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Case Report

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Device embolization during ruptured sinus of Valsalva aneurysm closure – ECG provides clue to the 'lost treasure'

Abstract

Ruptured sinus of Valsalva aneurysm (RSOV) is a less commonly encountered acquired defect that is often related to the underlying congenital deficiency in structural integrity of aortic sinuses in association with other congenital heart diseases. Surgical RSOV repair (SRR) has been traditionally the treatment of choice for symptomatic patients. However, with the advent of modern percutaneous techniques and refinement in hardware device closure is a safe and effective treatment in selected patients. We herein report a case of RSOV device closure related complication in which the continuous intra-procedural electrocardiogram monitoring assisted in rapid identification of the problem that was resolved successfully in a timely fashion.

Keywords: Ruptured sinus of Valsalva aneurysm, device closure, pulmonary artery embolism, device snaring, surgical repair

INTRODUCTION

Ruptured sinus of Valsalva aneurysm (RSOV) comprises up to 3.5% of all congenital cardiac defects and a mere 0.14-3% of all open-heart surgical procedures (1,2). RSOV having male predominance, commonly originates from the right or noncoronary sinus and communicates with the right heart structures and rarely into left sided chambers (1,3). Commonly associated congenital cardiac defects include ventricular septal defects (VSD), atrial septal defects (ASD) and aortic regurgitation. Epidemiological data has revealed that RSOV is more common among the Asians (1.2-4.9%) compared to the West (1,2). RSOV is symptomatic in majority of the cases, most common symptoms being breathlessness, palpitations and chest pain resulting in heart failure. RSOV requires prompt diagnosis and management. The mean period of survival for untreated cases of RSOV is only 1-2 years (3). Surgical RSOV repair (SRR) of the defect has been the traditional treatment of choice for RSOV. However, in recent times, transcatheter device closure (TDC) has proved to be an attractive alternative to surgery in appropriately selected patients of RSOV (4). Transcatheter treatment of RSOV can be considered on a case-to-case basis in hemodynamically stable patients not in cardiogenic shock or heart failure, small single defects with sufficient rim of tissue avoiding proximity to adjacent structures such as coronaries and aortic valve (at least 5 and 7 mm away from coronaries and aortic annulus respectively, without having significant AR or associated VSD) and high surgical risk (5).

CASE REPORT

23-year-old Hepatitis-B seropositive female presented with recent onset non-ischemic chest pain followed by exertional breathlessness and episodes of paroxysmal nocturnal dyspnoea (PND) since then. Clinical evaluation revealed bounding pulses, low diastolic blood pressure with wide pulse pressure, displaced left ventricular type hyperdynamic apex, grade II left pansystolic parasternal heave, loud pulmonary component of second heart sound and grade 4/6 systole-diastolic murmur best heard at lower left sternal border. Electrocardiogram (ECG) showed sinus tachycardia with volume overload type of left ventricular hypertrophy (LVH) (Figure 1). Transthoracic echocardiogram (TTE) revealed dilated left and right ventricle with normal contractility, ruptured right coronary sinus of Valsalva into

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right ventricle with significant left to right shunting, mild aortic regurgitation, moderate tricuspid regurgitation and severe pulmonary arterial hypertension without any evidence of additional intra-cardiac shunt (Figure 2). CT angiogram showed a large windsock-shaped aneurysm of right aortic sinus measuring 29.0 x 14.5 mm, ruptured at its apex with a defect measuring 9.3 mm into RV outflow tract with right coronary artery (RCA) arising 9 mm above the defect from ruptured sinus. Additional investigations were done to rule out acquired aetiologies like infective endocarditis, trauma, post-surgical (iatrogenic), vasculitis, collagen vascular disease and connective tissue disorders. Following the non-invasive assessment, she was taken up for device closure.

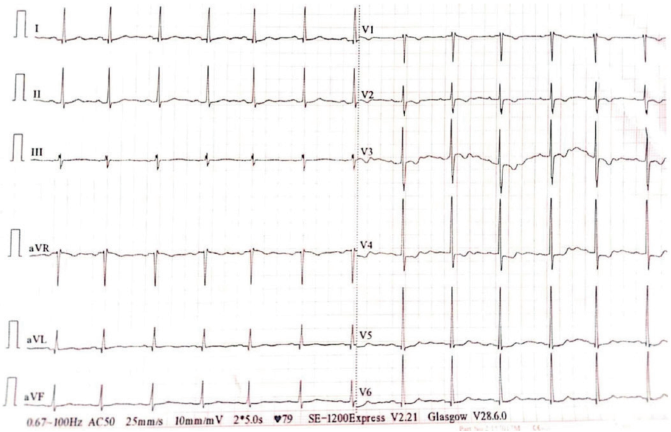

Figure 1. Baseline 12 lead ECG showing left ventricular hypertrophy (volume overload type)

