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CASE SERIES
Structural intervention

Apical post-infarction ventricular septal defect blockage using the direct externalization and enmeshment to the right ventricular moderator band concept: case series

Bryan Rene F. Toledano *, Ramon Miguel M. Rivera, Jose Paolo A. Prado, Michelangelo L. Sabas, and Dexter Eugene D. Cheng

The Medical City, Cardiovascular Institute, Cardiology Department, Ortigas, Pasig, 1600 Metro Manila, Philippines

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Background Transcatheter treatment in post-infarction ventricular septal defects can be unique and complex; hence, the development of a new technique is needed to improve outcomes.

Summary We describe two cases in which large and complex apical post-infarction ventricular septal defects were treated with a novel transcatheter approach as salvage and the other due to refusal for open surgical repair. By direct externalization and enmeshment of a device to the right ventricular moderator band, the defect was blocked and immediate improvement of haemodynamics was achieved.

Conclusion In large, complex, apical post-infarction ventricular septal defects with no apical rims, the DEXTER technique allows for exclusion of the defect and vestigialization of the right ventricular apex. An immediate and dramatic haemodynamic improvement can therefore be achieved.

Keywords Post-infarction ventricular septal defect • Right ventricular moderator band • Case series

ESC curriculum 7.4 Percutaneous cardiovascular post-procedure • 3.2 Acute coronary syndrome

Learning points

- To describe a novel transcatheter approach in treating an apical post infarction ventricular septal defect.
- To understand the pros and cons of the direct externalization and enmeshment to the right ventricular moderator band.

Introduction

Post-infarction ventricular septal defect (PIVSD) is an acquired life-threatening complication with a high mortality rate and 1-month survival of only 6% without intervention.[1] It has a bimodal incidence and may occur 3–5 days or later due to erosion of an infarcted myocardium (type II) or rupture from a significant thinning of a left ventricular (LV) aneurysm (type III) as classified by Becker and Mantgem.[2] These types of PIVSD commonly involve the left anterior descending (LAD) artery and are more likely to cause apical defects as it supplies this area of territory.[3,4] Current guidelines recommend surgical repair with concomitant coronary grafting as indicated if no evidence of ongoing ischaemia is suspected that warrants primary percutaneous coronary intervention strategy.[5,6] However, more often, factors that increase operative risk such as advanced age, haemodynamic instability from the shunt and or pump failure, chronic kidney disease, and sepsis are present. Transcatheter treatment

