EJCRIM
European Journal
of Case Reports
in Internal Medicine

A RARE CAUSE OF HEART FAILURE IN A YOUNG FEMALE: RUPTURE OF A SINUS OF VALSALVA ANEURYSM INTO THE RIGHT ATRIUM

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Received: 02/06/2025
Accepted: 09/06/2025
Published: 18/06/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Written informed consent for publication was obtained from the patient.
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How to cite this article: Bigdelu L, Maadarani O, Azari A, Bitar Z. A rare cause of heart failure in a young female: rupture of a sinus of Valsalva aneurysm into the right atrium. *EJCRIM* 2025;**12**:doi:10.12890/2025_005582

ABSTRACT

The aortic sinuses of Valsalva are small pouches typically located above the aortic valve and play a crucial role in allowing the aortic valve leaflets to move freely and in supporting the valve itself. Enlargement and dilatation of one or more of these sinuses may result in aneurysm formation that can be complicated with either compression-like effects or spontaneous rupture. A sinus of Valsalva aneurysm (SVA) is a rare cardiac anomaly. We present a case of acute heart failure in a young female caused by spontaneous rupture of an SVA that was diagnosed by echocardiography and required surgical treatment.

KEYWORDS

Sinus of Valsalva, aneurysm, rupture, heart failure

LEARNING POINTS

- Sinus of Valsalva aneurysm is a rare cardiac anomaly that can be either congenital or acquired and usually remains silent until rupture, which is considered a life-threatening condition with a high mortality rate.
- Echocardiography is a diagnostic tool that plays a crucial role in arousing suspicion for and confirming the anatomical connection between the ruptured sinus and the cardiac chamber. Timely diagnosis is critical, and management depends on the aneurysm's size, location, and whether rupture has occurred.
- Asymptomatic cases may be monitored, while symptomatic or ruptured aneurysms generally require surgical repair or transcatheter intervention to prevent catastrophic outcomes.

INTRODUCTION/BACKGROUND

Aortic sinuses (sinuses of Valsalva) function to permit aortic valve opening during systole without the occlusion of coronary artery ostia. Sinus of Valsalva aneurysm (SVA) refers to abnormal dilatation of the sinus of Valsalva and is a cause of thoracic aortic dilatation. SVA can be either congenital or acquired. A congenital SVA is typically identified in younger individuals, whereas the acquired type tends to present in older adults. An acute heart failure presentation in a young population should raise suspicion

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of rare conditions like rupture of SVA (RSVA), where the mortality rate is high without prompt treatment.

CASE DESCRIPTION

A 30-year-old female presented to the emergency department with acute onset dyspnoea and chest pain for one day. There was no relevant past medical history or significant family history. She denied smoking, alcohol consumption, or recreational drug use. There was a negative history of allergies. Her vital signs included a blood pressure of 110/70 mmHg, a pulse rate of 105 beats per minute, a respiratory rate of 20 breaths per minute, and an oxygen saturation of 94% on room air. Her physical examination was remarkable for jugular venous distension extending to the mandible and a continuous machinery murmur, best heard at the left parasternal border during cardiac auscultation. Lung auscultation revealed bilateral basal fine inspiratory crackles. The remainder of the examination was unremarkable.

