



CASE REPORT

A Giant Sinus of Valsalva Aneurysm in a Patient with Aortic Valve Replacement

Sefik Gorkem Fatihoglu^{1*}, Sercan Okutucu²

1 Akhisar Mustafa Kirazoglu State Hospital,
Department of Cardiology, Manisa, Turkey

2 Lokman Hekim University, Department of
Cardiology, Ankara, Turkey

*Corresponding Author: Sefik Gorkem Fatihoglu,

Manisa Merkezefendi State Hospital, Department
of Cardiology Manisa/Turkey, P.O: 45120
Phone: +90 236231 45 87
Phone: +90 533 7229620
Fax: +90 236 234 60 26
E-mail: sgfatihoglu@gmail.com

Abstract

Sinus of Valsalva aneurysms are rare cardiovascular problems that may be acquired or congenital. Herein, we presented a 67-year-old female patient with a giant sinus of Valsalva aneurysm, years after aortic valve replacement surgery. Although small-sized aneurysms without rupture in asymptomatic patients may be followed; ruptured or complicated cases require intervention, and surgery is the gold standard therapy.

Introduction

Sinus of Valsalva aneurysms (SVAs) are rare cardiovascular problems that may be acquired or congenital. Acquired SVAs are caused by conditions affecting the aortic wall, such as trauma, infections, or connective tissue disorders. Rarely, iatrogenic causes, such as surgical trauma to the aortic wall during aortic valve replacement (AVR), might be the etiology of SVAs. SVAs are usually asymptomatic until rupture, and it most commonly involves the right or non-coronary sinuses.^{1,2}

Case Report

A 67-year-old female was admitted to the hospital with complaints of exertional dyspnea. Past medical history was unremarkable except for AVR 15 years ago (due to severe aortic insufficiency). She had been using her medical therapy based on warfarin and metoprolol. Sinus rhythm and flattened T waves in the leads of V4-V6 were seen on her electrocardiogram. On her physical examination, heart sounds were rhythmic, and systolic murmur was auscultated with metallic valve sound on the right second intercostal region. Standard echocardiographic

imaging and real-time 3D-transthoracic echocardiography study demonstrated a giant SVA measuring 103 × 111 mm in diameter (Figure 1). The prosthetic heart valve in the aortic position had normal function. Her left ventricular systolic ejection fraction, right ventricular diameters, and functions were normal. Mild left ventricular diastolic dysfunction was present. The patient was referred for surgical repair of the lesion. The surgery was performed with a median sternotomy. After the pericardium was opened, a giant aneurysm was seen. Aortic and venous cannulation was applied, and cardiopulmonary bypass was started. Aorta was cross-clamped, and the heart was arrested with isothermic blood cardioplegia. The aneurysm was opened. A new non-coronary sinus was created with a xenograft pericardial patch sutured between the annulus and the ascending aorta. The remained aneurysm tissue was closed over the neo-sinus. Cross clamp and cardiopulmonary bypass times were 26 minutes and 40 minutes respectively. The patient recovered uneventfully after the operation and was discharged on the 14th postoperative day. Postoperative echocardiographic images are shown in Figure 2.

