



## Case Report

## Transcatheter closure of iatrogenic Gerbode defect with an Amplatzer duct occluder in a 23-year-old patient



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## ABSTRACT

A 23-year-old man was referred to our center with hematuria and hemolysis. The patient had undergone mitral and tricuspid valve replacement 3 months previously. Echocardiography and catheterization revealed a Gerbode-type ventricular septal defect. A decision was made to occlude the defect interventionally. The patient's hematuria ceased immediately after the occlusion of the defect.

**<Learning objective:** Iatrogenic ventricular septal defects (especially Gerbode-type) are relatively rare complications after valvular surgery. Correction of such defects can be done both surgically and interventionally, but since the risk of another operation for correction is high, percutaneous ventricular septal defect closure is usually the preferred treatment option. Using an appropriate approach will increase the success rate.>

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## Introduction

The transcatheter closure of ventricular septal defects (VSDs) is a well-described method for the closure of certain types of VSDs.

Gerbode-type defects are more complex for interventional approaches, and there is a paucity of information in the existing literature on intervention for iatrogenic Gerbode defects [1,3]. We herein introduce a patient with an iatrogenic Gerbode defect after mitral and tricuspid valve replacements (as the second open heart surgery) whom was hematuric due to hemolysis.

## Case report

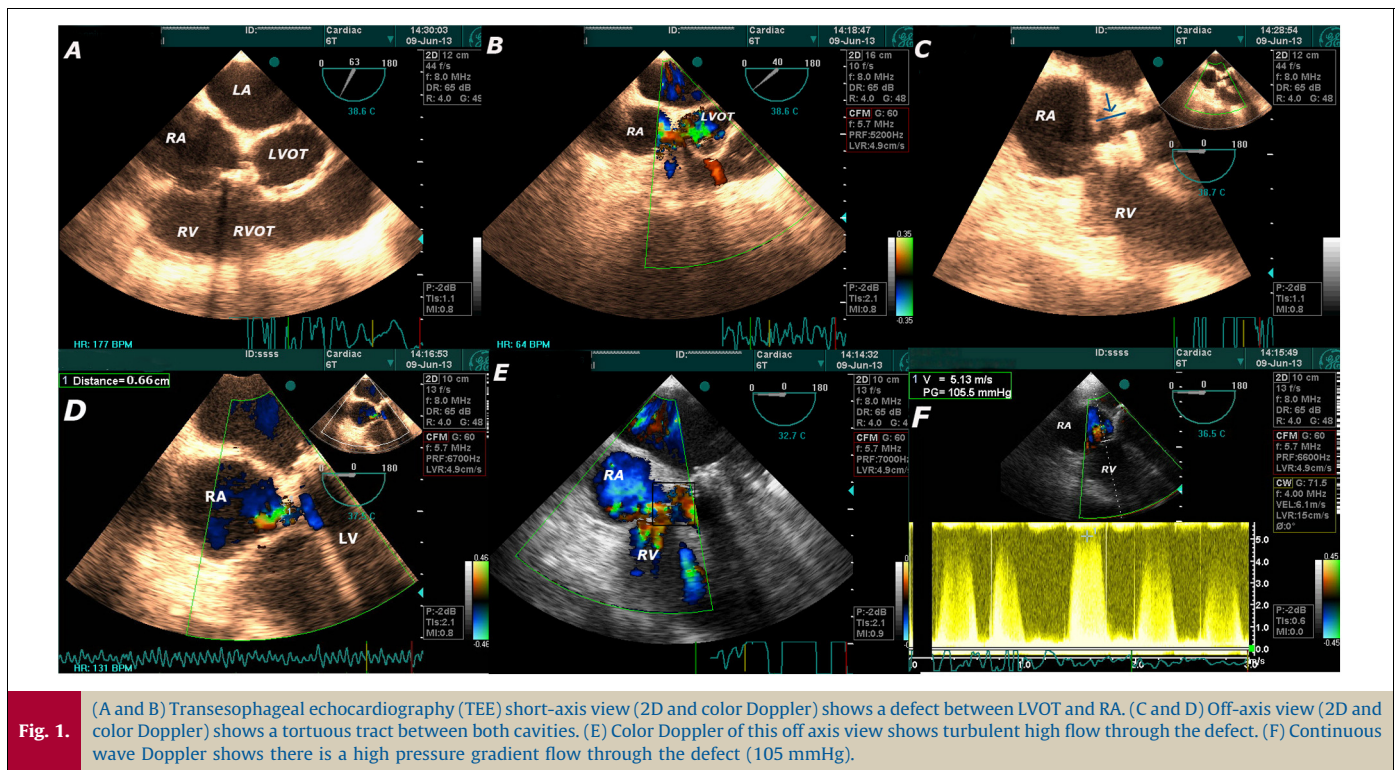
A 23-year-old man presented with hematuria 3 months after the second valvular surgery. Hemolysis was determined as the etiology of the hematuria after paraclinical evaluation, revealing hemoglobin of 8.1 with normal mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) along with hemoglobinuria, raised lactate dehydrogenase (LDH: 680 IU/L) and indirect bilirubin (Indirect bili.: 3 mg/dL), and presence of schistocytes (1.2%) in the peripheral blood smear (compatible with moderate hemolysis). Physical examination revealed loud pansystolic