The electrocardiogram showed sinus tachycardia with no acute or chronic ischemic changes. Blood workup, including a metabolic panel, complete blood count, D-dimer, and troponin, was normal, apart from an elevated plasma N-terminal pro-brain natriuretic peptide (NT-proBNP) level at 4000 pg/ml. A chest X-ray revealed mild bilateral interstitial infiltrates, consistent with pulmonary congestion, without cardiomegaly. A transthoracic echocardiogram (TTE) was performed and demonstrated an ejection fraction of 65%, borderline to mildly dilated right atrium (RA) and right ventricle, with diastolic flattening of the interventricular septum suggestive of volume overload. On short-axis and four-chamber apical views, a dilated structure was seen at the level of the aortic leaflets and left ventricular outflow tract. Colour flow Doppler revealed significant turbulent flow originating from the left ventricular outflow tract and flowing into the right-sided chambers. A systolic-diastolic flow was detected on continuous wave Doppler *(Fig. 1, Video 1 and 2)*. Patent ductus arteriosus, coarctation of the aorta, and an aortopulmonary window were ruled out. To delineate the exact nature of the dilated structure and determine the origin of shunting flow, a transoesophageal echocardiogram (TEE) was performed and showed evidence of trileaflet aortic valve with an aneurysm of noncoronary aortic sinus with windsock appearance directed toward the RA and colour Doppler revealed a significant turbulent flow from the aneurysm of noncoronary sinus into the RA confirming the diagnosis of rupture of aneurysm of the noncoronary sinus into the RA with significant left to right shunt *(Fig. 2, Video 3-5)*. There were no associated atrial or ventricular septal defects or other congenital anomaly. The diagnosis of rupture of the noncoronary sinus of Valsalva into the RA with a significant left-to-right shunt and volume overload was established as the cause of acute heart failure presentation in this young patient. The cardiothoracic team was consulted, and the decision was to proceed with immediate surgical intervention. The patient underwent surgery with excision of the aneurysm followed by pericardial patch repair to close the communication between the ruptured noncoronary sinus and the RA. The patient had an uneventful postoperative

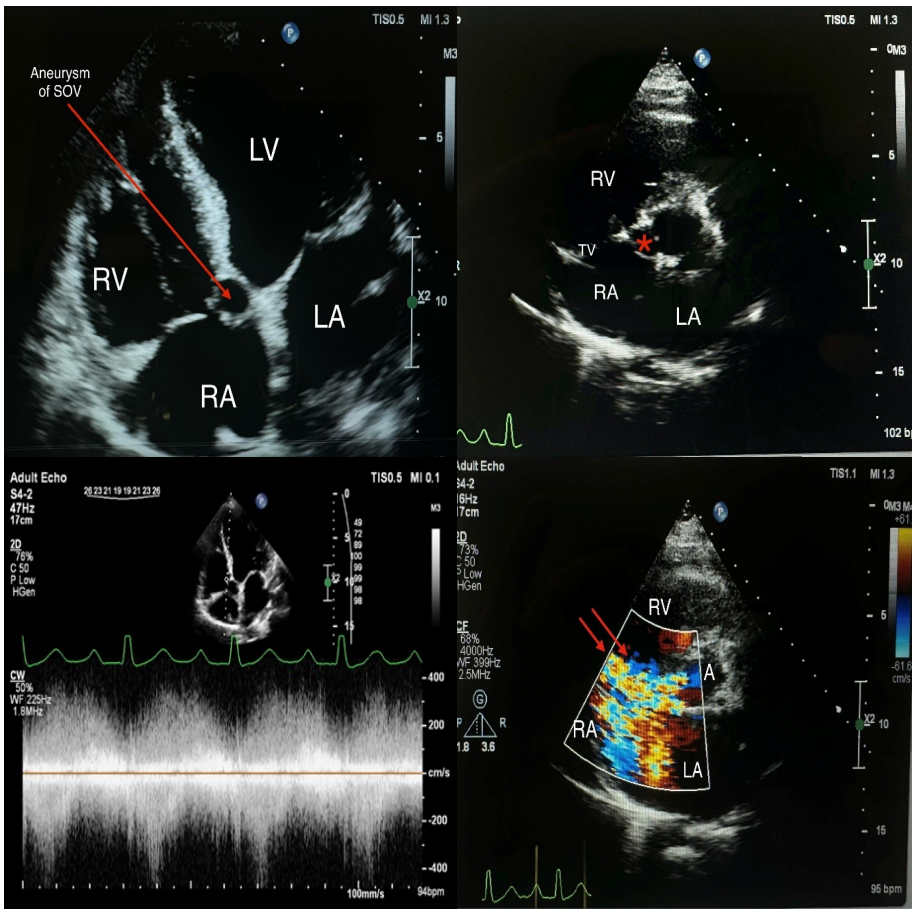
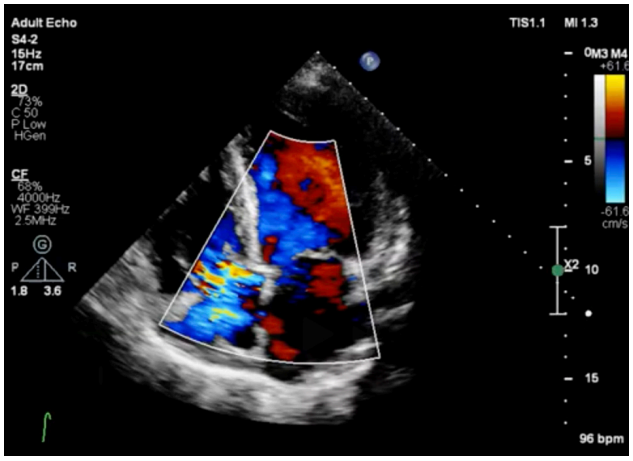


