Transcatheter Closure for Acute PIVSD

Preprocedure management and current techniques used in this challenging procedure to treat postinfarction ventricular septal defect.

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An acute postinfarction ventricular septal defect (PIVSD) is a perforation or rupture of the muscular ventricular septum occurring in an area of acutely infarcted myocardium. PIVSD develops in approximately 1% to 3% of patients who do not undergo reperfusion therapy and in approximately 0.2% to 0.3% of those who do undergo reperfusion therapy.[1,2] Clinical signs of PIVSD (loud systolic murmur at the left lower sternal border with thrill) typically develop approximately 1 to 2 days after acute myocardial infarction (AMI). The time of occurrence of PIVSD tends to have a bimodal peak, with the appearance of the defect between 24 hours and 5 days.

Ventricle rupture can be classified into simple and complex types. Simple rupture consists of a direct through-and-through defect. Usually, this type of defect is located in the anterior part of the septum. Complex rupture consists of a serpiginous dissection tract that is remote from the primary perforation (Figure 1). These defects are typically located in the inferior part of the septum.[3] In some instances, multiple defects may develop. Transesophageal echocardiography (TEE) is ideal for determining the location and size of the defect, ascertaining right and left ventricular function, and estimating pulmonary artery pressures.[4]

The American College of Cardiology's current guidelines recommend immediate surgical closure of the VSD after AMI regardless of the clinical status of the patient (class I recommendations).[5]

Surgery or intervention is indicated in patients who experience cardiogenic shock, which constitutes a true emergency. Death in these patients results from multisystem organ failure secondary to hypoperfusion; hence, intervention is recommended at the outset. A small percentage of PIVSD patients are completely stable.[6] In these patients, intervention can be safely postponed for 3 to 6 weeks. However, a significant number of patients fall into the intermediate group; these patients are not in shock but require support and should undergo intervention in the first few days of rupture because they have congestive heart failure with a rising blood urea nitrogen level and marginal urine output. Routine use of an intra-aortic balloon pump, whenever technically feasible, frequently results in transient reversal of hemodynamic deterioration.[7] Unfortunately, surgical closure of PIVSDs carries a high morbidity and mortality rate, particularly when associated with cardiogenic shock.[8] The presence of friable tissue surrounding the area of ischemia makes surgical closure challenging, requiring ventriculotomy, which results

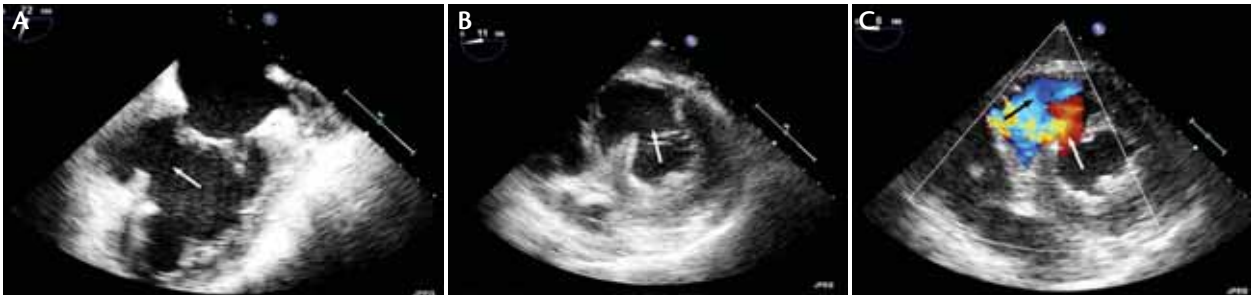

Figure 1. Complex VSD close to the mitral valve with serpiginous pathway. This is not a suitable defect for device closure. PIVSD in modified four-chamber view. The defect (white arrow) is just below the mitral valve leaflet (A). Short-axis view that shows the VSD (white arrow) opening into a chamber formed by the splitting of the ventricular septum. Note the right ventricle cavity is father away from the true defect (black arrow pointing toward the opening into the right ventricle) (B, C).

(Courtesy of St. Jude Medical, Inc.)

