

Transcatheter Closure of Iatrogenic Perimembranous Ventricular Septal Defect following Surgical Tricuspid Valve Replacement in Patient with Congenitally Corrected Transposition of the Great Arteries

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Abstract

Iatrogenic membranous ventricular septal defects (VSD) are rare complications of cardiothoracic surgery, most commonly seen as a complication of aortic valve replacements. An iatrogenic VSD can lead to right sided heart failure, systemic hypoxia, and arrhythmias, and closure is often necessary. Considering the increased mortality associated with reoperations, percutaneous transcatheter closure of these iatrogenic VSDs has increasingly become the preferred choice of therapy. We describe the case of iatrogenic perimembranous VSD closure following surgical tricuspid valve replacement in a patient with the congenitally corrected transposition of the great arteries, using the HeartR ASD Occluder (Lifetech Scientific (Shenzhen) Co., Ltd.).

Keywords: Tricuspid valve replacement; Heart septal defects; Ventricular; Iatrogenic disease; Septal occluder device

Background

A hemodynamically significant Ventricular Septal Defect (VSD) following aortic valve replacement is rare, but represents an important complication. Reoperation to repair an iatrogenic VSD entails a high surgical risk since it involves another surgery in friable tissue, but transcatheter device closure offers the possibility of a less invasive therapeutic option. We review the first described case of transcatheter closure of iatrogenic VSD following surgical tricuspid valve replacement with a mechanical prosthesis in a patient with a Congenitally Corrected Transposition of the Great Arteries (CCTGA).

Case Presentation

A 36-year-old man presented with history of CCTGA, prosthetics of a tricuspid valve with a mechanical prosthesis (MedEng №29). The surgery was complicated by complete heart block and the consequent implantation of a dual-chamber pacemaker. A routine postoperative Transthoracic Echocardiogram (TTE) revealed a perimembranous VSD estimated to be 6 mm in diameter. Pharmacological treatment was ineffective. The patient presented on eight months with deterioration. A clinical examination showed: systolic murmur on the left side of the sternum in the third intercostal space. Thorax radiography showed increased vascularity of the lungs. In postoperative period according to the control Transthoracic Echocardiogram (TTE) confirmed a

perimembranous VSD, estimated to be 9-11 mm in diameter (Qp/Qs=1.9). CT showed VSD size of 10.5 x 5mm localized in the *membranous ventricular septum* (Figure 1 and 2).

