https://doi.org/10.3329/cardio.v15i2.65635

Case Report

Ruptured sinus of Valsalva aneurysm: A Case Report

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Key Words : *Sinus of Valsalva aneurysm, echocardiography, ruptured sinus of Valsalva aneurysm.*

Abstract:

One of the rare cardiac anomaly is Sinus of Valsalva aneurysm (SVA) that can rupture spontaneously into other cardiac chambers or the pericardial space.[1] Here, we report a rare case of a right coronary sinus of Valsalva aneurysm with rupture into the Left ventricular outflow tract (LVOT).

(Cardiovasc j 2023; 15(2): 182-184)

Introduction:

Sinus of Valsalva aneurysm (SVA) is a rare cardiac anomaly of the coronary sinuses which is characterized by absence of elastic tissue between the aorta and the annulus fibrosis.[2] One or more of the aortic sinuses at the aortic valve and sino-tubular junction may be dilated or enlarged.[3] It can be congenital or acquired. SVAs are found in 0.09% of the general population and 0.15%-1.5% of patients of heart surgery.[4] Typically more men are affected than women (4:1) and more seen among Asians.[5] SVAs account for 0.1%-3.5% of all congenital cardiac defect.[6] In this report we present a rare case of right coronary SVA ruptured into the LVOT with acute heart failure.

Case presentation:

A 41 years old gentleman normotensive, nondiabetic presented with sudden onset of breathing difficulty and uneasiness for 3 days. He did not report any history of heart disease. He also had no history of fever or chest trauma. On examination, he was afebrile, heart rate- 110 beats/min, regular and bounding in character, blood pressure- 100/50 mm of Hg, respiratory rate- 18/min, SPO2- 98% on room air. Patient was conscious, oriented, with mild basal creps on chest and loud grade V continuous murmur heard over entire precordium. ECG showed sinus tachycardia. Chest x-ray showed mild lung congestion, trans thoracic echocardiogram showed- Aneurysmal dilatation of right aortic sinus of Valsalva rupturing into LVOT (at level of basal IVS) resulting in to and fro flow between aorta and LVOT. Noncoaptation of aortic valve cusps with severe AR. Holodiastolic flow reversal seen in proximal descending aorta. Tethering of mitral leaflets seen with fluttering of AML because of eccentric jet of AR causing severe secondary MR. Moderate TR- RVSP-58 mm hg, TV annulus- 4.0 cm. IVC is dilated-2.2 cm with >50% inspiratory collapse. Global hypokinesia of LV (LVEF-30%) with moderate LVH. Normal RV systolic function (TAPSE-1.9 cm). Grade I diastolic dysfunction. Coronary angiogram showed normal coronaries and confirmed the aorto-LVOT fistula with shunt.

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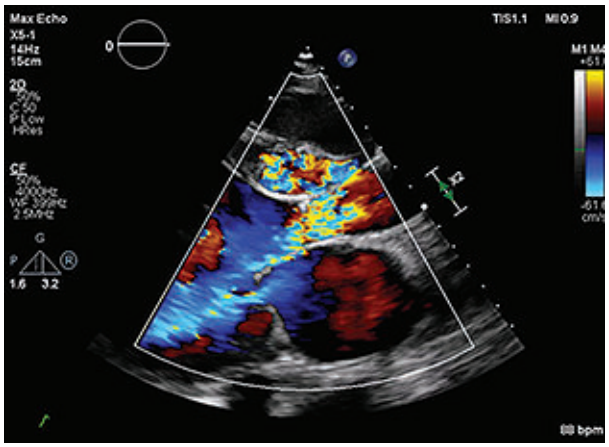

Fig.-1: *Echocardiography with colour doppler showing a high velocity multicolored mosaic of blood flow from right sinus to LVOT.*

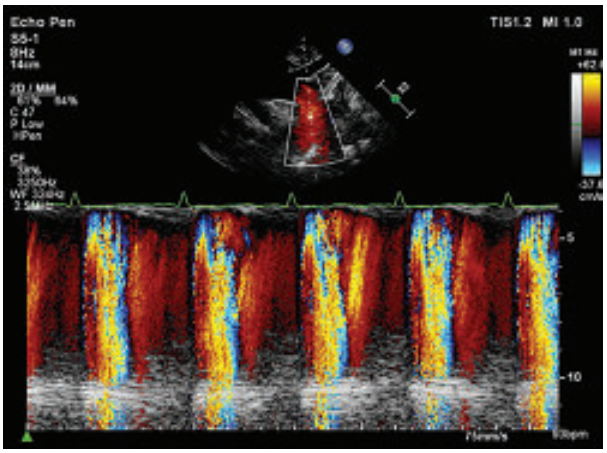

Fig.-2: *Transthoracic echo in suprasternal view with colour doppler showing diastolic flow reversal in the descending thoracic aorta.*

