A Very Unusual Cause for Presyncope after Bypass: What a Surprise

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INTRODUCTION

Clinical presentation of saphenous vein graft (SVG) aneurysms (SVGAs) is variable.1-4 We present a case of SVGA that caused a patient to have recurrent bouts of presyncope. We review the clinical presentations of SVGAs in addition to the diagnostic and management challenges SVGAs pose. We also highlight the importance of multimodality imaging in the diagnosis of this rare case.

CASE PRESENTATION

History of Present Illness

A 79-year-old man presented to our hospital for management of methicillin-sensitive Staphylococcus aureus bacteremia and cardiac implantable electronic device (CIED) infection. Upon questioning, he complained of multiple episodes of presyncope that had been ongoing for a few months. Further information was fairly limited given the patient’s diagnosis of Alzheimer’s dementia. His vital signs were notable for temperature 36.4°C, blood pressure 160/77 mm Hg, heart rate 57 beats per minute, and respiratory rate 18 breaths per minute. He was in no acute distress, he was alert and oriented × 3, his cranial nerves II-XII were intact, and he maintained 5/5 strength throughout. Upon auscultation of his heart, he was in regular rhythm with III/VI systolic crescendo-decrescendo murmur over the base of the heart.

Medical History

The patient’s history is notable for coronary artery disease status post—coronary artery bypass graft × 2 (SVG left circumflex, left internal mammary artery graft to the left anterior descending artery) over 20 years ago, dual-chamber pacemaker placement for sinus node dysfunction, paroxysmal atrial fibrillation, abdominal aortic aneurysm status post—endovascular stenting, hypertension, Alzheimer’s dementia, and multiple strokes.

Differential Diagnosis

The common cardiac differential diagnosis of presyncope includes structural (hypertrophic obstructive cardiomyopathy), valvular (aortic stenosis), and arrhythmic etiologies (high-degree block, long QT interval, supraventricular tachycardia, and idiopathic ventricular tachycardia). Noncardiac causes include orthostatic hypotension, seizures, vasovagal syncope, and medication induced.

Investigations

Transthoracic echocardiogram (TTE) revealed normal left ventricular size and systolic function with ejection fraction of 63% and normal right ventricular size and function. A large echogenic mass compressing the right ventricular outflow tract (RVOT) and proximal pulmonary artery (PA) was seen adjacent to the ascending aorta on parasternal long-axis images and on parasternal short-axis images (Figures 1 and 2, Videos 1-3). Moderate tricuspid valve regurgitation (TR) was seen with peak TR velocity of 4.2 m/sec and estimated right ventricular systolic pressure of 78 mm Hg (added right atrial pressure of 3 mm Hg based on inferior vena cava size <2.0 cm and collapse of >50%), and Vmax through RVOT was 2.7 m/sec (Figure 3A, 3B). Transesophageal echocardiogram confirmed severely narrowed RVOT with associated turbulence of flow through the RVOT and at the level of the pulmonic valve due to extrinsic compression from echogenic mass. The differential for this mass was broad—notably, aortic aneurysm, pericardial cysts, or tumors such as lymphoma or cysts.
Cardiac computed tomography (CCT) ultimately demonstrated a 7.5 × 6.5 cm partially thrombosed SVGA compressing the main PA (Figures 3-5).

**Management (Medical/Interventions)**

Methicillin-sensitive *Staphylococcus aureus* bacteremia was managed with antibiotics (intravenous cefazolin), and lead extraction was considered for definitive treatment. Device check demonstrated that his underlying rhythm was sinus bradycardia with low pacing burden. Initially, cardiothoracic surgery was consulted for SVGA resection with or without regrafting plus dual-chamber pacemaker extraction. As part of preoperative workup, a left heart catheterization was performed that demonstrated the SVGA and patent left internal mammary artery graft. Cardiac computed tomography scan also revealed that his left internal mammary artery graft was midline, nonmobile, and directly below and adherent to the sternum, significantly increasing the risk for redo surgery, so resection of the SVGA was not recommended by cardiothoracic surgery. Percutaneous coil- ing/occlusion of the SVGA with or without revascularization of that territory was not performed due to procedural risk and technical difficulty. Transvenous CIED extraction alone was recommended and successfully completed by the electrophysiology team without the need for any open surgical intervention. The patient was recommended to adequately hydrate to maintain his volume status and preload and encouraged to avoid preload-reducing medications such as nitroglycerin to prevent near-syncope episodes due to RVOT compression. The patient was eventually discharged on parenteral antibiotics for CIED infection.