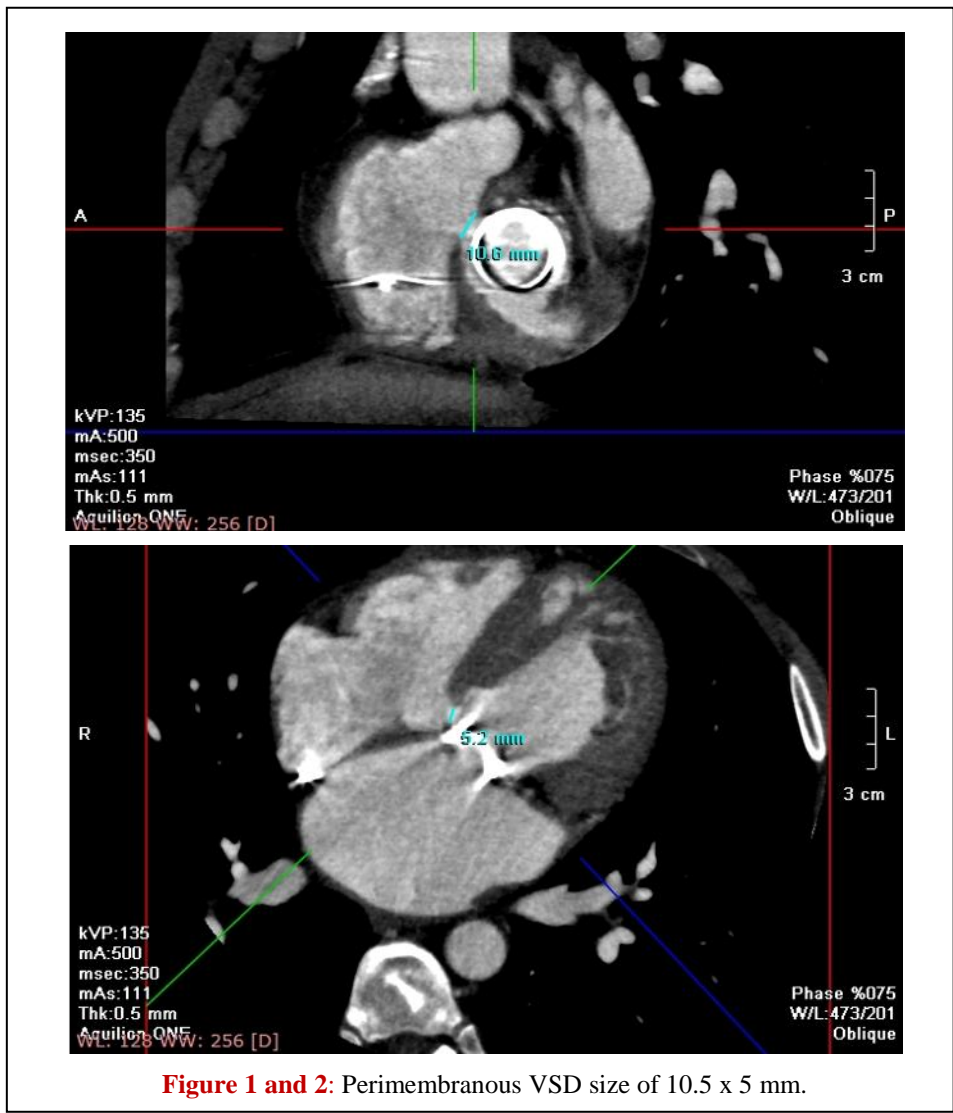


Figure 1 and 2: Perimembranous VSD size of 10.5 x 5 mm.

The procedure was performed under local anesthesia and general sedation. Then heparin (1 mg/kg intravenously) was administered systemically. The right artery and femoral vein were cannulated. Then the 4F and 6F introducer were inserted into the femoral artery and vein, respectively. The aortic valve was crossed by retrograde approach using a 260 cm 0.035” *inQwire^R* guide wire on which a 4-F pigtail was advanced into the arterial ventricle cavity. Angiography in the Left Anterior Oblique (LAO 45) form was confirmed by the VSD, which was located in the perimembranous part of the arterial ventricle inflow. Cardiac catheterization showed an 8-10 mm perimembranous VSD. Right atrial pressure 12 mmHg, Pulmonary Artery (PA) 43/12 (20) mmHg, ABP 129/80 (90) mm Hg. Subsequently, using a JR 5-F diagnostic catheter, the Terumo wire was guided across the VSD to the RV (anatomically LV) and then to the right pulmonary artery where the distal was snared and withdrawn from the femoral vein thus establishing an arteriovenous loop. The JR catheter was exchanged for an 8 Fr sheath introducer and a size HeartR ASD-008 Occluder (Lifetech Scientific (Shenzhen) Co., Ltd.) was implemented through that sheath. The size of the device was determined by the diameter of the VSD obtained by TTE (9-11 mm) and angiographic measures (8–10 mm (Figure 3-5). Intervention was guided transthoracic echocardiography in addition to fluoroscopy. The total duration of the procedure was 2 h, including 41.4 min of fluoroscopy.