Figure 1. Transthoracic echocardiography: 4 chamber apical view and short axis view showed aneurysmal dilatation of the SOV (long red arrow and star) whereas continuous wave Doppler revealed systolic-diastolic flow and color Doppler showed significant turbulent flow directed into the RA (2 short red arrows).

Abbreviations: LA, left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle; SOV, sinus of Valsalva.



Video 1. Transthoracic echocardiography. 4 chamber apical view showing dilated sinus of Valsalva.
https://youtu.be/3N57P0rBqiY



Video 2. Transthoracic echocardiography. 4 chamber apical view with color Doppler showing turbulent flow from left ventricle out flow tract into right sided chambers.
https://youtu.be/yduhyVeCkI4

period and was discharged home. At routine outpatient 4-week follow-up, she reported no further symptoms and on physical examination there was no continuous murmur. Repeat TEE revealed successful repair.

DISCUSSION

SVA is a rare cardiovascular anomaly characterized by outpunching of one or more of the three aortic sinuses that are located between the aortic annulus and sinotubular junction, caused by weakness in the elastic lamina at the junction of the aortic media and the annulus fibrosus. The estimated incidence of SVAs in the general population is 0.09%[1]. The percentage of SVAs among all congenital cardiac anomalies is up to 3.5%[2]. This rare condition can be congenital in origin and associated with other cardiac anomalies such as bicuspid aortic valves and septal defects or acquired as a result of previous cardiac surgery, trauma, infection (syphilis, tuberculosis, and infective endocarditis), or connective tissue disorder (Marfan syndrome and Ehlers–Danlos syndrome). SVAs occur in approximately 0.2% to 0.9% of patients who have had cardiac surgery. The congenital type of aneurysm arises from the abnormal development of the bulbus cordis during embryogenesis, resulting in subsequent structural weaknesses in the aortic wall. The highest incidence has been observed among Asian populations, suggesting a genetic predisposition[3], with a male-to-female predominance ratio of 4:1[4]. The upper limit of normal of aortic sinuses for men is 4 cm and 3.6 cm for women, with slight variations when adjusted for body surface area[5]. A SVA is diagnosed when the diameter of the aortic sinus exceeds normal limits. Established guidelines for imaging-guided diagnosis are lacking. While echocardiography is the primary diagnostic tool for SVAs, some controversies exist regarding the specific imaging techniques and interpretation, particularly in complex or atypical cases. A comprehensive approach involving both TTE and TEE, along with other imaging modalities as needed, is recommended for accurate diagnosis.

Usually, SVAs are isolated and most commonly occur in the right coronary sinus (70%), however, in rare cases, the aneurysms may present in two or three sinuses. SVA often remains clinically silent until rupture, which is considered the most critical complication of SVA. SVA ruptures in 35%, leading to acute symptoms in one fourth of the patients[6] which makes the diagnosis of this condition and subsequent management important.