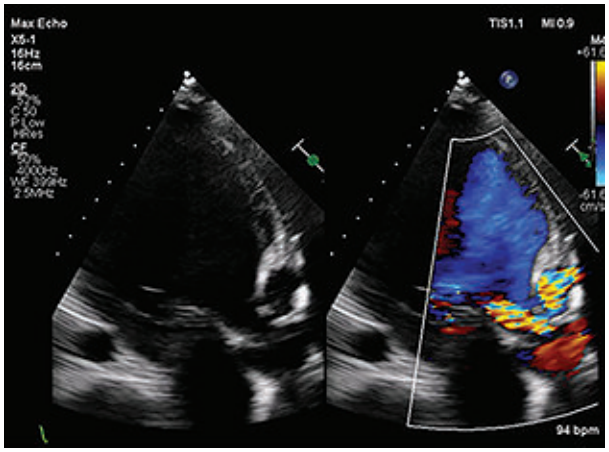

Fig.-3: *Transthoracic echo with colour comparative view showing a mosaic blood flow from right sinus of Valsalva to LVOT.*

Patient underwent DVR (21 mm aortic,25 mm mitral) and RSOV repair done. His post operative period was uneventful and he was discharged in a stable condition. Post operative echo showed that PHV in situ with no shunt from aorta to LVOT.

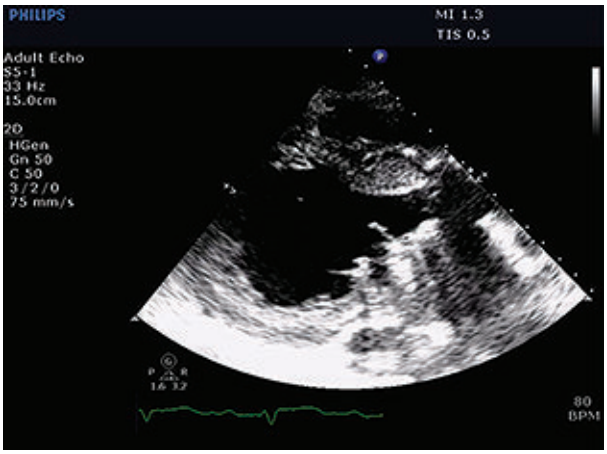

Fig.-4: *Transthoracic echo showing repaired RSOV with aortic and mitral PHV.*

Discussion:

The majority of the SVAs present as congenital but it can be acquired too, most of which is secondary to trauma, infective endocarditis, connective tissue diseases like Marfans syndrome or atherosclerosis, systemic inflammatory diseases.[6] Our patient was considered to have congenital SVA due to lack of family history and tissue disorder screening. Other cardiac anomalies like VSD, aortic regurgitation, aortic bicuspid valve, mitral and tricuspid regurgitation and coarctation of aorta are reported to be associated with congenital SVAs.[5] SVAs are frequently asymptomatic. They commonly arise from the right coronary sinus (70%), noncoronary sinus (25%).[3] If rupture occurs, a shunt more commonly develops into the right ventricle or right atrium.[7] Cases of RVOT obstruction, coronary artery compression with infarction, conduction disturbances, endocarditis and thrombus within the aneurysmal cavity also have been reported.[5]

Congenital SVAs classically rupture between 20 to 40 years of age.[1] After rupture of SVAs lead to form fistulous tracts from the aorta into the adjoining chambers of the heart. Non coronary sinus aneurysms tend to rupture into the right

atrium; right coronary sinus aneurysms can rupture into either the right atrium or right ventricle. Rarely coronary sinus ruptures in left ventricle. Ruptured SVA can present with dyspnea, palpitation, fatigue, syncope, chest pain.[3] In our patient SVA ruptured into LVOT and patient presented with sudden onset of breathlessness and uneasiness.

Transthoracic and transesophageal echocardiogram with Doppler is the first line of imaging although CTCAG and Cardiac MRI can provide an excellent anatomic depiction of either ruptured or unruptured aneurysm.[8] MRA is a potential supplementary approach but it is expensive and influenced by heart rate. CAG can not only define the anatomy of the coronary sinus but can also readily guide percutaneous closure for SVA.[5] The patient in our case was diagnosed as right coronary SVA ruptured into the LVOT with a shunt from aorta to LVOT. Transthoracic echocardiogram diagnosed the ROSV first then after CAG she underwent surgical closure with good recovery. Compared to surgery percutaneous closure intervention has the advantages of noninvasiveness and a quicker recovery with occlusive devices.[4]

The median survival of untreated ruptured SVA is 3-4 years.[7] The operation mortality rate is 1.9% to 3.6% with 90% survival at 15 years.[6] The choice of percutaneous vs surgical intervention depends on patients age, comorbidities, hemodynamic status at presentation, coexisting congenital heart defects, size of ruptured SVA and location, decision best made by multidisciplinary team.[4]

Conclusion:

SVAs when ruptured into right heart the fistulous tract will cause left to right shunting and lead to congestive heart failure. Ruptured SVAs are usually managed surgically. In selective patients, percutaneous closure is a safe alternative to surgery. We report a rare case of ruptured sinus of Valsalva aneurysm into LVOT which was successfully treated surgically by DVR and RSOV repair.

Conflict of Interest - None.

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