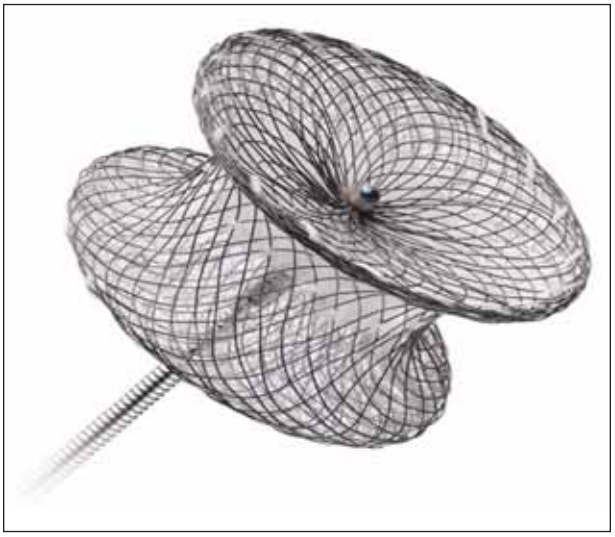

Figure 2. Amplatzer postinfarction muscular VSD occluder (St. Jude Medical, Inc., St. Paul, MN). The design is similar to the congenital muscular VSD device.

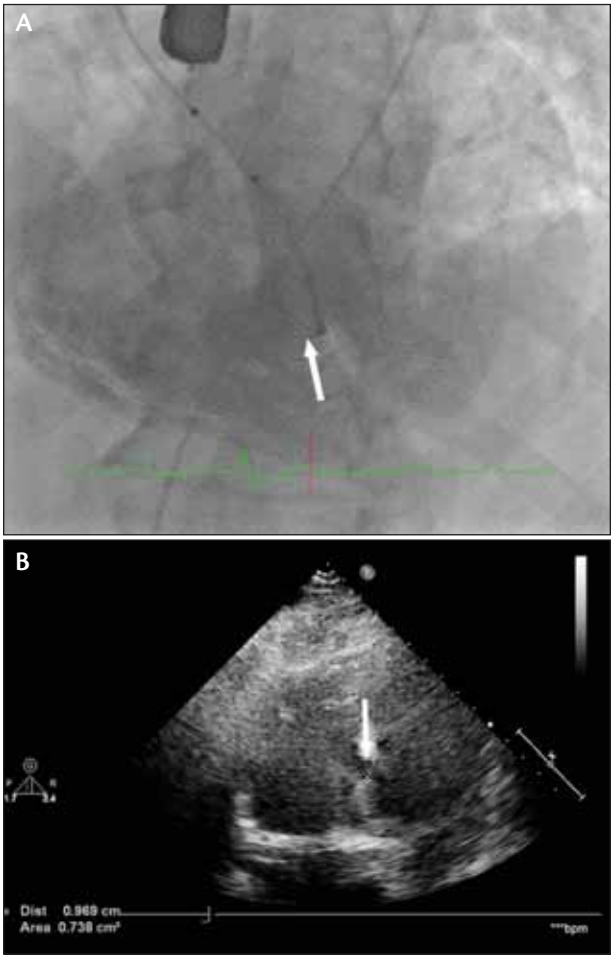

Figure 3. Left ventricular angiogram with the camera in left anterior oblique view with cranial tilt. The arrow shows a muscular VSD located in the middle of the septum (A). TEE image corroborates the angiographic findings of a PIVSD; the white arrow highlights the VSD (B).

in further deterioration of function. Despite adequate initial patch closure of the defect, the risk of residual VSD is high.[9]

Transcatheter closure (TCC) has emerged as an alternative treatment in patients with PIVSD. It has been shown to be relatively safe and effective for primary closure and for residual defects.[10-13] When compared to surgery, TCC is a less-invasive procedure that does not require sternotomy, cardiopulmonary bypass, or ventriculotomy and therefore seems to have an advantage when compared to surgery. However, given the critical status of most of these patients, it is not a straightforward procedure. The VSDs in these patients are not simple, circumscribed, and uniform as are congenital VSDs.[14] Pediatric interventional cardiologists are better trained to close VSDs, but the hemodynamic compromise in elderly patients is beyond the realm of their care. Therefore, a collaborative approach for TCC of PIVSD is highly desirable, wherein pediatric interventionists and adult interventionists work hand in hand and improve procedure success rates.