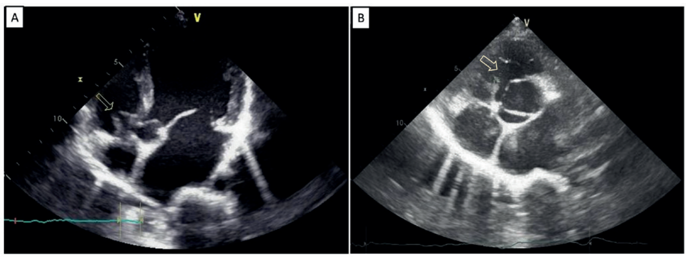

Figure 2. 2D echocardiographic images shows windsock shaped sac rupturing at its apex (arrow) into RV in apical 5-chamber view (panel A); Short axis shows aneurysmal right coronary sinus with a defect (panel B)

16/18 mm Cocoon duct occluder (Vascular Innovations, SMT, Surat, India) was deployed via antegrade approach from venous side after arterio-venous loop formation. However, within few minutes after device deployment, the device was dislodged from its position (Figure 3). Patient complained of severe chest pain with the significant drop in blood pressure to 80/10 mm Hg. Concomitantly, the surface ECG showed sinus tachycardia with S1Q3T3 pattern which provides a clue to the possible site of embolization as one of the branch pulmonary arteries (Figure 4). Check angiogram confirmed the embolization of device to main pulmonary artery that mimicked as pulmonary artery embolism on ECG. Device was immediately retrieved using 10 mm Amplatzer goose-neck snare (Medtronic plc, Minneapolis, MN, USA) and patient was immediately shifted to OR for SRR as patient was hemodynamically unstable (Figure 5). Prosthetic patch reconstruction of the sinus defect with aortic valve repair was done using transaortic approach via midline sternotomy achieving aneurysmal sac amputation, aortic leaflet coaptation and defect closure. Post-operative TTE showed no flow across the surgical patch with mild aortic regurgitation and normal LV contractility following which patient was discharged and remained asymptomatic at 1-year follow-up.

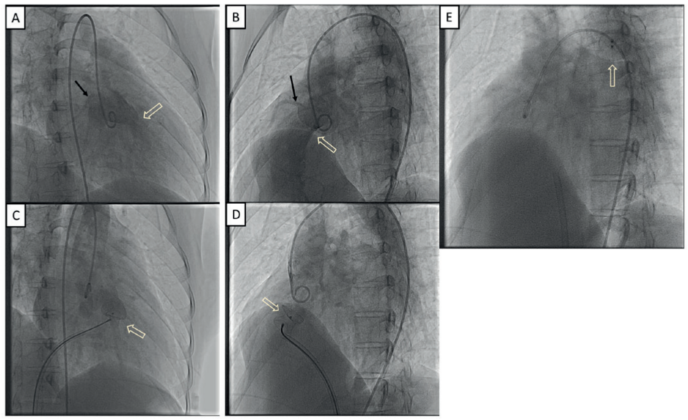

Figure 3. Ascending aortogram showing RSOV with contrast opacifying right ventricle (hollow arrow) in RAO (panel A) and LAO view (panel B); RSOV is seen at a significant distance from the origin of right coronary artery (black arrow); Duct occluder positioned at the defect in RAO (panel C) and LAO view (panel D). After deployment device gets embolized into left pulmonary artery (panel E)

