

HUGE ANEURYSM OF THE SINUS OF VALSALVA COMPRESSING THE LEFT ATRIUM

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Sinus of Valsalva aneurysms are rare cardiac anomalies. They can be congenital or acquired, and mainly involve the right or non-coronary sinuses. Unruptured aneurysms are usually asymptomatic unless they compress other structures or produce thrombi. A sinus of Valsalva aneurysm can also produce myocardial infarction through thrombus formation secondary to the turbulent flow in the Valsalva aneurysm. We report a case of a huge sinus of Valsalva aneurysm involving the non-coronary sinus, which was diagnosed as the presumed source of acute myocardial infarction.

KEY WORDS: Sinus of Valsalva · Aneurysm.

INTRODUCTION

A sinus of Valsalva aneurysm (SVA) can be congenital or acquired. A congenital SVA is more common and most often caused by weakness at the junction of the aortic media and the annulus fibrosus.¹⁾ An acquired SVA is caused by secondary degeneration of the elastic connective tissue that occurs from atherosclerosis or infection (bacterial endocarditis, syphilis, and tuberculosis). Unruptured SVAs usually remain asymptomatic, but sometimes can cause cardiac rhythm abnormalities, myocardial ischemia, and systemic embolic events.²⁾

CASE

A 48-year-old female was admitted through the emergency room with chest pain that developed 5 days earlier and was aggravated 2 days prior to admission. She had suffered frequently from exertional dyspnea and chest discomfort for approximately 1 year. Her heart sounds were regular without murmurs, and crackles were auscultated in both lung fields. Her chest x-ray showed a voluminous heart and diffuse ground glass opacities, suggesting pulmonary edema. Her admission electrocardiogram (ECG) revealed T

wave inversion at V2-6, I, aVL, II, and aVF. The initial creatinine kinase isoenzyme MB (CK-MB) was 8.6 ng/mL and troponin I was 2.1 ng/mL. Together, the symptoms and clinical findings were suggestive of an acute non-ST-segment elevation myocardial infarction. We performed a transthoracic echocardiogram (TTE), then recognized the dilated SVA arising from the non-coronary cusp and compressing the left atrium (7.6×5.8 cm in diameter in an apical 4 chamber view; Fig. 1).

The SVA was also noted on transesophageal echocardiogram (TEE). The SVA extended to the interatrial septum without shunt flow and severe spontaneous echo contrast (SEC) was observed (Fig. 2). A chest CT revealed a huge SVA arising from a non-coronary cusp (Fig. 3). A coronary angiogram showed a dilated SVA and an intermediate lesion on the proximal left anterior descending artery. Resection of the aneurysm and a Bentall operation were performed. Then, her symptoms were improved and she is now symptom-free.

DISCUSSION

A SVA is rare cardiac anomaly from 0.1 to 3.5% of all

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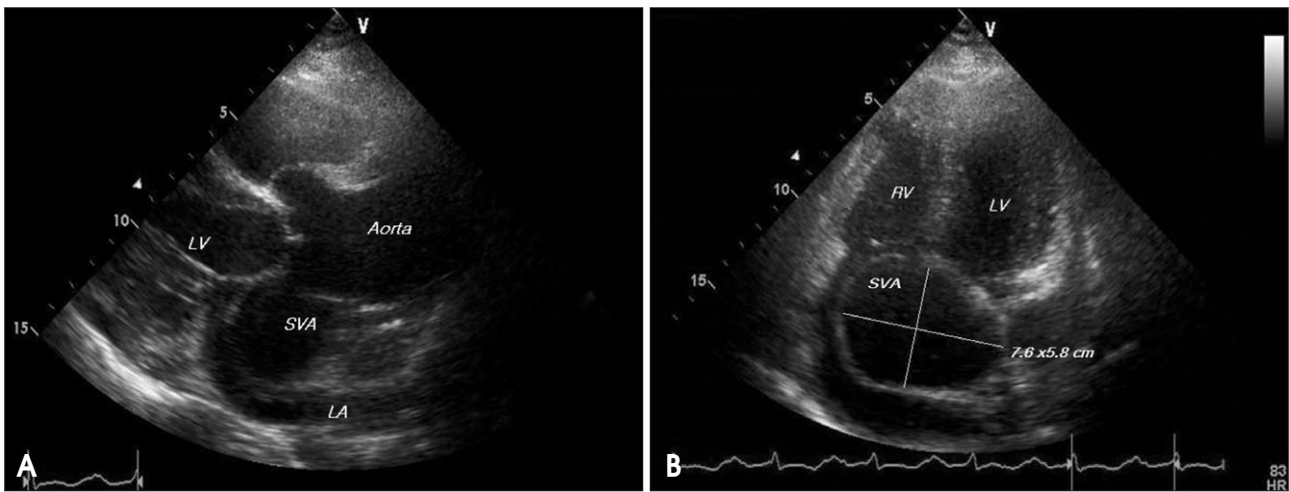


Fig. 1. TTE images of the SVA. A huge SVA (7.6 × 5.8 cm in diameter) is compressing the LA in the parasternal long axis (A) and apical 4 chamber views (B). SVA: sinus of Valsalva aneurysm, LA: left atrium, LV: left ventricle, RV: right ventricle.