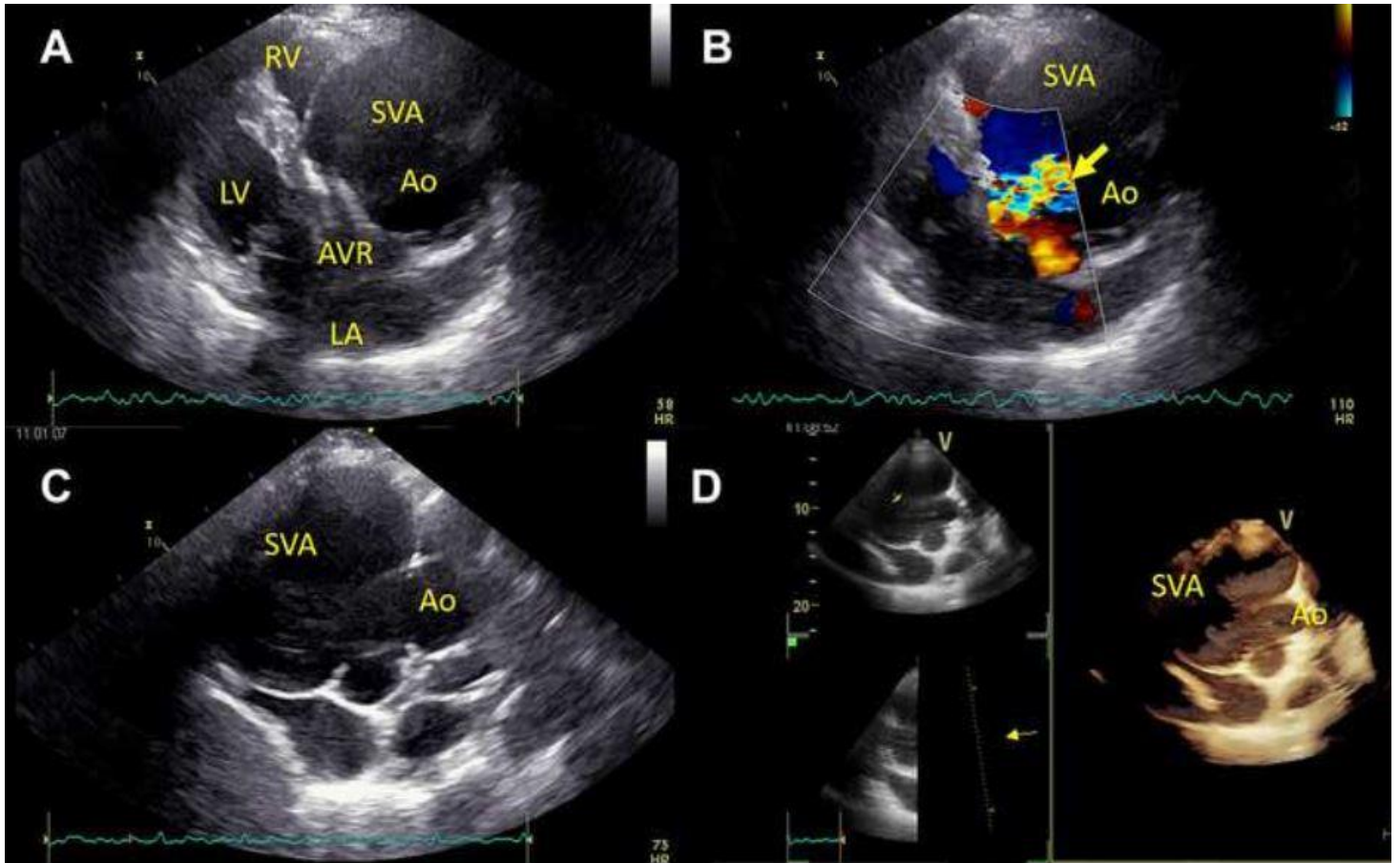


Fig.1: Echocardiographic images of the patient. A. Transthoracic echocardiography demonstrating SVA on parasternal long axis, B. Color Doppler imaging demonstrating outflow from AVR and relation with SVA on parasternal long axis, C. Image demonstrating SVA on parasternal short axis, D. Real-time 3D-transthoracic echocardiography demonstrating SVA. Ao, Aorta; LA, left atrium; LV, left ventricle; RV, right ventricle; SVA, sinus of Valsalva aneurysm.

