Partial Tricuspid Valve Stenosis during Device Closure of Perimembranous Ventricular Septal Defect

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INTRODUCTION

We present two cardiac catheterization cases performed to close perimembranous ventricular septal defects (pmVSDs), in which, during each case, the delivery sheath created tricuspid valve (TV) obstruction that resulted in hemodynamic instability, evidenced by real-time echocardiographic imaging. Hypotension occurred after crossing the pmVSD with the delivery sheath, and intervening action by the operating physician was needed. The echocardiographic images demonstrated right atrial distension, leftward deviation of the atrial septum, and tethering of the TV anterior leaflet.

Closure of pmVSD requires creation of an arteriovenous (AV) loop, described here. The ventricular septal defect is crossed from the left ventricular side with a catheter, and a guide wire is advanced into the pulmonary artery. A snare catheter is advanced from the femoral vein, and the guide wire is withdrawn from the femoral venous sheath. The delivery sheath is advanced over the guide wire from the femoral vein into the left ventricle before advancing the device through the delivery sheath. A fall in blood pressure while advancing the delivery sheath over the wire has been described and attributed to the sheath resulting in tricuspid valve leaflet immobility and resultant stenosis, but it has never been documented. Our images clearly document atrial septal bowing toward the left and right atrial distension because of TV obstruction caused by tension on the AV loop with the delivery sheath and the subsequent reversal of this obstruction and the hemodynamic consequences of loosening the AV loop.

This is the first documented description of TV obstruction and hemodynamic instability occurring after advancing a delivery sheath across a pmVSD defect.

CASE PRESENTATION

Case 1 is a 15-month-old child with dextrocardia and situs inversus with a pmVSD. During the transcatheter procedure to perform a device closure of the pmVSD, an AV loop was formed. When a 7F T orqueVue Amplatzer (Abbott, Plymouth, MN) delivery sheath was advanced across the TV and pmVSD, the patient became hypotensive and hypoxic. The previously neutral atrial septum position deviated toward the left atrium secondary to TV obstruction created by the delivery sheath, as illustrated in Videos 3 and 4 (see also Figures 3 and 4). With release of tension on the AV loop, the atrial septum returned to normal position (Video 4, Figure 4) and blood pressure and oxygen saturation returned to normal.

TorqueVue Amplatzer (Abbott, Plymouth, MN) delivery sheath was advanced across the TV and pmVSD. There was an immediate fall in blood pressure. The TV anterior leaflet opened incompletely with simultaneous right atrial enlargement and stasis (Video 1, Figure 1). Once the device was released, the TV opened normally, blood pressure rose, and right atrial size decreased (Video 2, Figure 2).

Case 2 is a 2-year-old child with normal situs and a pmVSD. During the transcatheter procedure to perform a device closure of the pmVSD, an AV loop was formed. When a 7F TorqueVue Amplatzer delivery sheath was advanced across the pmVSD, the patient became hypotensive and hypoxic. The previously neutral atrial septum position deviated toward the left atrium secondary to TV obstruction created by the delivery sheath, as illustrated in Videos 3 and 4 (see also Figures 3 and 4). With release of tension on the AV loop, the atrial septum returned to normal position (Video 4, Figure 4) and blood pressure and oxygen saturation returned to normal.

DISCUSSION

This is the first documentation of TV stenosis with right atrial enlargement and deviation of the atrial septum toward the left atrium that occurred when a delivery sheath was advanced over an AV loop.
Figure 1  Still image from Video 1. Diastolic frame before device release in a patient with dextrocardia and situs inversus, demonstrating an open mitral valve below the left atrium (LA), a TV held in a closed position (presumably by the delivery sheath), and an enlarged right atrium (RA). RV, Right ventricle.

Figure 2  Still image from Video 2. Diastolic frame in a patient with dextrocardia and situs inversus after device release demonstrating an open mitral valve below the left atrium (LA), a TV in an open position, and a right atrium (RA) decreased in size. RV, Right ventricle.

Figure 3  Still image from Video 3. Transesophageal echocardiogram prior to AV loop formation demonstrating rightward bowing of the atrial septum (arrow) at normal atrial pressures. LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
across a pmVSD. In each instance, the obstruction was relieved with repositioning of the delivery sheath. Cyanosis in the second case was presumably due to a patent atrial communication with a right to left shunt. Hemodynamic instability and cyanosis are known to occur during balloon dilation valvuloplasty for pulmonary valve stenosis (right ventricle “outflow obstruction”). Hemodynamic instability during transcatheter pmVSD closure is usually attributed to valvar insufficiency (aortic insufficiency or tricuspid regurgitation) or simply to cardiac distortion from a large stiff delivery system. These images illustrate that TV obstruction by the delivery system (right ventricle “inflow obstruction”) is a reversible cause of hypotension with or without cyanosis that can be relieved without abandoning the transcatheter closure, and when hypotension occurs, attention of ultrasonographers should be directed immediately to the TV and atrial septum. Simply reducing tension on the AV loop or reestablishing the AV loop will allow completion of closure.

It is known that passing through right ventricular trabeculations can prevent advancing the delivery sheath through the right ventricle. The mechanism of TV tethering by the delivery sheath is probably due to passage behind TV chordae when establishing the AV loop. Using a balloon-tipped catheter to pass from the vena cava to pulmonary artery and a large curve J-tipped guide wire through the aortic catheter across the pmVSD to enter the pulmonary artery may reduce the likelihood of this complication.

**CONCLUSION**

This is the first description of TV obstruction and hemodynamic instability occurring with advancing a delivery sheath across a pmVSD defect. This problem may occur more frequently than recognized if there is only mild obstruction or if there is a larger atrial communication. In addition to using TEE to monitor the position of the device during implantation, it is important to use TEE to examine the TV when hypotension and/or cyanosis occur with advancing a delivery sheath across a pmVSD.

**SUPPLEMENTARY DATA**

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2020.04.007.

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