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Case Report

Hemolysis induced by PMIVSD occluder

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A B S T R A C T

Hemolysis related to occluder, prosthetic valve, and prosthetic ring used for mitral valve annuloplasty are not very unusual. However, hemolysis related to transcathetor closure of post-myocardial infarction ventricular septal defect (PMIVSD) is infrequent. A close follow-up for spontaneous resolution with or without blood transfusion has been reported in a few cases. Occasionally, surgical retrieval is unavoidable or lifelong blood transfusion is required if surgery cannot be done because of higher risk. In this illustration, we have showed a close follow-up of a case of hemolysis induced by atrial septal occluder used for VSD closure after myocardial infarction. Despite successful device closure of PMIVSD which is difficult, a close watch is needed for complications like residual leak, device embolization, and hemolysis.

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1. Introduction

Hemolysis induced by prosthetic material used to occlude, replace, or repair the structural cardiac defects is not very infrequent either in intervention cardiology or cardiovascular surgery. The encounter has a rising trend because of significant rise in device-based treatment. The unavoidable residual leaks, which are featured by high velocity, vortices, and rapid deceleration, impose significant shear on red blood cells leading to hemolysis. Transcathetor closure (TCC) of post-myocardial infarction ventricular septal defect (PMIVSD) is difficult and not practiced frequently. Also, the experience of treating hemolysis induced by occluder used in this condition is rare (0.02–0.035%)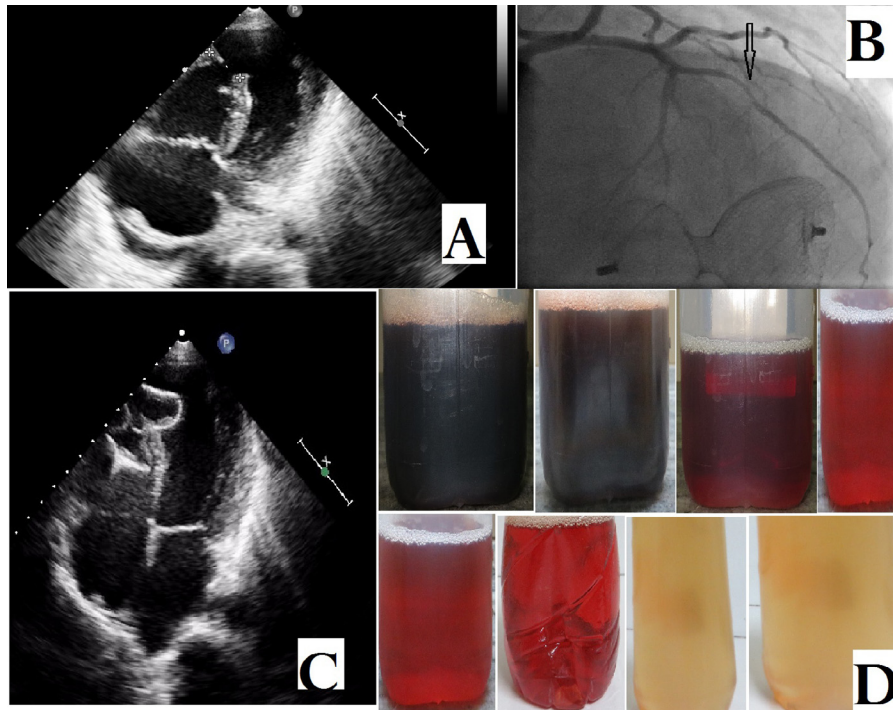.[1,2] A close follow-up for spontaneous resolution with or without blood transfusion have been reported in a few cases.[3,4] Occasionally, surgical retrieval and repair are unavoidable.[5]

2. Illustration of the case

A 49-year-old male presented with history of ventricular septal rupture after thrombolysis for anterior wall myocardial infarction. He was in Killip's class III for last 6 weeks post-myocardial infarction. At presentation, he had features of significant left heart failure (orthopnoea and crepitation in both the lung fields) and right heart failure (pedal edema, raised JVP, and ascites). Precordial examination revealed grade III parasternal lift, loud P2, and III/VI pansystolic murmur.