murmur at the left sternal border along with S1 and S2 and closure clicks of the mechanical valve. Transesophageal echocardiography showed a Gerbode-type VSD with size about 6.5–7 mm and high pressure gradient flow (105.5 mmHg) (Fig. 1). Left ventricle (LV) and right ventricle (RV) sizes were normal, and there was mild RV systolic dysfunction. The mechanical prosthetic valve which was placed in mitral position was normal but moderate tricuspid valve regurgitation (TR) was present. The Gerbode defect was considered as the cause of the hemolysis (i.e. traumatic hemolysis). The Qp/Qs was estimated 1.1 by catheterization and the pulmonary artery pressure was 30/10 mmHg. LV contrast injection illustrated blood shunting from the LV to the right atrium (RA) with a normal LV systolic function. We decided to close the defect by percutaneous interventional approach.

Given the defect size and shape, an Amplatzer duct occluder, size 10–8, was chosen. We tried antegrade approach and used a 7 F sheath (Fig. 2). Under fluoroscopic and transesophageal echocardiography guidance, the sheath tip was positioned at the correct location in the LV. By pulling back the sheath, the disc of the device was expanded up to its tubular stent part. The whole system was then repositioned and withdrawn slightly, enabling the stent part of the device to close the defect with the disc lying on the LV side of the defect near the left ventricular outflow tract (LVOT). At this point, multiple LV contrast injections along with transesophageal echocardiography guidance confirmed the correct position of the device and absence of any blood shunting and impingement of nearby structures. Pulling back the introducer sheath the device

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was released from the cable to occlude the VSD with no residual shunt (Fig. 2).

After the procedure, the patient had a stable hospital course and the hemolysis and resultant hematuria subsided with normal long-term follow-up.

