts similar compression of the mitral valve with anterior displacement of the posterior leaflet of the mitral valve by the echolucent mass (white star). (C) M-mode image through the plane of the mitral valve. The image shows the posteriorly located echolucent mass (white star) as well as restricted motion of the posterior leaflet of the mitral valve (upgoing arrow) with normal motion of the anterior leaflet (downgoing arrow). (D) Continuous wave Doppler through the mitral valve showing significant respiratory variation in mitral inflow velocities, with more than 30% variation from inspiration (In) to expiration (Ex) concerning for obstructed flow through the mitral valve. LV: left ventricle; RV: right ventricle; IVS: interventricular septum; MV: mitral valve; AV: aortic valve; DA: descending thoracic aorta; P: pericardium

On arrival, the patient had a heart rate of 121 beats per minute, was tachypneic with 25 breaths per minute, had a blood pressure of 103/80 mm Hg, and his SpO<sub>2</sub> was above 90% on 4L oxygen via nasal cannula, but he continued to report chest pressure. Repeat labs revealed a downward trend in hemoglobin

from 14.6 g/dL to 11.6 g/dL, a rise in troponin I to 6.54 ng/mL, and a BNP of 1308 pg/mL. Repeated EKG did not show significant interval change compared to the outlying facility EKG. Chest x-ray revealed left perihilar infiltrate but otherwise identified no acute cardiopulmonary processes. A repeat CTA of the



**Figure 3.**

Coronary bypass graft angiographic images of the saphenous vein graft (black arrows) to the right posterior descending coronary artery. (A) Angiographic image shows a perforation (white arrow) near the anastomosis of the bypass conduit and the native vessel. (B) Angiographic image post deployment of 2.8 × 16 and 3.5 × 15 mm Graftmaster RX covered stents (Abbott Vascular). Perforation is still noted (white arrow) after deployment of the stent, and a posteriorly located hematoma is also visualized (white dotted lines). (C) Angiographic image post stent deployment and successful balloon tamponade with a 4.0 mm NC Quantum Apex balloon (Boston Scientific). Posteriorly located hematoma is still present (white dotted line).

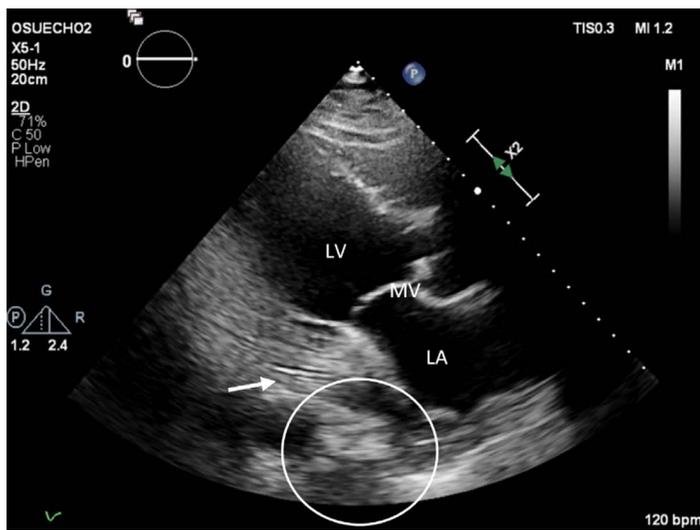
chest was performed to assess interval change of the previously identified retrocardiac mass and revealed enlargement to 6.4 × 11.9 × 9.8 cm that appeared to be affecting the adjacent left atrium (Figure 1). The mass was posterior to the left atrium and hyperdense on the noncontrast portion of the exam; additionally, there was an internal enhancing structure adjacent to the distal posterior bypass graft that was suspicious for a rupture or aneurysm. Echocardiogram revealed a large posteriorly located mass within the pericardium compressing the left atrium and nearly obstructing flow into the left ventricle (Figure 2).

Following the echocardiographic findings, the patient underwent urgent left heart catheterization, and coronary angiography showed similar severe disease of his native coronary arteries. He then underwent saphenous vein graft angiography, which identified a distal perforation of the SVG-rPDA between two overlapping previously placed drug-eluting stents (Figure 3). After an emergent discussion with cardiothoracic surgery, the decision was made to continue with catheter-directed intervention due to the complexity of redo-sternotomy or any invasive surgical procedure in the setting of recent clopidogrel administration. A 6F multipurpose guide catheter was used to engage the SVG-rPDA, and a Hi-Torque Balance Middleweight Universal Guide Wire (Abbott Vascular) and Mailman wire (Boston Scientific) were used as a buddy system to deploy a 2.8 × 16 mm Graftmaster RX covered stent (Abbott Vascular); however, repeat angiography revealed continued extravasation. A 3.5 mm NC Quantum Apex balloon (Boston Scientific) was inserted and inflated to 20 atm, and angiography was repeated to demonstrate that the balloon

was occlusive, but it was unsuccessful at closing the rupture and was removed. A second 3.5 × 15 mm Graftmaster RX covered stent was placed proximal to the previous stent in an overlapping fashion. Angiography was repeated and continued to demonstrate extravasation. Use of an OptiCross intravascular ultrasound catheter (Boston Scientific) demonstrated that the stents were well opposed to the vessel wall. A 4.0 mm NC Quantum Apex balloon was inserted at the level of the perforation and inflated to 19 atms for 5 minutes and then 20 atms for 10 minutes. Repeat angiography showed no further evidence of extravasation from the vein graft (Figure 3).