Fig. 1 – (A) 2D echo shows a large ventricular defect of size 2.1 cm in middle part of interventricular septum with mid-distal septal aneurysm; (B) Coronary angiogram in right anterior oblique (RAO) view shows critical type B stenosis in the middle part of LAD which was stented and the scene angiogram shows atrial septal occluder (Cardi-O-Fix, 28 mm) across the ventricular septal defect immediately following the TCC; (C) Echo shows a stable and better aligned device plugging the VSD at the end of one-month follow-up; (D) Serial changes in color of urine from 2nd day device closure till the day of discharge.

12 Lead electrocardiogram and X-ray chest were consistent. The coronary angiogram which was done outside, revealed critical stenosis of mid part of left anterior descending coronary artery (LAD). The patient was nondiabetic and nonsmoker. 2D echo showed hypokinetic anterior wall, septal aneurysm of distal IVS and apex, ventricular septal defect of size 2.1 cm, significant left to right shunt, gradient of 35 mmHg across VSD, and near systemic right ventricular pressure with significant right ventricular dysfunction [Fig. 1A, Video 1 and Video 2]. After medical stabilization for 48 h and proper consent, we planned for TCC of VSD and angioplasty of LAD. After hemodynamic study on oxygen, we successfully plugged the VSD using an atrial septal occluder of size of 28 mm (Cardi-O-Fix) via right internal jugular venous approach. A critical lesion in the LAD was directly stented using 2.5 mm × 12 mm Taxus stent [Fig. 1B and Video 3]. This was followed by post-procedure hemodynamic study to evaluate the impact of device closure. Intra-aortic balloon pump was not used in this case because of stable hemodynamic. There were tiny residual leaks around the device with insignificant left to right shunt. But unfortunately, this patient developed intense hemolysis on second day of the procedure marked by significant drop in hemoglobin, hemoglobinuria (Coca-Cola colored urine), significant elevation of lactate dehydrogenase (LDH), systocytes in peripheral smear, mild elevated reticulocyte count, high level of unconjugated bilirubin, positive urobilinogen in urine, and jaundice. Echo on day three showed mild residual shunt across the device due to poor apical rims which is common in

large ventricular septal defect after myocardial infarction. The device was almost parallel to the muscular septum and not well seated on the RV side [Fig. 1C, Video 4]. One unit of fresh packed red blood cell (PRBC) was transfused when hemoglobin dropped from 13 g% to 7 g%. Urine color changed gradually from Coca-Cola to normal over a period 11 days of his ICCU stay [Fig. 1D]. An abridged summary of his periprocedural clinical condition, hemodynamic study (non-invasive and invasive), and experience of device closure including one-month follow-up have been included in Table 1. He was successfully discharged without further episode of hemolysis. At the end of first-month follow-up, device position was stable, there was further significant decrease in residual leaks [Video 5] and no more evidence of hemolysis.

3. Discussion

TCC of VSD after MI is an effective alternative to surgical closure especially in subacute or chronic condition.[1] The closure by surgery or device is not immune to residual leaks.[6,7] Though the persistent residual leak is frequent with TCC, it is rarely associated with hemolysis by several mechanisms as have been mentioned before. We have not come across hemolysis related to TCC of PMIVSD in experience of consecutive 7 cases from 2012 to 2015.[8,9] The unavoidable residual leaks, which are featured by high velocity jets, vortices, and rapid deceleration, impose significant shear on

Table 1 – Periprocedural observation of TCC of PMIVSD using atrial septal occluder.