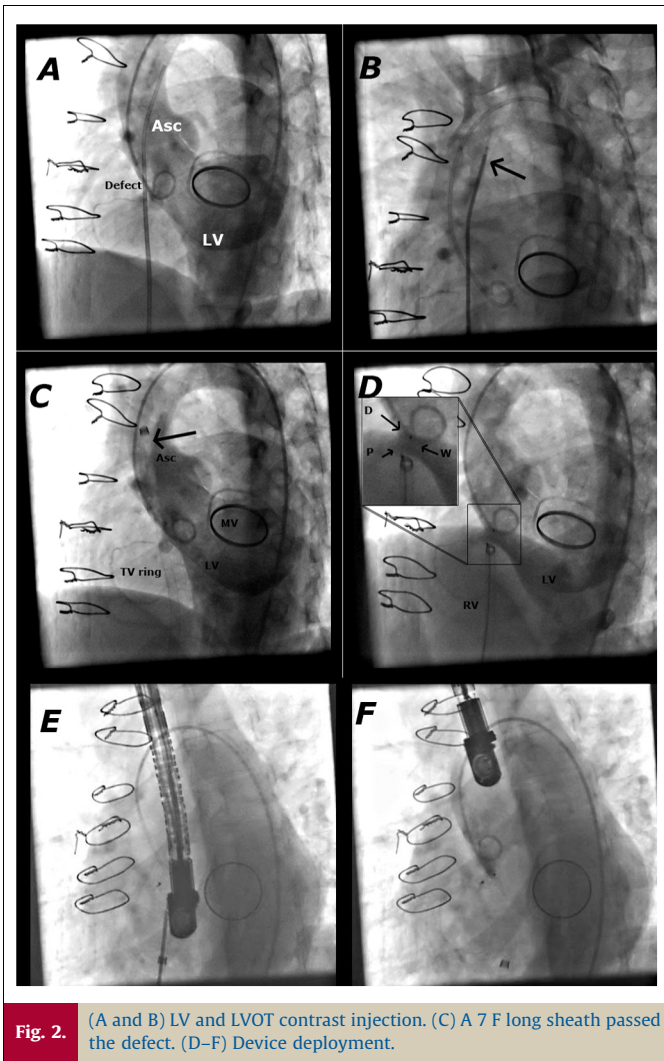
## Discussion

Although iatrogenic Gerbode defects are rare, the hemodynamic consequences of such defects can cause progressive congestive heart failure with high mortality rates if left uncorrected. So it appears that the correction of this type of defect, irrespective of clinical symptoms and shunt degree, is necessary [2]. To avoid creating this defect (i.e. as a prophylactic measure), the operating surgeon must exercise caution so as not to injure the membranous septum during aortic or mitral valve replacement, especially if vast calcium debridement is needed. Also the surgeon should examine the IVS to find a thrill that is a sign of such defect, if it was created during the procedure [2]. These patients are at a relatively high risk for surgical correction since they have undergone previously a surgical operation with a relatively short time interval for the second operation.

Rothman et al. reported a patient with iatrogenic Gerbode defect who presented with dyspnea 6 months after a second cardiac surgery for mitral valve replacement [1]. They used an Amplatzer duct occluder (size 10–8) and chose the retrograde approach. In contrast to our patient, in their case hemolysis had started after device placement which was resolved spontaneously. In two cases reported by Matsumoto et al. [4], non-Gerbode-type iatrogenic VSDs after surgical aortic valve replacement were corrected interventionaly. The authors mentioned that the deployment of the device in such defects was not possible via the anterograde approach, because of the angulated anatomy and trabeculation of the RV; although retrograde approach could not be possible because of presence of mechanical prosthetic valve in

aortic position, so the trans-septal approach was tried. In this approach, after atrial septostomy was performed and a balloon floating catheter was placed in the LV. The catheter was then crossed through the VSD and entered into the PA. Then a wire was passed into the PA and snared in the PA to form a venous arterial venous loop to insert a sheath and deploy the device over the venous side.

Traditionally surgery has been the treatment of choice for iatrogenic non-coronary fistulous pathways. Because percutaneous repair is considered as an off-label method for these defects, there is no consensus on the optimal approach. Appropriate sizing of the device and device type, choosing the best approach for delivery system and positioning the device are the most important issues. As Gerbode defects are relatively longer and are more probable to be tortuous than muscular and membranous types the Amplatzer duct occluder is more suitable than VSD occluder devices in such defects. Also a sizing balloon can be used in order to find the most accurate device size although it is not mandated because it can injure adjacent structures by itself. Choosing between antegrade or retrograde approaches for the delivery system depends on operator's experience, anatomy of the defect, and patient's peripheral vascular conditions. We opted for antegrade approach to avoid snaring, which may cause damage to the nearby structures due to tension in the arteriovenous loop. In our opinion, the antegrade approach should be considered as the first method, and only if this approach fails should we contemplate the retrograde approach as a second option (considering the presence of certain types of prosthetic aortic valves). Multiple contrast injections along with TEE guidance can reduce the risk of device malposition and its consequences, valvular regurgitation, obstruction of major veins (IVC or SVC), erosion of nearby structures, and hemolytic anemia. One potentially lethal condition is device loss which can cause obstruction of blood flow of large vessels and even LVOT. This can also be avoided by appropriate sizing and placement of the device.



There are many other case reports in which hemolysis was initiated after the VSD closure. Also hemolysis after prosthetic valve implantation (whether bioprosthetic or mechanical valves) is one of the major possible complications. Therefore, although our patient's hemolysis was relieved after the procedure, there was an unrecognized probability of the aggravation of hemolysis after the procedure. Indeed, the appropriate sizing and placing of the device to avoid residual shunting and flow turbulence nearby may contribute to the elimination of hemolysis.

### Conclusion

Iatrogenic Gerbode defects must be occluded. The transcatheter correction of an iatrogenic Gerbode defect can be done by an experienced operator via the antegrade approach, considering the same rules and techniques for the occlusion of congenital muscular and membranous VSDs.

### Conflict of interest

None declared.

### References

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