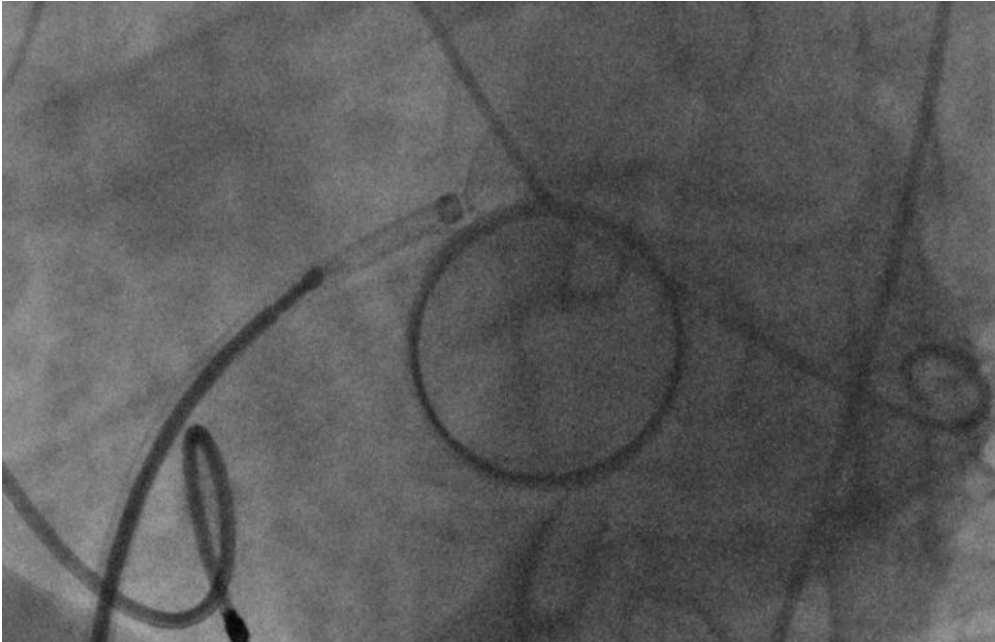


Figure 3: Releasing the left side disk of HeartR ASD Occluder. Arrow shows the position of the occluder (the left side disc was released from the sheath).

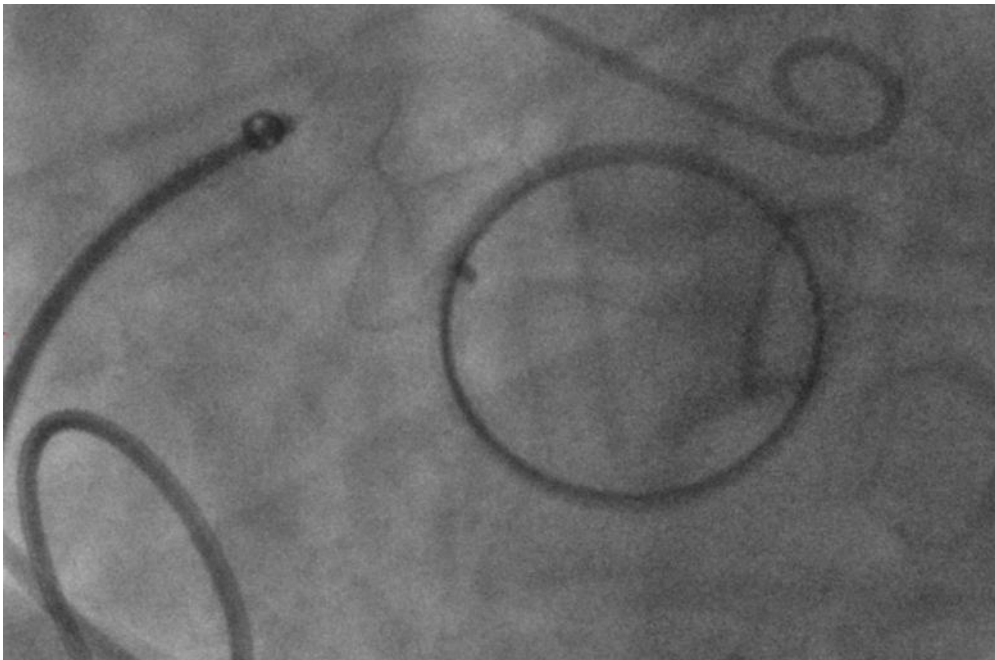


Figure 4: HeartR ASD *occluder* to close selected perimembranous VSDs.

