Percutaneous ventricular septal defect closure causing acute aortic regurgitation

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Intravascular hemolysis and aortic regurgitation (AR) are rare complications of transcatheter closure of perimembranous ventricular septal defects. The current study reports a case of an acute AR, which resulted from acute right coronary cusp perforation by the ventricular septal defect occluder. The current manuscript discusses the possible causes of early cusp erosion owing to occluder, advantages of early operation in such cases, and role of thorough perioperative transesophageal echocardiography in identifying acute AR.

Keywords:
aortic regurgitation, perioperative transesophageal echocardiographic, transcatheter closure, ventricular septal defect

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Introduction

New-onset massive hemolysis and acute aortic regurgitation (AR) are rare complications after transcatheter closure of perimembranous ventricular septal defect (pmVSD). Intravascular hemolysis following percutaneous VSD closure is rare and usually transient [1]. Conservative treatment is sufficient but surgical intervention may be required rarely. Acute AR may occur after transcatheter closure of pmVSDs with an incidence of 1.27% [2]. It may result from iatrogenic injuries to aortic valve by catheter manipulation and VSD occluder and can be detected during routine echocardiography. In this reported case, we detected right coronary cusp (RCC) perforation in a patient undergoing VSD device removal under cardio-pulmonary bypass for device-induced refractory hematuria. The importance of a thorough routine postcardiopulmonary bypass (CPB) transesophageal echocardiographic (TEE) examination to rule-out any surgical issues before leaving the operating room is crucial to the management of such cases.

Case report

A 5-year-old female patient was diagnosed with 10-mm pmVSD with adequate aortic rim (7 mm). She underwent standard transcatheter pmVSD closure under fluoroscopic and TEE guidance 5 days back using nitrinol-based Amplatzer (AGA Medical, Golden Valley, MN, USA) muscular VSD occluder (waist size 12 mm). Aspirin 5 mg/kg/day was started within 12 h of the procedure. Patient started passing cola-colour urine from first postprocedure day and was managed conservatively with fluids and blood transfusions. Echocardiography revealed a left to right high-velocity jet across the VSD device occluder. Hemoglobin reached a nadir of 5 mg/dl on the third day after procedure following which 2 U of packed red blood cell were transfused. Hemolysis was refractory to medical treatment, and blood hemoglobin was not maintained in spite of multiple blood transfusions. Laboratory tests were performed to exclude any immune or drug-induced mechanism of hemolysis. It appeared that the intravascular hemolysis was due to blood flow from left to right across the VSD device. Surgery was planned for retrieval of VSD occluder and polytetrafluoroethylene patch closure of pmVSD.

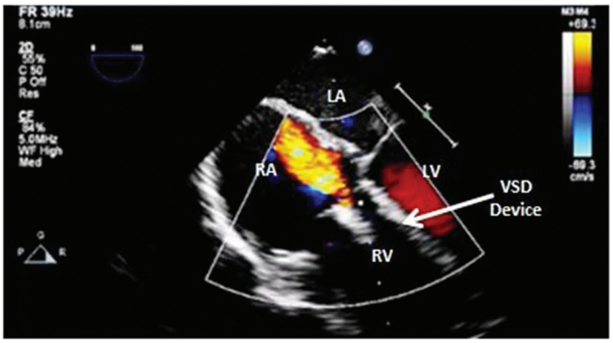
Preanesthetic assessment showed healthy patient weighing 12.5 kg with hemoglobin level of 7.8 mg/dl using Lovibond comparator technique with arterial blood pressure of 94/56 mmHg, and heart rate of 92 beats/min. Standard anesthesia management was carried out as per the institutional protocol. Pre-CPB TEE was done using 2D TEE probe (6VT-Dprobe of GE vivid E9 echocardiography system; GE Medical Systems, Horten, Norway). It revealed high-velocity jet coming across the

device (left to right) (Fig. 1) and no AR jet was visualised (Fig. 2). A right atriotomy was performed to control and remove the occluder through the tricuspid valve, and polytetrafluoroethylene patch closure of pmVSD was done under CPB. Conventional ultrafiltration was carried out, and diuresis was maintained with fluids and furosemide 10 mg in divided doses. After coming off CPB, TEE examination showed mild aortic insufficiency with vena contracta of 0.3 cm (Fig. 3) and perforation of RCC. CPB was reinstituted and after aortotomy 4×3 mm oval perforation of RCC was identified (Fig. 4). The mechanism of AR appeared to be caused by occluder which was impinging on RCC. Primary closure of RCC perforation was done. The patient was successfully weaned off CPB with nor adrenaline 0.05 µg/kg/min. The post-CPB TEE revealed no AR and no residual VSD. ICU course was uneventful. Patient was successfully weaned off ventilator and extubated after 6 h of mechanical ventilation and shifted to ward on the second postoperative day. After an uneventful recovery, patient was discharged from hospital on the fourth postoperative day.

