Case Report

Percutaneous Closure of Acquired Hole- First Case Report from Bangladesh

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Abstract:

Rupture sinus of Valsalva (RSOV) is an uncommon condition with a wide spectrum of presentation, ranging from an asymptomatic murmur to cardiogenic shock or even sudden cardiac death. Our case presented at 62 yrs with progressive onset of dyspnea, palpitation with deterioration of exercise capacity. Diagnosis of ruptured sinus of Valsalva was made by echocardiography, it was aneurysmal and opened into right ventricular outflow tract. Coronary artery disease was excluded by coronary angiogram. Probable cause of rupture was atherosclerosis.We closed percutaneously with ADO I device. The procedure was completed uneventfully. Patient discharged with duel antiplatelet and is on follow up.

Key word: *Ruptured sinus of Valsalva (RSOV), Amplatzer duct occluder I (ADO I), Percutaneous closure, atherosclerosis.*

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Introduction:

Ruptured sinus of Valsalva is a rare cardiac anomaly which is mostly due to congenital, can be also acquired. Presentation vary from asymptomatic to sudden cardiac shock even death. It can be diagnosed simple noninvasive echocardiography. Surgery is the choice of treatment, but isolated uncomplicated case can be closed by percutaneous closure though it is very much challenging. Percutaneous closure can avoid many hazards of surgery so now a days it is becoming more acceptable and demanding treatment option.

Case report:

62-year-old man was admitted to our hospital with the complain of progressive onset of dyspnea, palpitation, and deterioration in exercise capacity. He had no history of fever, trauma or any previous surgery. On examination, he was functionally New York Heart Association class III, pulse-70/min, blood pressure-160/60 mmhg, with continuous murmur IV/VI in left parasternal area. Chest X-ray showed cardiomegaly with increased pulmonary blood flow. Transthoracic & trans oesophageal echocardiography was performed and showed non coronary sinus ruptured into proximal right ventricular outflow tract (maximum & minimum diameter of RSOV, and the length of the aneurysm were taken). The ventricular septum showed no defect and no aortic regurgitation was present.

Cardiac catheterization was performed and did not reveal any coronary artery disease and showed noncoronary sinus ruptured into right ventricular outflow tract; the mean pulmonary artery pressure was 40/15 mmHg and the Qp/Qs was 1.8:1. The RSOV was crossed with a 6F Judkin Right catheter (Cordis Corporation) and a 0.035" × 260 cm straight tipped Terumo wire (Terumo Corp, Japan) from the aortic side. The wire was manipulated into pulmonary artery (PA) and snared through the right femoral vein with a 10 mm Goose Neck Snare (Microvena, MN, USA), to form an arteriovenous loop. The delivery sheath

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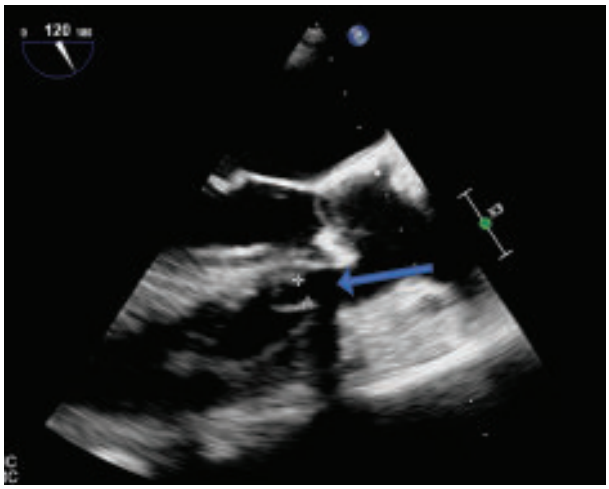

Fig.-1: *TEE showed ruptured non coronary sinus into right ventricular outflow tract.*

