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BRIEF COMMUNICATION

An Unusual Complication of the Ventricular Septal Defect Closure by Device: Late Right Aortic Cusp Perforation

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Aortic regurgitation (AR) is a rare complication of transcatheter closure of perimembranous ventricular septal defects (pmVSD). It results from iatrogenic pinching of the aortic valve by the VSD occluder or perforation by the catheter. It is usually detected during control echocardiography (ECHO). The current study reports the first case of a late AR, which resulted from late right coronary cusp perforation by the VSD occluder.

The current manuscript discusses the possible causes of late cusp erosion due to occluder, advantages of early operation in such cases, and an alternative treatment method where the occluder removal is not possible at the operation.

Keywords Ventricular septal defect • Transcatheter closure • Aortic regurgitation • Occluder • Aortic valve

Introduction

PmVSDs are among the most common congenital heart defects [1]. Standard treatment is open cardiac surgery, which requires cardiopulmonary bypass (CPB) [2]. To avoid complications of open cardiac surgery, transcatheter VSD closure was reported in 1988 [3]. Hijazi et al. used Amplatzer VSD occluder for pmVSD closure for the first time, and reported that the method could be an alternative to surgery [4]. Despite technologic advances in recent years, a high rate of complications (2.9-5.7%) is still reported after transcatheter occluder closure, including heart block (5.6%), valve regurgitation (5.7%), and occluder migration. Due to the high rate of these complications open cardiac surgery is still the treatment of choice for pmVSD treatment in countries such as the United States. However, with the redesign of occluders, and the revision of the transcatheter closure criteria, successful results have been reported [8,9].

Aortic regurgitation, which originates from an occluder, usually results from the occluder pinching the cusp, and this event is detected in the early stages [7]. There are no reports of late-stage cusp perforation from the occluder causing progressive AR.

The current case presentation discusses the possible causes of aortic cusp perforation due to occluder, which caused AR in the late-stage, and its surgical treatment.

Case presentation

A 10 year-old male was admitted to our clinic with severe AR. According to his medical history, the patient underwent transcatheter pmVSD closure three years prior in another centre. The patient was discharged without any complications, as control aortography after occluder placement did not show AR (Figure 1). However, moderate AR was observed in the control echocardiography (ECHO) six months later, and the degree of AR increased in the subsequent follow-ups. On admission, his blood pressure was 120/50 mmHg, heart rate 108/min. Cardiac auscultation

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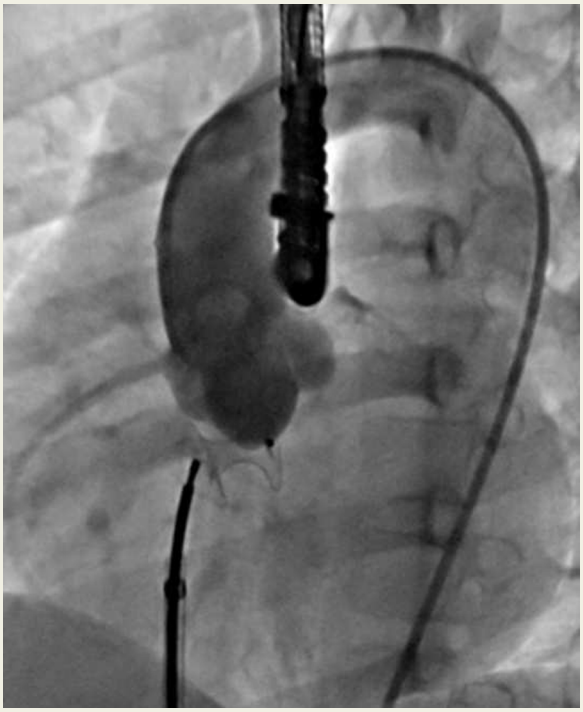


Figure 1 Angiography showing no signs of AR after transcatheter VSD closure.