PREPROCEDURE MANAGEMENT

The primary goal of preprocedure management is to improve organ perfusion, decrease left-to-right shunting through the VSD, improve coronary blood flow, and decrease afterload. Therefore, the use of an intra-aortic balloon pump plays an integral role in preprocedure management of hemodynamically unstable patients. Another technique that has been used is venoarterial extracorporeal membrane oxygenation. Nevertheless, every effort should be made to improve the patient's hemodynamic status in the shortest possible time to avoid delay in closing the defect.

DEVICES

The first TCC of a PIVSD was reported in 1988 with the Rashkind double umbrella.[15] Subsequently, the CardioSeal device (formerly manufactured by NMT Medical, Boston, MA) (not currently available) was approved by the US Food and Drug Administration for high-risk protocol. The Amplatzer postinfarction muscular VSD occluder was introduced in the late 1990s and became the device of choice for many interventionists (Figure 2). It was the only device designed to close PIVSDs.[16] However, it is not approved by the US Food and Drug Administration and is only available under high-risk protocol. As with other Amplatzer devices, this

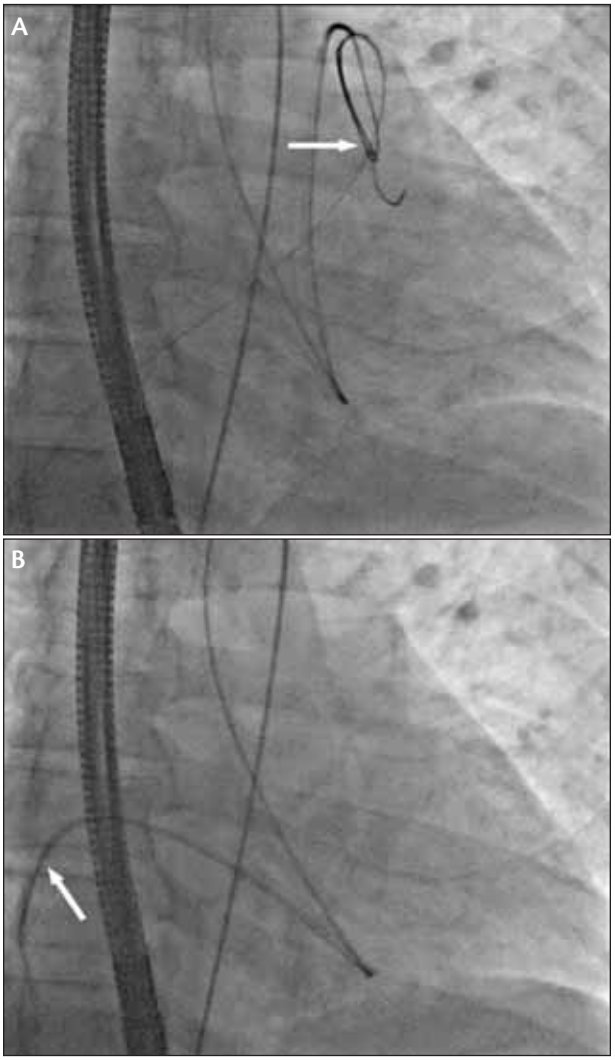

Figure 4. Wire through the VSD into the pulmonary artery. Arrow shows a snare catheter in the pulmonary artery to snare the wire and exteriorize it from the femoral-venous side, forming an arteriovenous loop (A). Arrow shows the arteriovenous loop from the femoral-venous side (B). The location of the defect was mid-muscular, which made it possible for us to close it from the femoral-venous side.

is a self-expanding, self-centering device made from nitinol. The device consists of two discs with a connecting waist. The discs are 10 mm larger than the waist throughout their circumference. To optimize effective closure, Dacron mesh is hand-sewn into the device waist and the discs. The available sizes range from 16 to 24 mm in 2-mm increments. The delivery sheath that is commonly used for closure is 9 to 12 F. Defects that are > 24 mm in diameter require an Amplatzer atrial septal defect device (an off-label use), or these patients should be referred for surgery.