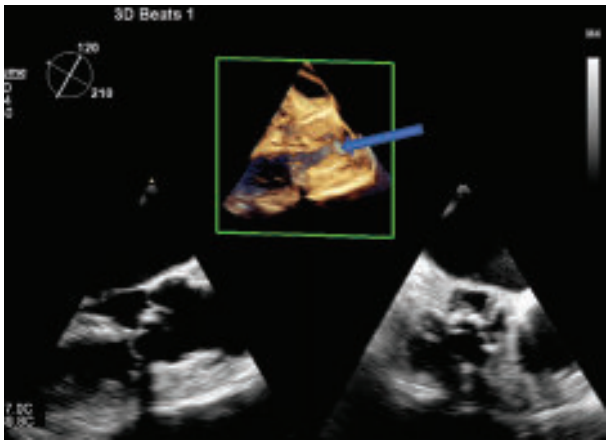

Fig.-2: *TEE 3D showed ruptured non coronary sinus from different plane into right ventricular outflow tract.*

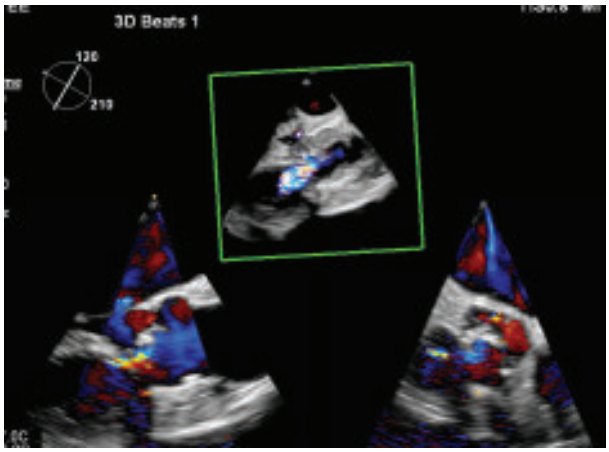

Fig.-3: *TEE 3D color mode showed ruptured non coronary sinus from different plane into right ventricular outflow tract with left to right shunt.*

was passed from the venous end and pushed over the wire across the RSOV. The device was loaded into the sheath. The aortic retention disc was opened into the ascending aorta and the entire system was pulled back till it anchored at the aortic end of the RSOV. At this point, the other end of the device was delivered by stabilizing the loading cable and pulling back the sheath. The entire maneuver was performed under fluoroscopic and transesophageal echo guidance. A check angiogram was done to confirm the position of the device. Once it was found to be optimum & quantify no AR & then the device was released. He was given Aspirin (5 mg/kg/day) and clopidogrel for six months following the procedure.

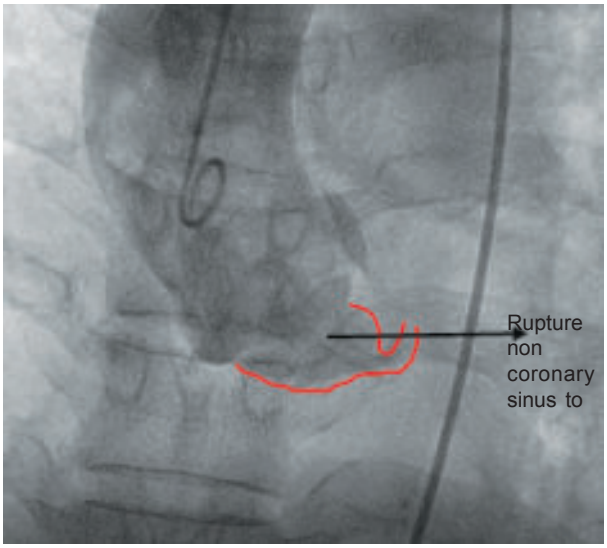

Fig.-4: *Root aortogram showing aneurysmal ruptured non coronary sinus to Right ventricular outflow tract.*