Items	Days in hospital			
	1–4th	5th	6th–15th	16th
Clinical	Killip's III, Anasarca PSM: III/VI	TCC	Dramatic improvement in general condition, HR and BP was stable. Jaundice appeared but improved on follow up toward discharge. PSM – III/VI	Discharged
ECG	Baseline is Q in V1–V4 but developed RBBB after device closure			
X-ray chest	Cardiomegaly; pulmonary edema and right sided pleural effusion which improved dramatically after TCC			
ECHO	Baseline: Aneurysm of mid part of IVS, size of VSD is 2.1 cm, RVSP of 90 mmHg, LVEF – 35–40%, TAPSE of 0.8 cm, LV-RV gradient of 35 mmHg and thin rim of pericardial effusion Post-procedure: Tiny residual shunts through or around ASO, RVSP – 30 mmHg 1-Month follow-up echo: Stable device position, tiny residual shunt and normal PA pressure			
Invasive hemodynamic (on O$_2$)	Baseline: HR – 85 min^{-1}, Saturations (%): SVC – 44, IVC – 59, RA – 55, RV – 97, PA – 96, FA – 99; Pressure (mmHg): RA – 16, RVs – 90/10, PA – 90/26/52, PCWP – 20, LV – 128/16, FA – 120/80/94, significant left to right shunt Post-ASO closure: Saturation (%): RA – 54, RV – 58, PA – 59; FA – 99.2; Pressure (mmHg): RA – 12, RV – 58/10, PA – 55/26/38, FA – 150/80, PCWP – 16, QP/QS: reduced significantly			
Urine	Pre-procedure: Amber color and normal Post ASO closure: Urine color changed deep Coca-Cola color on day-2, gradually returned to amber color on 8th day post procedure with one unit blood transfusion. There was no hematuria. Test for Urobilinogen was positive			
Liver enzymes	Baseline: Mildly elevated (SGOT and SGPT) Post closure: Mild changes			
Hemoglobin (g/dl)	Baseline: 13 Post-procedure: Lowest 7.2 on the 8th day of TCC, improved to 9.2 after 1 unit of blood transfusion and remained stable			
Bilirubin: total/ conjugated (mg/dl)	Baseline: 1.9/0.9 Post-procedure: Peak – 7.2/0.9, peak conjugated bilirubin was 2.2 and at discharge it was 3.2/0.8			
Blood urea/serum Cr (mg/dl)	40/1.2 at baseline remained near normal during hospital			
Other tests	Platelet count: 6 × 10^5 mm^{-3} (periprocedural mild changes) LDH: 2361 IU/L on 2nd day of procedure which improved gradually Reticulocyte count – No significant changes Platelet aggregation test by light aggregometry: 10% Peripheral smear for systocyte: 1–5/HPF Test for malaria parasite: Negative Coomb's test: Negative Viral markers: Negative			
Treatment	Improving CHF	TCC and PCI	Dual antiplatelet (Prasugrel and aspirin) was changed to only aspirin and again changed to dual antiplatelet at time of discharge 1 unit of PRBC Watched Discharged	

red blood cells leading to hemolysis.[10,11] In cases of PMIVSD, multiple defects, ongoing remodeling, serpiginous pathway, aneurysm, improper size of the device, and incorrect alignment of device during implantation are the several reasons for device malposition leading to tiny high velocity residual leaks. In our case, we feel a little oversizing of device and associated aneurysm of apical interventricular septum were the reasons for underexpansion of the neck of the device and its discs leading to residual shunt, which may be the reason for hemolysis. The proper evaluation of location, size, shape, number of defects, and aneurysm should be done prior to the TCC, which may reduce the incidence of device-related hemolysis. There are several ways to reduce residual leaks, which have been addressed as follows. The approved device for TCC of PMIVSD is dedicated Amplatzer PMIVSD occluder (AGA Medical Corp., Golden Valley, Minnesota, USA). The maximum size of VSD which can be closed by this device is 25 mm.[12] If the size of VSD is larger than 24 mm, ASD occluder can be used. A previous report suggests a defect size of 15 mm should be treated with percutaneous therapy, either as a temporary strategy and to stabilize the patient until a definitive surgical repair can be accomplished because larger size occluder leads to higher rate residual leak because of malposition.[7] Routinely, the size of device chosen for TCC of PMIVSD is 50% higher than defect (waist of device to defect size ratio is 150%) in acute condition because of unreliable borders, shape, and ongoing remodeling. In chronic condition, the size of waist of the chosen device should be 4 mm higher than defect size, which would adjust any further remodeling. In our case, though the age of defect was more than 6 weeks, the borders of the defect could not be reliably assessed by 2D echo. Therefore, we have chosen a device of size 28 mm with defect to device ratio of 21:28 (≈130%). We have chosen ASD occluder in our case for two reasons: (1) a defect size of 21 mm and (2) we thought a larger disc on LV side would provide a stable device position. Because of the complex shape and nature of the defects, 2D and 3D echo do not seem to fully describe the anatomy as the proper window is limited in such critically ill patients as in our case. Therefore, computed tomography or magnetic resonance imaging should be used to better