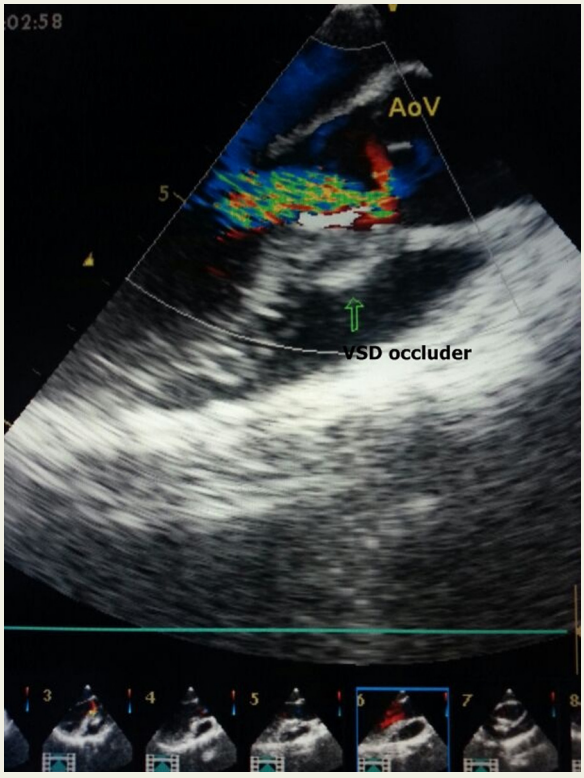


Figure 2 ECHO image of eccentric AR after transcatheter closure. AoV: aortic valve.

revealed a grade 3 early diastolic murmur in the right second intercostal space. Electrocardiograms demonstrated normal sinus rhythm. Chest radiography revealed an enlarged heart. ECHO showed severe AR and revealed left ventricle dilatation (Figure 2).

The patient was operated on CPB with mild hypothermia. After aortotomy we identified a 5x4 mm oval perforation at the base of the right aortic cusp (Figure 3). We observed that the anterosuperior region of the left side disc of occluder was not epithelialised and was not displaced from the septum and protruded into the left ventricular outflow tract. We thought that the right aortic cusp perforation was caused by this occluder which was very near to the right aortic cusp (Figure 4).

A right atriotomy was performed to control and remove the occluder through the tricuspid valve. Theocclude was epithelialised and adherent to the surrounding tissues. Known techniques were employed to remove the occluder, but it could not be removed due to the amount of adhesion. In order to not damage neighbouring tissues, excessive force was not applied. We decided to remove partially the left disc of occluder which caused aortic leaflet perforation. Therefore, the anterior-superior region of the occluder was partially cut without disrupting the occluder integrity. To prevent the formation of a thrombogenic occluder surface on the resected side and avoid occluder migration, the VSD was closed with autologous pericardium. The sutures were placed on the edge of occluder on the left ventricular side of the septum, in order to also secure the occluder The perforation in the coronary cusp was also closed with a patch of autologous pericardium. Aortic valve configuration and cusp coaptation were tested by filling the aortic root with saline. Configuration and coaptation of aortic valve was normal.

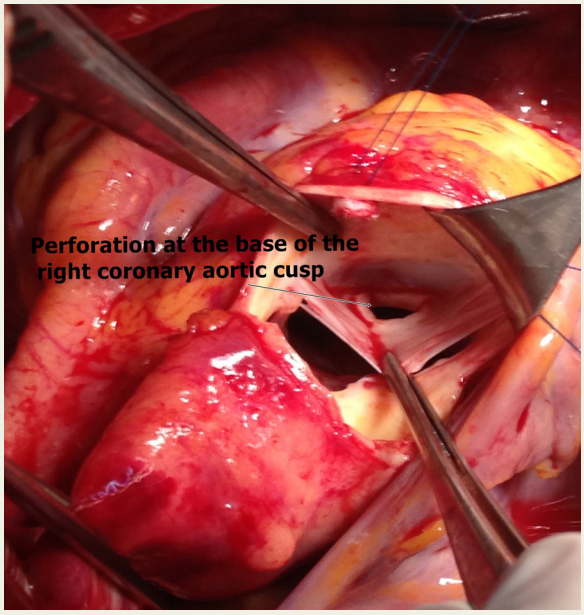


Figure 3 Perforation at the base of the right coronary aortic cusp.