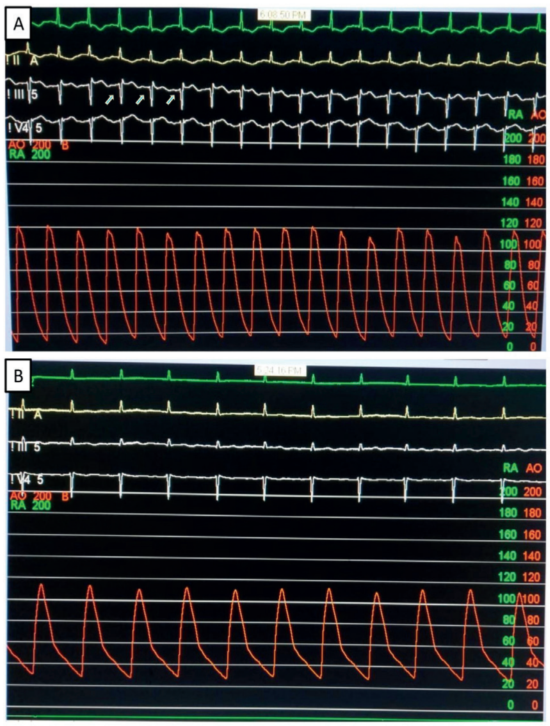

Figure 4. Intraprocedural hemodynamic recording at the time of device dislodgement showing prominent Q in lead III with inverted T wave in I, II, III suggestive of pulmonary embolism (upper panel); Lower panel shows reversal of pathological ECG changes after device retrieval. Make note of wide pulse pressure with very low diastolic blood pressures

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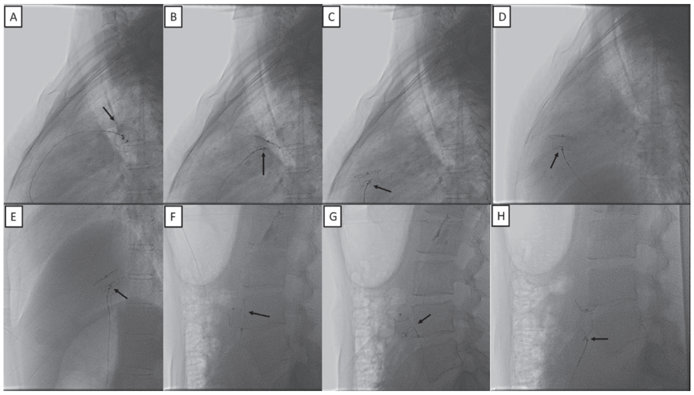

Figure 5. Step-wise percutaneous device retrieval using goose-neck snare from right femoral venous access (panel A-E); While withdrawing, the device slipped off the snare in inferior vena cava (panel F) following it was again snared out successfully (panel G-H)

DISCUSSION

Currently, both SRR and TDC are approved for the definitive treatment of RSOV. SRR, which has been the traditional treatment of choice, has excellent immediate and long-term results with a mean survival of 95% after 20 years of repair (6). SRR is preferred when there are associated congenital cardiac defects, suspicion or evidence of infective endocarditis, large RSOV with >12 mm at the aortic end, associated significant aortic regurgitation requiring repair or replacement, failed TDC attempt, persistent severe hemolysis after TDC, RSOV with multiple rupture sites and proximity of coronaries to the defect (<5 mm). Despite this, about 70% of the cases with favourable anatomy are eligible for TDC which is an attractive and safe alternative to conventional SRR (7).

Attempts to percutaneously close a RSOV dates back to 1994 using Rashkind umbrella device by Cullen et al. (8). Successful closure of RSOV using Amplatzer VSD device, atrial septal occluder, Amplatzer duct occlude and Gianturco coils is reported (7,9-11,12) The device deployed is usually oversized to 2-4 mm larger than the landing zone in case a duct occluder is deployed and equal to the landing zone when a muscular VSD occluder is chosen, after careful assessment with a combination of echocardiography and angiogram. The success rate of transcatheter closure of RSOV ranges from 75-85% (7,13,14). Complications of transcatheter closure of RSOV include device migration/embolization to pulmonary or systemic circulation, thrombosis, encroachment of aortic leaflets, AV conduction disturbances, coronary compromise and hemolysis. Embolized device can be successfully handled by snaring or open surgery.

In our case, although the device chosen was more than 50% larger than the defect size yet it got embolized. Multiple hypothesis suggests that due to persistent dilatation and aneurysm formation of the involved aortic sinus, the tunica media fails to fuse with annulus fibrosus resulting in undue thinning of the aortic wall in and around the defect (15). This often leads to inadequate rim support to the device being deployed at the site of the defect often leading to under sizing and embolization. Therefore, some interventionists recommend sizing the waist of the device to at least twice the defect diameter. Device dislodgement most often occur towards the venous side in direction of the flow of blood. In left anterior oblique or left lateral view one may get a false impression of the device being embolized into the descending thoracic aorta as in these views left pulmonary artery overlays the descending thoracic aorta. To sort out this confusion one may look for acute changes in the surface ECG pointing towards pulmonary embolism due to device related pulmonary artery obstruction, as observed in our case.