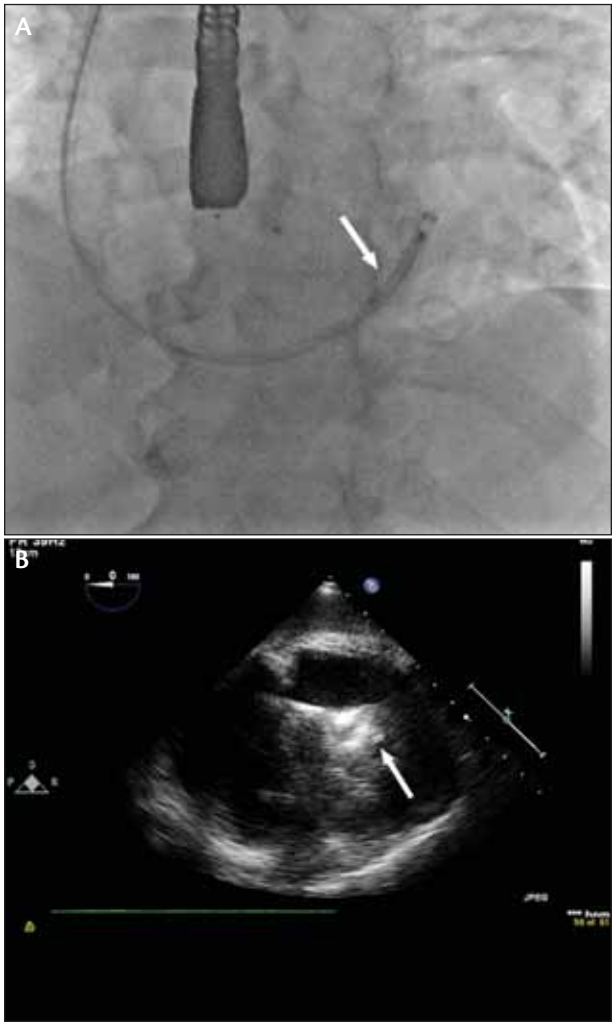

Figure 5. Arrow shows the device advanced almost to the tip of the sheath (A). The location of the defect was apical; therefore, the sheath course was optimal from the internal jugular vein. TEE image in four-chamber view with the left disc deployed in the LV (arrow) (B).

Many aspects of the device, including the mechanism of attachment of the device to the delivery cable, loading, delivery sheath, deployment, and release, are similar when treating atrial septal defect as with other Amplatzer devices and therefore will not be discussed further in this article.

PROCEDURE

As previously stated, TEE is essential in deciding which defects are suitable for device closure, as well as in guiding the interventionist in device placement. TEE is helpful in determining the size and location of the defect, the proximity of the defect to the atrioventricular valves, and in assessing ventricular septal rims for device stability.

When the AMI is secondary to the left anterior descending artery, the VSD is usually localized near the apex; however, when the AMI is secondary to the posterior descending artery, the VSD may be localized at the base of the ventricular septum (close to the diaphragm or the atrioventricular valves).[17] Determining the location of the defect assists in deciding the optimal access site (femoral vein or right internal jugular vein approach) for device closure. If the defect is close to the mitral or tricuspid valves, device closure may not be feasible because the device discs will impinge on the valves, making them incompetent. The procedure is usually performed under general anesthesia, with fluoroscopic and TEE guidance, antibiotics, and routine use of heparin recommended in all patients.

Ideally, the internal jugular vein is the best access site for closure of PIVSD because most of the defects are apical in location. The femoral vein approach can be used if the defect is anterior and high in the septum. Arterial access is essential not only for blood pressure monitoring but also to make an arteriovenous loop. At our institution, however, we access the femoral artery, femoral vein, and right internal jugular vein in all patients. Complete right and left heart catheterization is performed. Left ventricular angiography is performed in a left anterior oblique view with cranial tilt or in a hepato-clavicular view. The left anterior oblique view with cranial tilt allows assessment of the defect, and a road map can be drawn to ease in crossing the defect (Figure 3). It also helps in evaluating device position and residual shunt after device placement.