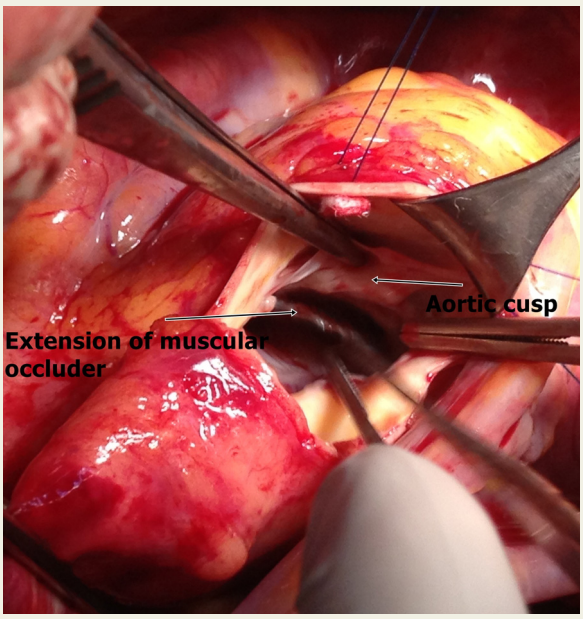


Figure 4 Extension of muscular occluder to the aortic valve and left ventricle outflow.



Figure 5 Postoperative ECHO of aortic valve and colour flow Doppler modes show no significant AR. AoV: aortic valve.

His postoperative course was uneventful. The postoperative control ECHO revealed no signs of AR and residual VSD (Figure 5). The patient was scheduled for control one month later, and was discharged.

Discussion

The standard treatment of pmVSD is open heart surgery, which requires CPB, and has minimal operative mortality and morbidity. The transcatheter pmVSD closure has become an alternative to open heart surgery due to its noninvasive nature. However, due to complications including heart block, valve problems, and occluder migration, physicians have certain doubts over transcatheter pmVSD closure (5, 6 and 7). To avoid these complications, eligibility criteria for transcatheter closure, in addition to the changes in occluder design, have been redefined [8]. Nevertheless, several cases that resulted from the catheter perforating the cusp or the occluder pinching the cusp, and requiring open heart surgery have been reported. Diagnosis is usually made during the aortography after occluder insertion (5.7). In the current case, the reason for AR was the perforation of the right coronary cusp due to the migration or expansion of the anterior-superior region of the occluder's left ventricle disk close to the aortic valve in the left ventricle outflow tract. The authors could not identify such a complication in the literature. The aortic valve perforation might be done by the occluder expansion due to its thermal memory. However, in the current case, occluder disk malrotation into the outflow tract was not been precisely reported. It is noteworthy that theVSD in the present case was considered to be malaligned.

The cardiologist preferred to use an Amplatzer muscular occluder for pmVSD closure in the current patient. Such a choice was made, because the aortic rim of VSD was estimated to be sufficient during the echocardiography prior to transcatheter closure.

The treatment strategy for occluder-associated aortic valve regurgitation is to remove the occluder, close the pmVSD, and then resolve the valve pathologies. In the early stages, the occluder can be removed by transcatheter extraction or at times necessitate surgery. In the late stages, occluder removal without damaging the neighbouring structures is difficult because of the extensive adhesions around the occluder. In these cases where the removal of the occluder is not possible, such as in the current case, excessive traction should not be applied to avoid damage to neighbouring tissues. For this reason we decided to resect the anterior-superior region of the left disc of device to prevent its contact with the valve. This can be an alternative method in cases where occluder removal is not possible. Potential complications of this technique are the exposure of a thrombogenic surface and the potential for occluder migration due to disruption of occluder integrity. To prevent these complications, we recommend closing the occluder, together with the VSD region, with an autologuous pericardial patch.

During transcatheter VSD closures, it is important to comply with the appropriate indications for transcatheter

closure, and to use a membranous occluder, especially in pmVSDs, to avoid possible potential complications. In addition, in case of aortic valve regurgitation, early surgery should be performed. Early surgical intervention may prevent the need for possible aortic valve replacement and can facilitate removal of the occluder.

Conclusion

For transcatheter pmVSD closures, membranous occluder should be the choice to avoid potential measurement errors, in spite of optimistic echocardiographic evaluation. AR after transcatheter closure should be considered an indication for early surgery. In cases in which the occluder cannot be removed, partial occluder resection and the closure of VSD by autologuous pericardial patch should be considered as an alternative surgical approach.

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