Discussion

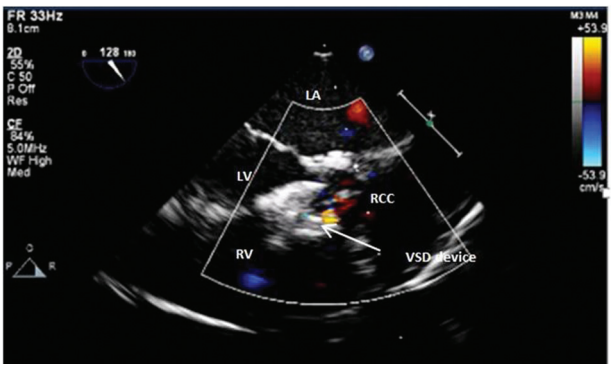
PmVSD is one of the most common congenital heart defects [3] and accounts for almost one-fifth of all defects. It is more frequent in Asian countries [4]. Standard treatment is open cardiac surgery under CPB [5]. The catheter-based approach for VSD as an alternative to surgical closure has encouraging results with acceptable mortality and morbidity [6]. The transcatheter pmVSD closure has become a novel alternative to open heart surgery owing to its noninvasive nature. Transcatheter closure has the advantages of reduced pain, less discomfort owing to the procedure, shorter hospital and ICU stay, faster recovery, less blood transfusion, less myocardial injury, and no chest scar [7–9]. Despite technological advances in transcatheter closure, complications (2.9–5.7%) are still being reported, which include heart block (5.6%), valve regurgitation (5.7%), and occluder migration (0.9%) [9]. Open cardiac surgery is still the treatment of choice for pmVSD given such high rates of these complications in children [9].

Figure 1



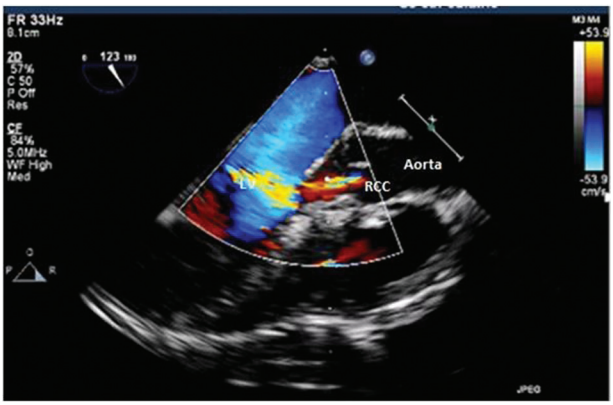
Midesophageal four-chamber view showing jet coming across the VSD device. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; VSD, ventricular septal defect.

Figure 2



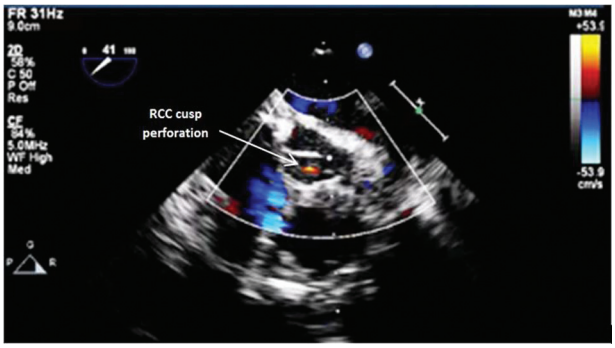
Midesophageal long axis view showing VSD device in left ventricular outflow tract impinging upon the right coronary cusp (RCC). LA, left atrium; RV, right ventricle; VSD ventricular septal defect.

Figure 3



Midesophageal long axis view showing aortic regurgitation. LV left ventricle; RCC, right coronary cusp.