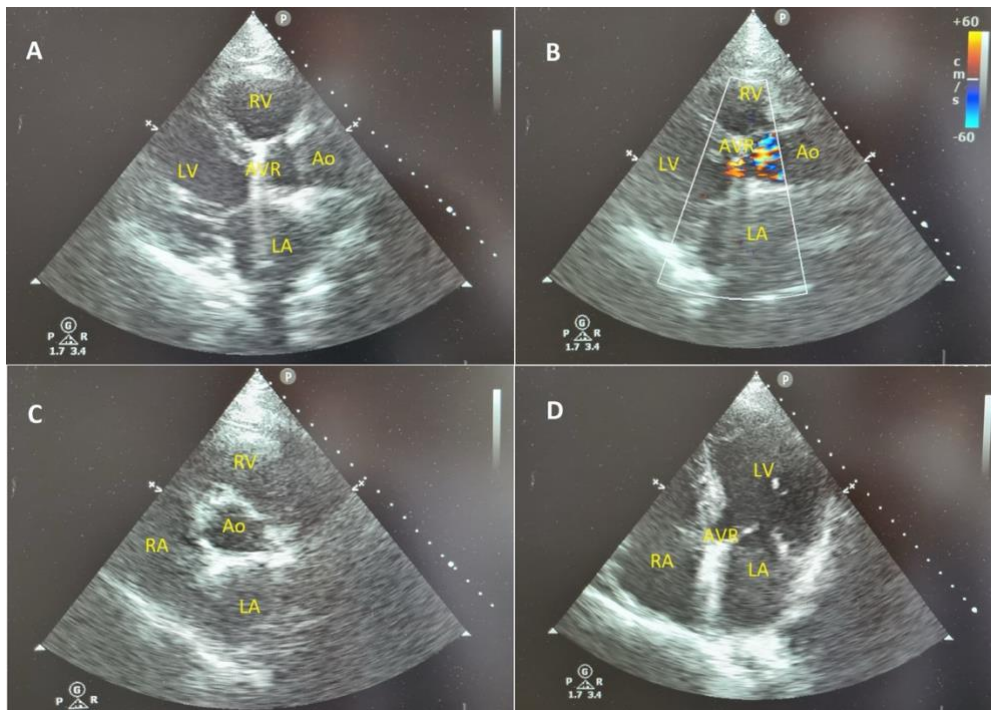


Figure.2: Postoperative transthoracic echocardiographic images of the patient. A. Parasternal long axis 2D image, B. Parasternal long axis color Doppler image, C. Parasternal short-axis image, D. Apical four-chamber image. Ao, Aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.

Discussion

A sinus of Valsalva aneurysm (SVA) is defined as an enlargement of the aortic root area between the aortic valve annulus and the sinotubular ridge. By adjusting for body surface area, the upper limit of normal for men is 4.0 cm and 3.6 cm for women. 3. Based on the autopsy series, the incidence of sinus of Valsalva aneurysms is 0.09% 3. SVAs comprise 0.1% to 3.5% of all congenital cardiac defects and usually affect the right coronary sinus, followed by the noncoronary sinus and the left coronary sinus 4.

The etiology of SVA may be congenital or acquired. Congenital causes are usually secondary to connective tissue disorders such as Marfan's syndrome or Ehler-Danlos syndrome. Acquired cases may result from infectious agents affecting the sinuses such as bacterial endocarditis, syphilitic or tuberculous infections. Elastic tissue weakening resulting from these infectious diseases underlies the pathophysiology of SVA. Chest trauma, vasculitic diseases, and iatrogenic injury during aortic valve replacement have been reported as causes of acquired SVA 3,5. Our patient did not define a particular chest trauma and did not have any congenital cardiac defects or collagen vascular pathologies which may lead to the condition. So, a history of valvular surgery seemed to be the cause of the pathology.

While most unruptured SVAs are asymptomatic, patients may present with dyspnea, palpitations, or angina. Arrhythmia, atrial fibrillation, or heart block may be rarely seen. Thrombus formation may be found in large SVAs, and this may lead to ischemic heart disease by obstruction of coronary

arteries 3,4. Ruptures typically occur between 20 and 40 years of age. According to the speed, size, and location of rupture, patients may present with chest pain, abdominal pain, dyspnea, acute heart failure, cardiac tamponade, hemodynamic compromise, and even sudden cardiac death 3,6. Echocardiography is useful to visualize the aortic sinuses and aortic valve. Our case is a good example of identifying the properties of an aneurysm and aortic valve. Although it is not required in most cases, cardiac magnetic resonance imaging is accepted as the gold standard diagnostic tool. Computed tomography angiography clearly presents the coronary pathologies and the structure of the aneurysm 3,7. Surgical intervention is recommended for a ruptured SVA. Patients with SVA and intracardiac abnormalities such as ventricular septal defect or significant aortic valve regurgitation also need surgery. An unruptured but symptomatic or enlarging SVA should also be considered for surgical repair 3.

References

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