After obtaining the hemodynamics and left ventricular angiography, the VSD is crossed using a Judkins right (Cook Medical, Bloomington, IN) or multipurpose catheter (Cook Medical). Once the catheter is in or adjacent to the defect, a Glidewire (Terumo Interventional Systems, Somerset, NJ) or Storq steerable guidewire (Cordis Corporation, Bridgewater, NJ) is advanced through the defect into the pulmonary artery or the superior vena cava. The wire should not be snared in the right ventricle because such attempts may injure the chordae of the tricuspid valve. The

TROUBLESHOOTING FOR PIVSD CLOSURE

1. Cannot cross the defect from LV to RV:
   - Ensure that LV angiography adequately profiled the defect
   - Use a different catheter, such as a modified pigtail catheter or Glide catheter (Terumo Interventional Systems)
   - Use echo and fluoroscopic images simultaneously

2. Drop in blood pressure after advancing the delivery sheath in the LV:
   - Check (by echocardiography) for increase in tricuspid or mitral regurgitation secondary to chordal entrapment by the sheath
   - If the sheath is across the aortic valve, pull it back in the LV
   - Withdraw the sheath and readvance
   - As a last resort, you may have to recross the defect and recreate the arteriovenous loop

3. Cannot advance the sheath from right ventricle to LV:
   - Relieve some traction or increase traction on the arteriovenous loop
   - You may be caught on the right ventricle or tricuspid valve chordae
   - Do not force the sheath in
   - As a last resort, you may have to create the arteriovenous loop again

4. Sheath kinking:
   - This is less of a problem with the introduction of Torq-Vu sheaths (St. Jude Medical, Inc.)
   - One of the ways to avoid this is to have a little more sheath in the LV than needed; you can withdraw the sheath back while advancing the delivery cable
   - Some physicians advance a wire beside the delivery sheath, which may prevent sheath kinking (if you choose to have wire beside the cable, you will have to use a larger delivery sheath)

5. Device pulled out of the defect while deploying the left disc:
   - Thorough assessment of the defect to choose an appropriate-sized device will prevent this from occurring
   - Deploy the left disc and the waist in the LV, pull the device in the defect, and deploy the right disc; if the device pulled out because of the angle of the left disc to the VSD, this maneuver will prevent the device from prolapsing