delineate the anatomy of the defect. Also, other devices like ASD and PDA occluder and coils can be used by experienced operators.[12–14] The role of balloon sizing is controversial in this condition unlike congenital ASD or VSD. If the defect is too much complex, it may need more than one device[15] and procedure can be staged. Till now, the shape of the chosen device is limited to the choice of the individual operator depending upon the 3D shape of defect.

Conflicts of interest

The authors have none to declare.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ihj.2016.02.011.

REFERENCES

1. Egbe AC, Poterucha JT, Rihal CS, et al. Transcatheter closure of postmyocardial infarction, iatrogenic, and postoperative ventricular septal defects: the Mayo Clinic experience. *Catheter Cardiovasc Interv*. 2015;86:1264–1270.
2. Xu X-D, Liu SX, Liu X, et al. Percutaneous closure of postinfarct muscular ventricular septal defects: a multicenter study in China. *J Cardiol*. 2014;64:285–289.
3. Turner MS, Hamilton M, Morgan GJ, Martin RP. Percutaneous closure of post-myocardial infarction ventricular septal defect: patient selection and management. *Interv Cardiol Clin*. 2013;2:173–180.
4. Ali TA, Fatimi SH, Hasan BS. Transcatheter closure of a traumatic ventricular septal defect using an amplatzer™ atrial septal occluder device. *Catheter Cardiovasc Interv*. 2013;82:569–573.
5. Martinez MW, Mookadam M, Mookadam F. A case of hemolysis after percutaneous ventricular septal defect closure with a device. *J Invasive Cardiol*. 2007;19:E192–E194.
6. Chessa M, Carminati M, Cao QL, et al. Transcatheter closure of congenital and acquired muscular ventricular septal defects using the Amplatzer device. *J Invasive Cardiol*. 2002;14:322–327.
7. Maltais S, Ibrahim R, Basmadjian AJ, et al. Postinfarction ventricular septal defects: towards a new treatment algorithm? *Ann Thorac Surg*. 2009;87:687–692.
8. Patnaik AN, Barik R, Kumari NR, Gulati AS. Device closure of post-myocardial infarction ventricular septal defect three weeks after coronary angioplasty. *J Cardiovasc Dis Res*. 2012;3:155–159.
9. Rao DS, Patnaik AN, Barik R, Nemani L, Prasad AS. Transcatheter closure of postinfarction ventricular septal rupture. *J Indian Coll Cardiol*. 2015;5:220–227.
10. Tiong Y, William F, Hartzell S, Thomas O. Mechanisms of hemolysis after mitral valve repair: assessment by serial echocardiography. *J Am Coll Cardiol*. 1998;32:717–723.
11. Morshed KN, Bark Jr D, Forleo M, Dasi LP. In: Cox D, ed. Theory to Predict Shear Stress on Cells in Turbulent Blood Flow. *PLoS ONE*. 2014;9:e105357.
12. Demkow M, Ruzyllo W, Kepka C, et al. Primary transcatheter closure of postinfarction ventricular septal defects with the Amplatzer septal occluder – immediate results and up-to 5 years follow-up. *EuroIntervention*. 2005;1:43–47.
13. Calvert PA, Cockburn J, Wynne D, et al. Percutaneous closure of post-infarction ventricular septal defect: in-hospital outcomes and long-term follow-up of UK experience. *Circulation*. 2014;129:2395–2402.
14. Trivedi KR, Aldebert P, Riberi A, et al. Sequential management of post-myocardial infarction ventricular septal defects. *Arch Cardiovasc Dis*. 2015;108:321–330.
15. Capasso F, Caruso A, Valva G, Lonobile T, Grimaldi MG, Santoro G. Device closure of 'complex' postinfarction ventricular septal defect. *J Cardiovasc Med*. 2015;16:S15–S17.