Figure 4

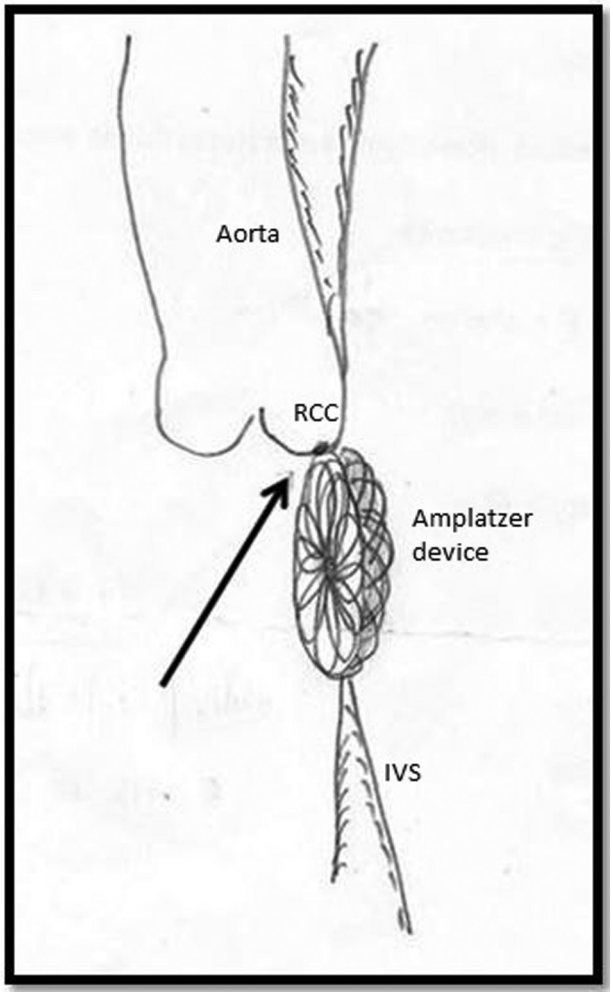


Midesophaageal Aortic valve short axis view showing Perforation of right coronary cusp measuring 2 × 3 mm. RCC, right coronary cusp.

Intravascular hemolysis is a rare but known potential complication following percutaneous closure of VSD [1,10]. It is usually transient and recovers with conservative treatment but intervention of some form may occasionally be necessary. Hemolytic anemia has been reported subsequent to such hemolysis when using Amplatzer device, which required multiple blood transfusions and finally removal of the device under CPB [1]. The Amplatzer muscular VSD device is a nitrinol construct with a short connecting waist corresponding to the size of the VSD. Amplatzer devices are self-centering and the discs and waist are filled with polyester patches. The patches improve device closure, which occurs via in-situ thrombosis. In addition, Amplatzer devices are retrievable after deployment for repositioning, if the initial result is unsatisfactory. They are not retrievable, however, once the procedure is completed. The intravascular hemolysis due to intracardiac mechanical devices with high leakage flow through or around the device is due to rapid acceleration, fragmentation, and collision of high-velocity blood causing high shear stress that leads to hemolysis [1,10]. High plasma hemoglobin and hemoglobinuria can lead to acute tubular necrosis [11]. Although no single strategy has been proven to be beneficial, maintenance of hemodynamics and diuresis is very important to protect the kidney injury owing to acidic heme and other inflammatory cytokines [12].

AR is one of the severe but rare complications that may occur after surgical repair and transcatheter closure of pmVSDs [2,6,13]. Liu *et al*. [6] reported 12 cases that developed AR in a case study involving 890 patients after the transcatheter occlusion of pmVSD, among which nine cases were mild regurgitation and three were moderate regurgitation. AR in such cases is mainly due to defects on the proximal edge of the RCC and the effect of an occluder on aortic valve closure. A pmVSD occluder shift after the separation may also be one of the reasons for AR. This result may be because of the occluder rotating and shifting in an instant of its separation from the push-pole after its release. A large occluder may get too close to the aortic valve, and the memory alloy of an occluder may gradually erode the surface in contact and be the cause of delayed AR. Moreover, iatrogenic injury of RCC during transcatheter placement of device or during retrieval of device cannot be ruled out. In case of any suspicion, in which the left ventricle occluder disk almost impinges upon the RCC, it is better to abandon the case and refer for cardiac surgery for fear of late aortic valve regurgitation [13]. In cases of clinically significant aortic valve regurgitation after procedure, early surgical retrieval of the device should be performed as it decreases the possibility of adhesions around the device making it difficult and complicated to retrieve at a later stage. Early surgical intervention could also prevent the need for possible aortic valve replacement later in life [9]. In this case report, the likely reason for AR was the perforation of the RCC by the anterosuperior margin of the occluder device (Fig. 5). The occluder device was close to the RCC in the left ventricle outflow tract which could have masked the jet of AR preoperatively. Once the device had been removed, the jet of AR was evident on TEE in post bypass period. Evidence for early perforation of RCC due to VSD occluder is not overwhelming in literature so far. However, late RCC perforation has been described [13]. The treatment strategy for such VSD device-associated AR is to repair the underlying perforation. Such cases require multidisciplinary teamwork and discussion from the beginning for

Figure 5



Perimembranous VSD Occluder disc impinging on the right coronary cusp (RCC). IVS, interventricular septum.

assessing the suitability of the case for device closure till management of the complication thereof.

Conclusion

In the aforementioned report, we describe the occurrence of massive intravascular hemolysis and acute AR secondary to cusp perforation in attempted device closure of a pmVSD. All previous reports attribute this to cusp attrition from a proximate device (either a consequence of location or over sizing) or catheter manipulation. The acute nature of the occurrence necessitates emphasis on a thorough and systematic examination of the aortic valve proximate to device insertion. TEE examination on coming off bypass should be done and is very useful for detecting possible complications arising because of surgical or device closure of VSD.

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Conflicts of interest

There are no conflicts of interest.

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