Following catheterization, the patient moved to the cardiac critical care unit. Despite successful tamponade of the perforated SVG, the patient reported intermittent chest discomfort, lightheadedness, and dizziness. Of note, his vital signs and laboratory values including hemoglobin remained stable. A repeat echocardiogram performed on day four of hospitalization revealed that the previously visualized hematoma was compressing the left atrium, compromising inflow of blood into the left ventricle.

Because echocardiographic findings were suggestive of tamponade, the patient underwent video-assisted thoracoscopic surgery (VATS) and received a pericardial drain and left thoracostomy tube on day five of hospitalization to evacuate the intrapericardial hematoma. Postoperatively, the patient developed new-onset atrial fibrillation with rapid ventricular response. Following three administrations of metoprolol tartrate 5 mg intravenously, the patient's heart rate



**Figure 4.**

Echocardiographic parasternal long-access image following video-assisted thoracic surgery to evacuate a large posteriorly located intrapericardial hematoma; also shows placement of a pericardial drain (white arrow) with relief of the compression on the left atrium and mitral valve. LV: left ventricle; LA: left atrium; MV: mitral valve

improved to between 70 and 90 beats per minute. He was transitioned to metoprolol succinate 25 mg twice daily, which was later increased to 100 mg daily for optimal heart rate control. His symptoms resolved following the VATS procedure, and he received apixaban 5 mg twice daily the night following the procedure. Hemoglobin values remained stable over the remaining course of his hospitalization. A third echocardiogram performed on postoperative day two showed a substantial reduction in the mass impinging on the left atrium and noted resolution of the compromised inflow into the left ventricle (Figure 4). The patient remained asymptomatic and was discharged home in stable condition.

## DISCUSSION

The short- and long-term risks surrounding CABG have declined significantly over the years; however, complications still exist.<sup>1</sup> Immediate vessel complications include anastomotic rupture at the aortocoronary graft site, aortic root dissection at the anastomotic and cannulation sites, and myocardial infarction. Late vessel complications include graft aneurysms, graft rupture, plaque formation within grafts from atheromatous and nonatheromatous processes, and anastomotic site aortic dissection. Aortocoronary graft ruptures, specifically saphenous vein graft ruptures, are rare events seen after CABG and may lead to major complications, including compression of adjacent structure, myocardial ischemia, and even sudden cardiac death.

Ruptured aortocoronary grafts can be diagnosed with CT imaging, transthoracic or transesophageal echocardiography, coronary angiography, or visually during surgery.<sup>1</sup> For our patient, CT imaging initially identified the retrocardiac hematoma, echocardiography further characterized the hematoma and its compressive nature, and coronary angiography confirmed the rupture of the saphenous vein graft and cause of the hematoma.

The exact cause of our patient's ruptured SVG is uncertain. It is worth noting he had two previously placed drug-eluting stents in the SVG-rPDA 9 years prior to presentation. It is possible that stent strut erosion caused the graft rupture, but this could not be confirmed on angiography nor was it visualized during the VATS procedure. Another consideration is that intimal fibrosis occurring over time within the vein wall may have further weakened the vessel wall, and exposure to arterial pressures may have culminated in the eventual rupture.<sup>1,2</sup> Rupture of vascular structures depends on several factors, including wall stress due to blood pressure and wall thickness.

In terms of management, surgical intervention for graft rupture is generally preferred as it allows for definite control of bleeding and repair,<sup>6</sup> and early intervention is preferred due to potential life-threatening complications associated with rupture.<sup>7</sup> Given the complexity of the case and the high risk of blood loss in the setting of recent clopidogrel administration, PCI was performed to avoid the possibility of serious surgical complications. Additionally, while there is utility in performing P2Y12 assays to determine the degree of platelet inhibition and stratify the risk of significant bleeding, this was not feasible since our facility used a send-out lab and the patient's acute presentation necessitated urgent intervention.

Overall, it is worth discussing that the incidence of SVG rupture may be underestimated due to the rarity of the condition and the atypical presentation<sup>4</sup>; in fact, existing literature suggests that rarity is the biggest barrier to timely diagnosis.<sup>2</sup> Angina with a history of CABG is typically assumed to be myocardial ischemia, which highlights the importance of considering SVG rupture on a differential, especially in a post-CABG patient with a new mediastinal mass.

### *Conflict of Interest Disclosure:*

The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

### *Keywords:*

saphenous vein graft rupture, SVG rupture, graft rupture, aortocoronary rupture, SVG perforation, graft perforation, CABG complications, VATS, interventional cardiology

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