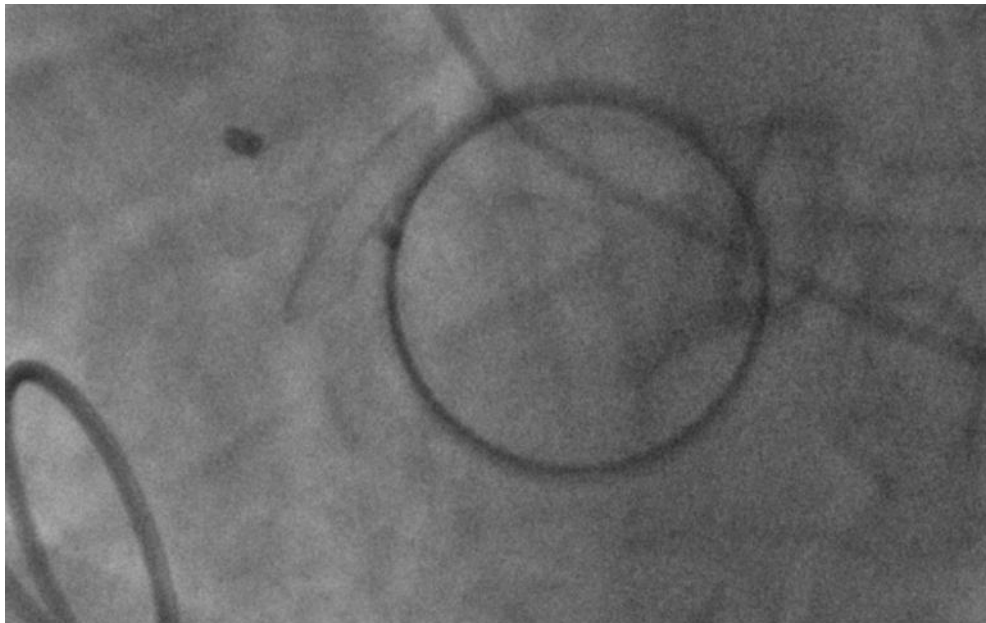


Figure 5: The occlusion is finished.

Discussion

To the best of our knowledge, this is the first description of endovascular closure of iatrogenic PmVSD following surgical tricuspid valve replacement in patient with CCTGA [1]. Ventricular septal defect is relatively rare in adult population. Acquired VSD can be caused by trauma, myocardial infarction or previous cardiac surgeries such as valve replacements or VSD closure attempts. A left-to-right shunt can result in significant LV overload and symptoms of heart failure. Shunts <1.5 are often well tolerated, although larger shunts need to be corrected [2]. Closure is also needed to prevent endocarditis, pulmonary hypertension, arrhythmias, and LV dysfunction. Surgery, for many years, has been considered as the gold-standard treatment but with a significant morbidity and mortality especially in patients with prior sternotomies [3]. Since the first percutaneous closure of VSD performed by Lock in 1988 [4], along with the development of many closure devices and deployment techniques, percutaneous PmVSD closure was also commonly followed in many institutes worldwide [5-8]. However, widespread use of these conventional PmVSD closure devices has been limited recently by the unacceptable high rate of Complete Heart Block (CHB) compared with surgical repair. Therefore, the search for alternative devices with good performance and low complication rate, especially CHB, is crucial in clinical practice. In our case, we presented the case of VSD closure after prosthetic tricuspid valve replacement in a patient with CCTGA. It also known as levo- or L-loop transposition (L-TGA) is a rare cardiovascular anomaly with inversion of the ventricles and great arteries. Patients with CCTGA have the anatomical right ventricle as their systemic pumping chamber, with ventricular dysfunction and CHF being relatively common in older adults. Tricuspid valvular regurgitation as in our patient is strongly associated with RV (anatomical right ventricle connected to aorta in CCTGA patients; systemic ventricle in CCTGA); whether it is causative or a secondary complication remains speculative.

To close the iatrogenic perimembranous VSD, we decided to use the Lifetech device is called the HeartR ASD Occluder. The device is made of braided Nitinol threads. Nitinol is a very elastic metal with wide-ranging memory properties. The device consists of two discs with a larger intermediate waist. Inside each of the discs, there is a Polyethylene (PET) patch, to support the immediate closure. The device was an alternative for this patient with the heart defect (Figure 5).

Conclusion

This case demonstrates that percutaneous repair of post cardiac surgery PmVSD using occluder device can be performed successfully with excellent immediate and long-term outcomes even in challenging localization and atypically anatomy of the

heart. The transcatheter closure of iatrogenic VSD can be considered as alternative to surgical correction, especially in high-risk patients of reoperations.

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Citation of this Article

Serezha M. Transcatheter Closure of Iatrogenic Perimembranous Ventricular Septal Defect following Surgical Tricuspid Valve Replacement in Patient with Congenitally Corrected Transposition of the Great Arteries. *Mega J Case Rep.* 2023; 6: 2001-2005.

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