Based on the position and location of the aortic sinus aneurysm, the rupture can happen in different adjacent chambers. Anatomically, the right, left, and non-coronary sinuses are adjacent to the interventricular septum, left ventricular free wall, and interatrial septum, respectively. The consequences of rupture are usually based on the anatomical location of the aneurysm[7]. The thin wall of the enlarged aneurysm can eventually rupture, with subsequent blood spilling into adjacent cardiac chambers or the pericardial space, forming an abnormal communication between the aorta and cardiac chamber, leading to hemodynamic instability and heart failure.

Typically, the aneurysm rupture of the right and non-coronary sinuses leads to a left-to-right shunt due to the communication between the aorta and either the right atrium or the right ventricular outflow tract, which can result in right-sided ventricular overload. Moreover, the clinical picture of a rupture of a left coronary sinus aneurysm is less pronounced due to communication with the left atrium or left ventricular outflow tract without left-to-right shunting[8]. In the case of a sizable aneurysm, it may distort the aortic valve, causing chronic aortic regurgitation with subsequent volume overload and heart failure.

The majority of patients with intact SVA are asymptotic and the pathology is usually discovered incidentally during imaging requested for other reasons. Some patients may experience symptoms due to the pressure effect of an aneurysm on adjacent structures or the cardiac conduction system. The devastating complication of SVA is rupture that can clinically manifest with sudden severe tearing or ripping chest pain, sudden shortness of breath due to a rapid onset

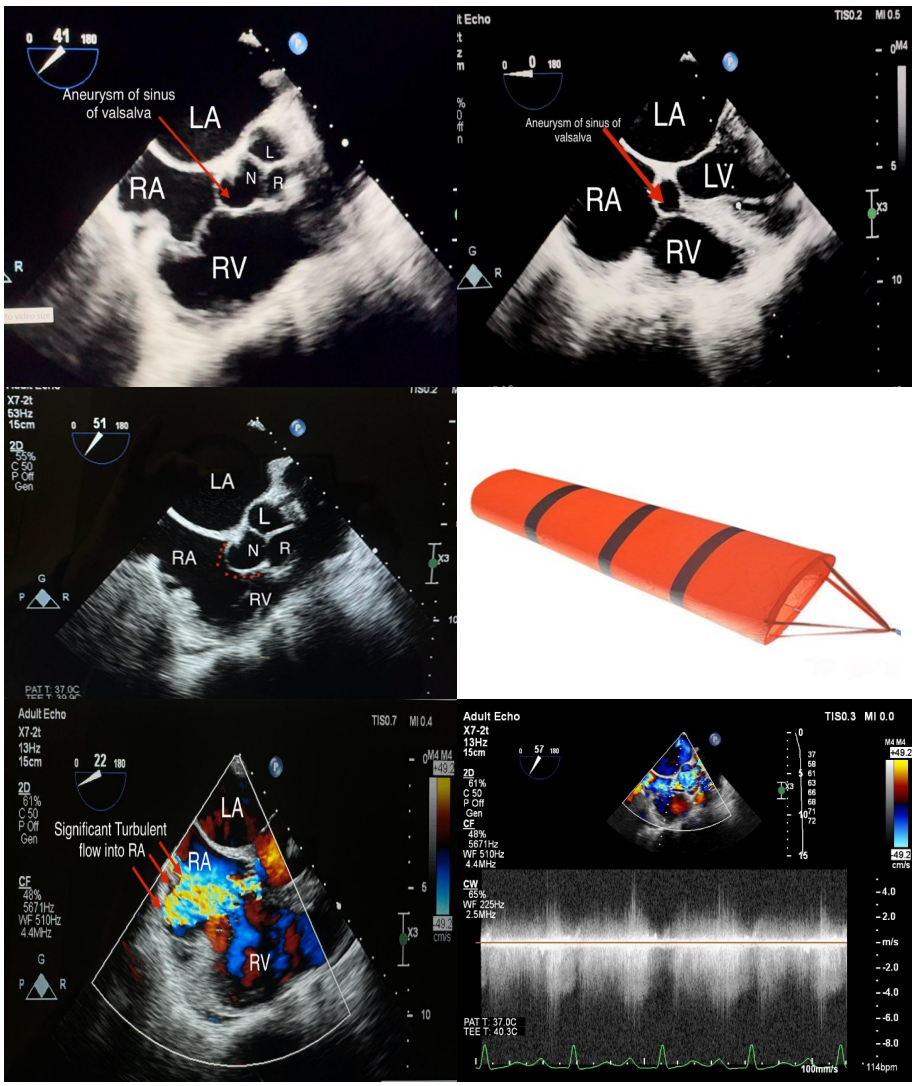
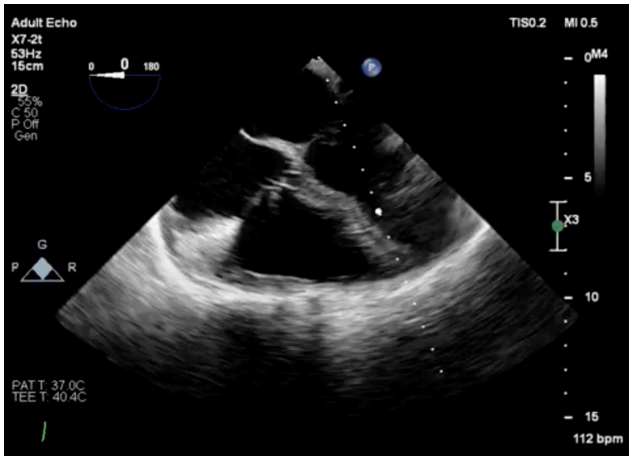