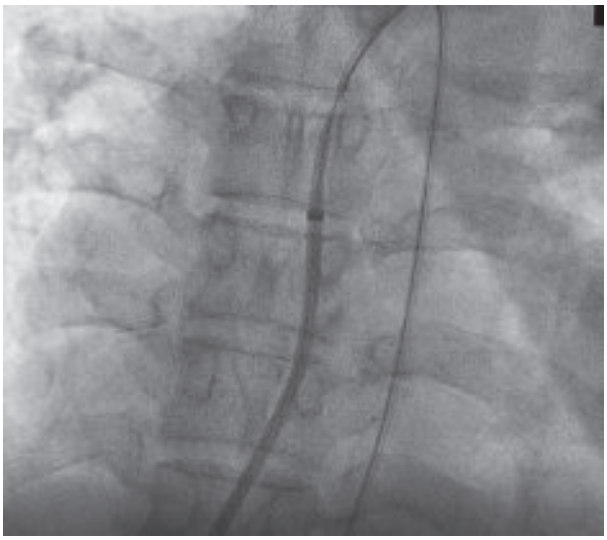

Fig.-5: *Introducing delivery sheath through antegrade approach.*

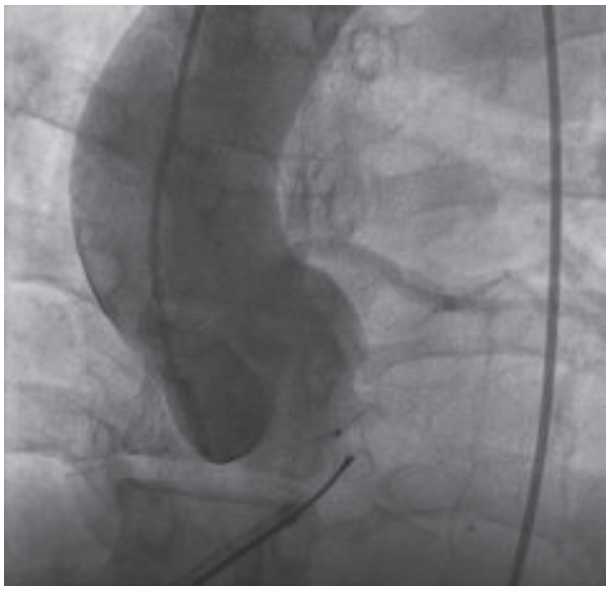

Fig.-6: *Deployment of ADO I device and confirming its secured and stabilized position.*

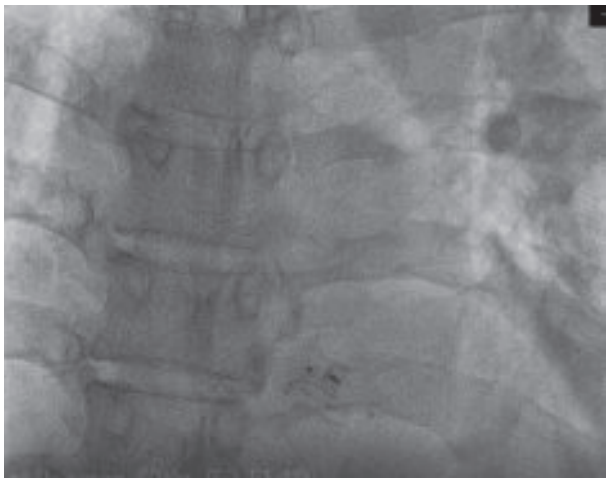

Fig.-7: *Deployed device with stabilized position.*

Discussion:

A sinus of Valsalva aneurysm is a rare cardiac anomaly where the root of the aorta may become aneurysmal & forms thin-walled saccular or tubular outpouchings. [1] They develop as a consequence of incomplete fusion of the distal bulbar septum and truncal ridges, leading to a weakness between the aortic media and the annulus fibrosus of the aortic valve.There is subsequent aneurysmal enlargement at this weak point caused by the high pressure head at the aortic root.[2,3]This dilatation progresses to rupture into the cardiac chambers(most frequently to the right ventricle or right atrium or mediastinum).[3,4] Valsalva aneurysms may involve all 3 sinuses, more frequently the right (75%– 90%) and noncoronary (10%–25%), and rarely the left coronary sinus are involved.[5] The left sinus is not derived embryologically from bulbar septum and therefore is rarely affected by congenital lesions. This anomaly can be unrecognized for many years.[6,7]RSOV are mostly congenital and comprise 0.15%–0.24% of congenital cardiac anomalies.[3]The incidence of rupture is higher in adolescence & adulthood in Eastern compared to Western populations.[8] In addition to a congenital etiology, these aneurysms may occur secondary to trauma, infective endocarditis, or tertiary syphilis, periaortic inflammation, atherosclerosis, aortic dissection, iatrogenic injuries to the sinuses during VSD closure or during debridement of a calcified aortic or mitral valve may also result in aneurysm formation. Ruptured Sinus of Valsalva (RSOV) are often associated with other congenital defects, most commonly VSD and aortic regurgitation [5]. The presence of an aneurysm may lead to a compression of an adjacent chamber, a coronary artery or the conduction system, leading to myocardial ischaemia and/or conduction disturbances. Symptoms occur in 80% of patients, most commonly between 30 and 45 years of age.[5]There is wide spectrum of presentation, ranging from an asymptomatic murmur to cardiogenic shock or even sudden cardiac death.[3] Symptoms are shortness of breath, chest pain, and fatigue, exercise intolerance, symptomatic heart failure. Symptoms depend upon severity of the shunt and presence of associated lesions.[5] There are different classification system.Guo et al. proposed a new system of simple surgical classification for right-sided defects according to rupture site.[9] This classification system identifies four types of rupture: type I, a rupture or protrusion into the right atrium; type II, a rupture or protrusion into the right atrium or right ventricle, near or at the tricuspid annulus; type III, a rupture or protrusion into the right ventricular outflow tract, under the pulmonary valve; and type IV, other types of ruptures or protusions.[9] Another system of angiographic classification uses the shape of the left-to-right shunt jet in order to facilitate the selection of occluders for percutaneous closure.[10] The four types of shunt jets identified are: type I, window-like; type II, aneurysmal; type III, tubular; and type IV, other rare conditions.10 Our patient is under type III (according to anatomy) & type II (according to angiography). Echocardiography is the gold standard for the diagnosis of RSOV and the identification of other co-existing congenital anomalies. [3] Catheterization is carried out in patients who require an evaluation of their coronary artery anatomy or if an interventional procedure is planned. [5] We have done cardiac catheterization to exclude coronary issue. Treatment of RSOV is surgical or percutaneous transcatheter intervention. Traditionally, surgical closure has been the mainstay of treatment for RSOV, with an

operative mortality rate of <5% and excellent long-term outcomes.[8,11] Nevertheless, these patients remain at risk of prolonged hospital stays and postoperative complications such as chest pain and septicemia, making percutaneous device closure an attractive alternative.[8]Lately isolated RSOV have been successfully closed percutaneously using transcatheter devices.[4]

Percutaneous closure RSOV was first attempted by Cullen et al in 1994 using a Rashkind umbrella. Since then a few reports have been published with the use of different available closure devices.[12-15]The size of the device used for RSOV closure must be accurately assessed, since a large device may interfere with coronary blood flow or aortic valve cusp movement. However, a device of suboptimal size might dislodge and embolize, or result in a significant residual shunt. Success rates up to 90% have been reported in catheter-based closures. Complications, although rare, include cardiac perforation, fistula formation, thrombosis, and device embolization into the systemic or pulmonary circulation. complications result in acute symptoms and hemodynamic compromise, requiring urgent surgical retrieval. Post-deployment follow-up includes the assessment of coronary blood flow, aortic valve function, and the presence of thromboembolism. After the deployment of a device, a short course of anticoagulants or antiplatelet drugs is recommended to prevent thromboembolism until the endothelialization of the device occurs .[16]

Conclusion:

Transcatheter closure of RSOV is an effective and safe treatment modality for isolated RSOV. Reduced pain for the patient, absence of surgical scar, shorter hospitalization and convalescence time are also important advantages. In patients where on-pump surgery is high risk, due to poor general condition and comorbidities, transcatheter device closure can be lifesaving. Extended follow-up is required to assess the long-term outcome of these patients.

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