**DISCUSSION**

Clinical presentation of SVGA is variable. In the cases in the literature, most common clinical presentations include chest pain (46.4%), shortness of breath (12.9%), and myocardial infarction (7.7%). Syncope or presyncope as a presenting symptom as in our patient was described in only up to 1.9% of cases.1,2 Although a rare cause, it is important to consider SVGA when evaluating the differential diagnosis of chest pain or syncope in a patient with a history of coronary artery bypass graft. In our patient, aneurysm impingement on the RVOT was most likely responsible for this patient’s near syncope, much like acute saddle pulmonary embolism causing RVOT obstruction, right ventricular strain, and right ventricular dysfunction. Physical exam can be helpful in evaluating graft aneurysms. The III/VI crescendo-decrescendo systolic murmur over the base of the heart found on physical exam in our patient was likely explained by the compression of the RVOT at the level of the main PA by the aneurysm. Complications of SVGA were seen in 35.9% of cases.1 A previous series by Ramirez *et al*1 described right atrial compression in 11.5% of cases, followed by aneurysm rupture (8.1%), fistula formation (7.7%), right ventricular compression (7.2%), and PA compression (3.8%).
Figure 3 (A) Two-dimensional TTE image demonstrating the peak TR velocity continuous wave (CW) Doppler profile. (B) Two-dimensional TTE image demonstrating the CW Doppler profile across the RVOT.

Figure 4 (A) This CCT oblique sagittal multiplanar reformatted (MPR) image shows compression of the distal RVOT/main PA. (B) CCT axial MPR image shows the SVGA, central lumen, and peripheral thrombus. (C) CCT oblique sagittal MPR image at the aortic valve shows the partially thrombosed aneurysm of the SVG with compression of the distal RVOT/main PA. LV, Left ventricle; RV, right ventricle.
While history and exam can give clues to diagnosis of SVGAs, multimodality imaging is essential in the diagnosis and evaluation of its complications. An SVGA can present as a mediastinal mass on chest X-ray, and location of the mass on X-ray can sometimes facilitate identification of which graft is enlarged. For example, masses at the right heart border are typically caused by enlargement of vein grafts to the right coronary, while masses at the left lower cardiac border are typically from grafts supplying the left anterior descending coronary. 2 Masses at the left upper cardiac border may be from enlargement of grafts supplying left anterior descending or left circumflex distributions. 2 Nevertheless, additional imaging modalities are required to confirm the diagnosis. 2 Cardiac catheterization (66.5% of cases) and CCT angiography (60.3%) are most widely used, followed by echocardiogram (28.2%) and less commonly cardiovascular magnetic resonance imaging (12.9%). 1 Multimodality imaging is crucially important as single modality alone can have limitations. Several cases of SVGAs appearing as atrial masses on TTE are well documented in the literature. 3-5 This underscores the importance of modalities such as CCT angiography or cardiovascular magnetic resonance imaging in confirming the diagnosis.

Through multimodality imaging, the best management approach can be offered to a patient. In one review, 58.4% of cases were managed surgically (aneurysmal resection or ligation), 15.8% percutaneously (via coil embolization, Amplatzer vascular occlusion, or covered stent placement), and 20.1% conservatively. 1 In our patient, due to increased surgical, percutaneous procedural risk and Alzheimer’s dementia, conservative therapy was pursued. The patient is due to follow up locally.

CONCLUSION

We describe a rare case of recurrent presyncopal episodes that were caused by a large SVGA leading to compression of the RVOT and main PA. Multimodality imaging is essential in diagnosis of SVGA and in determining the best approach to management.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2021.11.008.

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