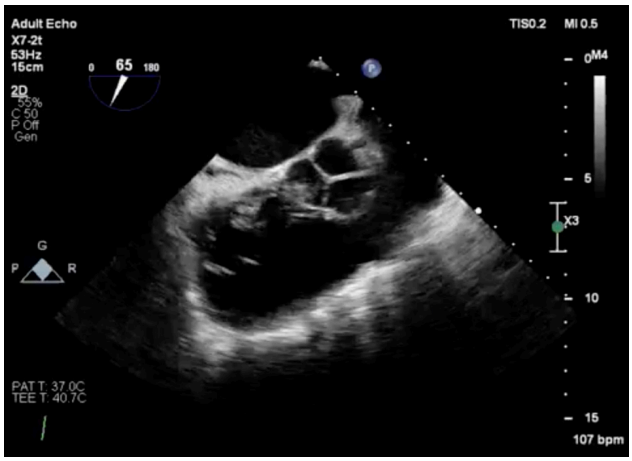
Figure 2. Two-dimensional transesophageal echocardiography in mid-esophagus in different views showing the aneurysmal dilatation of the non-coronary sinus of Valsalva (long red arrow) with a windsock appearance (red dots) and significant turbulent flow into the RA (3 short red arrows) denoting significant left to right shunt from the non-coronary sinus of Valsalva into the RA as a result of rupture. Continuous wave Doppler revealed the persistence systolic diastolic flow.

Abbreviations: RV, right ventricle; RA, right atrium; LA, left atrium; LV, left ventricle; R, right coronary cusp; N, non-coronary cusp; L, left coronary cusp.

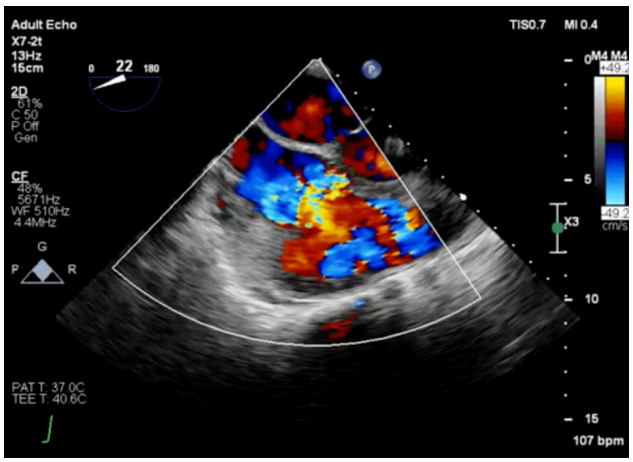
of heart failure or fainting or loss of consciousness due to severe hemodynamic instability. A continuous murmur on auscultation is an important physical sign, especially if it results from a rupture that creates communication between the aorta and the right atrium or ventricle, and it is best heard along the left sternal border. Other physical findings could be the signs of heart failure. Both TTE and TEE play an essential role in detecting pathological dilatation of sinuses, anatomical characteristics, and hemodynamic consequences, in addition to providing information on the function of adjacent structures and possible associated defect anomalies. Echocardiography also helps in ruling