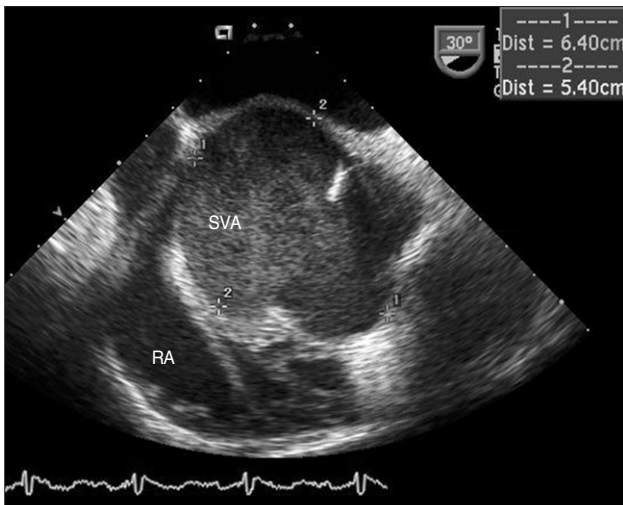


Fig. 2. TEE image of a SVA. The SVA originating from the non-coronary sinus extends to the interatrial septum and severe spontaneous echo contrast is observed in the short axis view. SVA: sinus of Valsalva aneurysm, RA: right atrium.

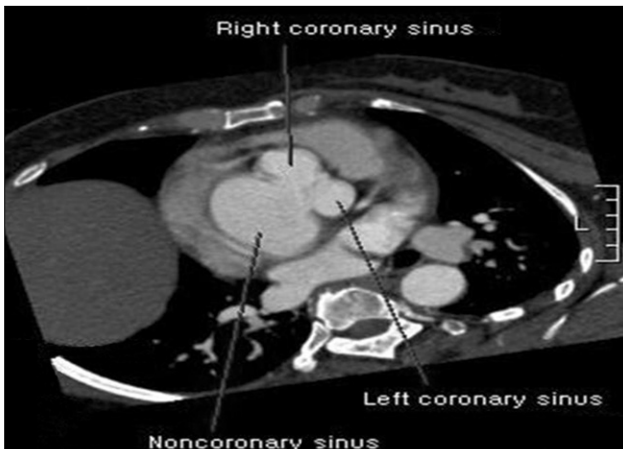


Fig. 3. A huge SVA arising from the non-coronary cusp is seen in the chest cardiac tomography image.

congenital heart defects and 0.14% of all open heart surgical procedures.³⁾ A SVA is defined as a dilatation of one of the three aortic sinuses between the aortic valve annulus and the sinotubular junction or supra-aortic ridge.¹⁾ SVAs originate most commonly in the right coronary sinus or the non-coronary sinus and, rarely, the left coronary sinus.^{2,4)}

The incidence of unruptured SVAs has been reported to be 20%, based on previous reports of necropsy and cardiac surgery findings.¹⁾ Unruptured SVAs are usually asymptomatic, but expanding SVAs can obstruct the right ventricular outflow tract or compress the coronary arteries, causing an acute ischemic attack, and they can cause severe arrhythmias, such as complete heart block, ventricular tachycardia, and atrial fibrillation. Furthermore, systemic embolic events may result from thrombus formation within an SVA.^{5,6)} In our case, we observed severe SEC in the SVA on TEE, even without thrombi, which may have been related to her chest pain by causing microthrombi-induced transient coronary obstruction. On the contrary, some have reported that exertional dyspnea, palpitations, and angina-like chest pain with unruptured aneurysms can develop, although the aneurysm might not be causally related to the symptoms.¹⁾

In unruptured SVAs, ECG findings are usually normal or voltage criteria for left ventricular hypertrophy and ST-T wave abnormalities. Several lead ST-T changes were also observed in our ECG. Currently, TTE is the initial choice to diagnose a SVA. The parasternal long- and short-axis views are considered best to visualize the SVA. Color flow imaging with pulsed and continuous-wave Doppler is useful for ruptured aneurysms.^{1,7)} TEE is the next choice when SVA is suspected, especially when the aneurysm is small and other congenital defects may be present.^{8,9)}

Cardiac catheterization should be used to confirm the diagnosis of the SVA and to evaluate the hemodynamics, the associated cardiac anomalies, and the coronary anatomy. Cardiac MRI and CT are useful for multiple congenital abnormalities and better visualization of the regional anatomy.^{1,2)}

Surgical indications for unruptured SVA include the production of malignant arrhythmias, obstruction of the coronary ostia or ventricular outflow tracts, or infection. Progressive enlargement of an aneurysm on serial evaluation should be considered as an indication for surgery as well.⁶⁾ For asymptomatic unruptured SVAs, although some argue that monitoring is the best approach, immediate SVA repair is usually best because surgery generally produces excellent outcomes in such cases.^{10,11)} Surgical corrective procedures include simple patch repair, patch repair with aortic root replacement, aortic valve repair or replacement, or reimplantation of the coronaries. The perioperative death rate is around 3.9%, mostly in the setting of preexisting sepsis or endocarditis.⁵⁾

In our case, the huge SVA compressed the left atrium, and severe SEC was observed during the TEE. Although we could not find a thrombus on coronary angiogram, transient thrombus formation may have been the cause of her clinical manifestations. We have reported a huge SVA arising from the non-coronary sinus which might be associated with myocardial infarction, and reviewed the literature on SVA.

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