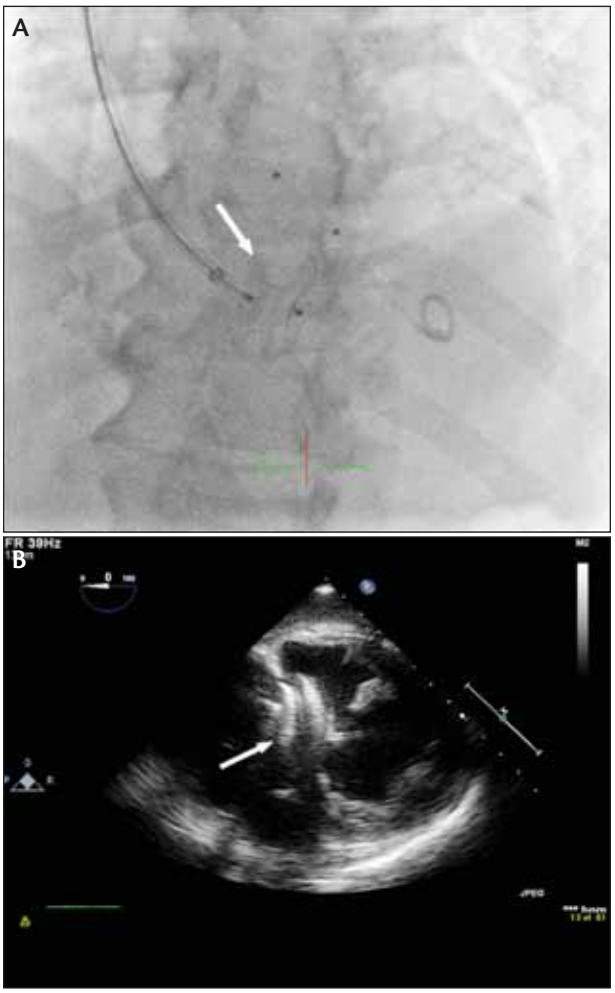

Figure 6. Deployment of the right ventricular disc while the device is still attached to the delivery cable. Arrow shows the right ventricular device (A). TEE image confirms that the device is well seated in the VSD (arrow) (B).

wire is either snared with the help of the Amplatz GooseNeck snare kit (Covidien, Mansfield, MA) or EN Snare system (Merit Medical Systems, Inc., South Jordan, UT) and exteriorized out of the jugular or right femoral vein, thereby establishing an arteriovenous circuit (Figure 4).

Careful assessment of the defect size is performed using TEE and angiographic measurements. It is recommended to use a device that is approximately 6 mm larger than the largest VSD diameter. This recommendation is crucial if the defect is being closed acutely (in the first week after AMI). The delivery sheath (St. Jude Medical, Inc.) is advanced over the wire with some traction from the arterial side. We strongly recommend advancing an endhole catheter from the arterial side across the defect. This catheter helps avoid direct wire contact with the

aortic valve and the fragile edges of the VSD and prevents any wire-related injury. As the dilator and the sheath come in contact with the endhole catheter, the endhole catheter is withdrawn while advancing the sheath. Once the dilator tip is in the left ventricle (LV), the sheath is advanced over the dilator into the LV. Some physicians advance the dilator and the sheath across the aortic valve and gently withdraw the dilator and allow the sheath to fall back into the LV. Both of the techniques are feasible. Once the tip of the sheath is in optimal location (mid-LV), the dilator and the wire are removed; the sheath is allowed to back bleed and is flushed with saline.

An appropriate-sized device is then advanced through the sheath, and the left disc is deployed in the LV. Care should be taken to ensure that the tip of the sheath is not meshed into the LV free wall or pushing on the LV apex, which may be aneurysmal and, consequently, susceptible to rupture. Subsequently, the device is advanced through the delivery sheath across the VSD into the left ventricle (Figure 5). Once the left disc is formed inside the LV cavity, part of the waist of the device is deployed. The sheath and the device are pulled back until the left disc approximates the septum. The right disc is deployed by withdrawing the sheath and while keeping traction on the delivery cable. After deployment of the right disc, the device and its relationship to the atrioventricular valves are assessed (Figure 6). Finally, left ventricular angiography is performed, and if the device appears to be in optimal position, it is released.

The experience of the operator plays an important role in this procedure; institutions without experience in TCC closure of VSDs should opt for surgical closure of these defects. Please see the *Troubleshooting for PIVSD Closure* sidebar for tips on how to navigate complicated TCC situations.

CONCLUSION

Device closure of PIVSD is an uncommon procedure. It is performed in hemodynamically unstable patients and hence is a challenging procedure, not only due to a patient's unstable status but also because of the complexity of the PIVSD. The serpiginous course of the defect, its location, and the unpredictable ultimate size of the defect make it not only technically challenging but also play a role in the critical decision-making process. In addition, the necessity to perform the procedure during the highest-risk period makes even an expert cardiologist sweat before embarking on device closure.

The currently available device comes in limited sizes, and the defect is usually larger than the largest available device for PIVSD closure. This forces interventionists to use devices on an off-label basis, which compromises the results.

Nevertheless, a combined approach between pediatric and adult interventionists, their expertise in such complex procedures, and optimal preprocedure management tends to improve patient survival and outcomes. The overall mortality rate remains high; consequently, there is definite room for improvement in terms of technique, physician education with recommendation for early referral, manufacturing of an ideal device, and enhancement of the delivery mechanism. A hybrid approach in some cases may be suitable as well (periventricular VSD closure). Our hope is that in the near future, TCC will become the procedure of choice for PIVSD, with low morbidity and mortality rates. ■

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