Video 3. Transesophageal echocardiography. Mid esophageal 4 chamber view at 0 degree demonstrating the aneurysm of Valsalva.
https://youtu.be/FQN0Cr2EGGQ



Video 4. Transesophageal echocardiography. Mid esophageal right ventricle inflow-outflow view at 65 degrees demonstrating the aneurysm of the non-coronary sinus of Valsalva directed toward the right atrium.
https://youtu.be/8Ouz0z_ZAjE



Video 5. Transesophageal echocardiography. Mid esophageal short axis aortic valve view at 22 degrees with color Doppler demonstrating significant turbulent flow from the ruptured aneurysm of the non-coronary sinus into the right atrium.
https://youtu.be/I7A62n3dzyk

out other diagnoses as a cause of heart failure. Colour Doppler can reveal continuous flow during both systole and diastole in cases of ruptured SVA. Computed tomography angiography is another imaging modality that offers high-resolution images of the aortic root and sinuses of Valsalva. It is considered the test of choice for quantifying the size and morphology of the SVA and is particularly useful for surgical planning and assessing the relationship of the aneurysm with surrounding cardiac and vascular structures. Cardiac magnetic resonance imaging is considered the gold standard for diagnosis; however, it is not required if other imaging modalities sufficiently provide the diagnosis and pertinent anatomic and physiologic details[9].

Cardiac catheterization and angiography is an invasive procedure that provides detailed images of the coronary arteries and aortic root and can help assess the hemodynamic impact of the aneurysm. The management plan of SVA includes medical treatment and surgical repair. Medical management of SVA is essentially a temporary measure to stabilize patients until definitive surgical or transcatheter intervention can be performed, as it is insufficient for definitive treatment. Patients diagnosed with unruptured SVA should undergo regular monitoring with imaging modalities, such as echocardiography, to track the size and progression of the aneurysm. Additionally, strict blood pressure control and avoidance of strenuous physical exertion and heavy lifting are recommended to prevent increases in intrathoracic pressure that could precipitate rupture. Medical stabilization is critically important in cases of rupture SVA, and it includes resuscitation with fluid and management of heart failure and shock until a definite surgical procedure can be performed.

The surgical treatment of a patient with an unruptured SVA should be strongly considered in cases of significant symptoms or large, rapidly growing aneurysms to prevent rupture. It is mandatory for all patients with ruptured aneurysms due to the high mortality risk[10]. The surgical management includes an open surgical repair with aortic root replacement using a synthetic graft (with a valve-sparing technique or valve replacement if the aortic valve is affected) or, alternatively, a patch repair, which involves reinforcing the aneurysm with a patch to preserve native aortic tissue, typically performed for less extensive aneurysms. In the last few years, a minimally invasive approach with transcatheter closure had been used to treat ruptured and unruptured aneurysms. This approach offers a viable alternative to traditional open-heart surgery, especially for high-risk surgical candidates due to age, comorbidities, or other factors.

Our case highlights a rare cause of acute heart failure at a young age and the role of echocardiography as a diagnostic tool. RSVA should be considered in case of acute heart failure symptoms in a young patient in the absence of any medical history or risk factor, especially when associated with continuous heart murmur on physical examination.

CONCLUSION

RSVA is a rare cause of acute heart failure which is considered a life-threatening condition with high mortality and should be kept in mind when other causes are ruled out.

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