CONCLUSION

Surgical repair has been the mainstay of treatment for RSOV. However, transcatheter closure in patients having suitable anatomy has now been proven to be safe and effective in short and intermediate term outcomes. Various factors including defect suitability for device closure, operator expertise, patient preference have to be considered while managing such patients. This case report highlights the importance of observing small changes in the ECG during the procedure that may give a clue to the occurrence of a complication. Pathognomonic changes suggestive of pulmonary embolism may be seen in a case of device embolization while attempting TDC. Surgical repair with or without percutaneous retrieval of the embolized device is the mainstay in managing such complications.

Conflict of Interests: The authors declare that there are no conflict of interests.

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Patient Informed Consent: Written informed consent has been taken from the patient. Care has been taken not to disclose personal identification details of the patient.

REFERENCES

1. Malik A, Babar TS, Khalil IK, Shah MA, Laiq N. Surgical repair of ruptured sinus of valsalva aneurysm: experience at cardiovascular department, Lady Reading hospital, Peshawar. Khyber Med Univ J. 2014;6:116-20.
2. Takach TJ, Reul GJ, Duncan JM, Cooley DA, Livesay JJ, Ott DA, et al. Sinus of valsalva aneurysm or fistula: management and outcome. Ann Thorac Surg. 1999;68:1573-7.
3. Jaswal V, Kumar V, Thingnam SKS, Puri GD. Surgical repair of ruptured sinus of Valsalva aneurysm: 13-year single center experience. J Card Surg. 2021;36:1264-9.
4. Kirklin JW, Barratt-Boyes BG. Congenital sinus of Valsalva aneurysm and aortico-left ventricular tunnel. In: Kirklin JW, Barratt-Boyes BG, eds. Cardiac surgery 3rd edition. New York: Churchill-Livingstone; 2003. p911-927.

DOI: 10.5455/azjcvs.2024.06.013

5. Xiao JW, Wang QG, Zhang DZ, Cui CS, Han X, Zhang P, et al. Clinical outcomes of percutaneous or surgical closure of ruptured sinus of Valsalva aneurysm. Congenit Heart Dis. 2018;13:305-10.
6. van Son JA, Danielson GK, Schaff HV, et al. Long-term outcome of surgical repair of ruptured sinus of Valsalva aneurysm. Circulation. 1994;90:II20-9.
7. Kerkar PG, Lanjewar CP, Mishra N, Nyayadhish P, Mammen I. Transcatheter closure of ruptured sinus of Valsalva aneurysm using the Amplatzer duct occlude: immediate results and mid-term follow-up. Eur Heart J. 2010;31:2881-7.
8. Cullen S, Somerville J, Redington A. Transcatheter closure of a ruptured aneurysm of the sinus of Valsalva. Br Heart J. 1994;71:479-80.
9. Arora R, Trehan V, Rangasetty UM, Mukhopadhyay S, Thakur AK, Kalra GS. Transcatheter closure of ruptured sinus of valsalva aneurysm. J Interv Cardiol. 2004;17:53-8.
10. Guan L, Zhou D, Zhang F, Pan W, Dong L, Chen H, Ge J. Percutaneous device closure of ruptured sinus of valsalva aneurysm: a preliminary experience. J Invasive Cardiol. 2013;25:492-6.
11. Chang CW, Chiu SN, Wu ET, Tsai SK, Wu MH, Wang JK. Transcatheter closure of a ruptured sinus of valsalva aneurysm. Circ J. 2006;70:1043-7.
12. Zhao SH, Yan CW, Zhu XY, Li JJ, Xu NX, Jiang SL, et al. Transcatheter occlusion of the ruptured sinus of Valsalva aneurysm with an Amplatzer duct occluder. Int J Cardiol. 2008;129:81-5.
13. Tong S, Zhong L, Liu J, Yao Q, Shu M, Song Z. The immediate and follow up results of transcatheter occlusion of the ruptured sinus of Valsalva aneurysm with duct occluder. J Invasive Cardiol. 2014;26:55-9.
14. Mahimarangaiah J, Chandra S, Subramanian A, Srinivasa KH, Usha MK, Manjunath CN. Trans-catheter closure of ruptured sinus of valsalva: different techniques and mid-term follow up. Cath Cardiovasc Interv. 2016;87:516-22.
15. Edwards JE, Burchell HB. The pathological anatomy of the deficiencies between the aortic root and the heart including aortic sinus aneurysms. Thorax. 1957;12:125-39.