* Corresponding author. Tel: +639054776260, Email: imbryantoledano@gmail.com
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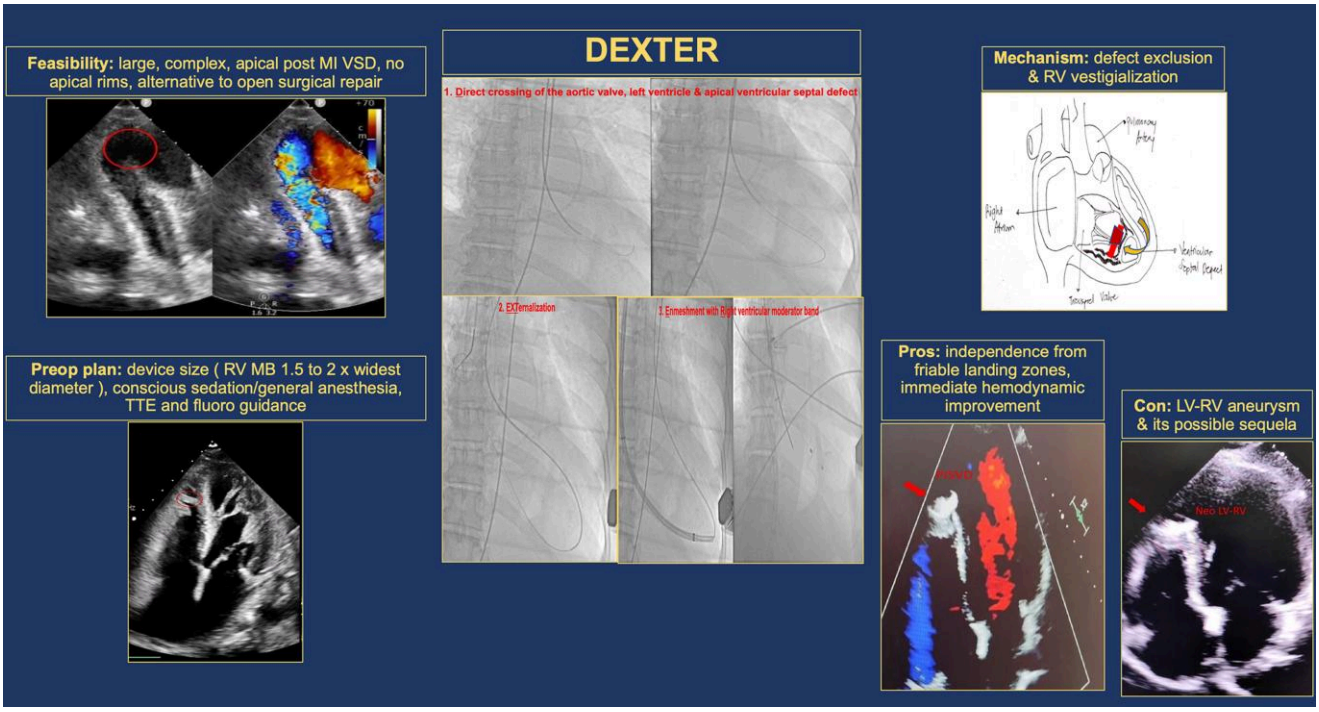
(TCT) is reserved as an alternative when open surgical repair is not feasible after thorough multidisciplinary heart discussion. As each defect can be unique and complex based on its size, number, rims, and tissue friability complications such as device embolization, major residual shunting and LV rupture may happen.[7] Hence, the development of new techniques is needed to improved outcomes in these situations.

Summary figure



Patient 1

A 70-year-old female with a past medical history of stage 2 hypertension, type 2 diabetes mellitus, and known stage 3 chronic kidney disease was referred due to symptoms of hypotension and heart failure. She experienced severe chest pain 5 days prior but did not seek medical consultation. A thrill, harsh holosystolic murmur and bilateral crackles were appreciated. Serial 12 L ECGs showed mild ST elevation in lateral and depression in the anteroseptal leads but no interval changes (*Figure 1A*). Transthoracic echocardiography (TTE) showed a normal LV ejection fraction and a small PIVSD in the apex. Coronary angiography showed a totally occluded LAD artery at its distal segment and non-obstructive lesions in other coronaries. She was referred to surgery for PIVSD repair and coronary artery bypass grafting. However, her condition quickly deteriorated, and on the 4th day of hospitalization, she was intubated with borderline normotension attained using triple vasopressors of norepinephrine, dobutamine, and vasopressin. Serial TTE showed an enlarging apical PIVSD ~1.8–2.1 cm with a Swiss cheese appearance on colour doppler (*Figure 2A*), moderate right ventricle (RV) dysfunction, and severe tricuspid regurgitation (TR). She was also being treated for hospital-acquired pneumonia with piperacillin–tazobactam and vancomycin. Severe acute kidney injury was present eGFR: 5 mL/min/1.73 m^2, along with liver injury AST: 5105 U/L (normal value 8–48 U/L) and ALT: 4803 U/L (normal value (7–55 U/L), both secondary to ischaemia. A multi-disciplinary heart team was held, and discussions on the prognosis and treatment options to her family were given; because of her current condition, her age, and the comorbidities of diabetes mellitus, hypertension, and chronic kidney disease, with high surgical mortality risk, a TCT was offered.

Patient 2

A 54-year-old male with a past medical history of stage 2 hypertension, non-ST elevation myocardial infarction (MI), and three vessel coronary artery disease, who underwent complete revascularization using percutaneous coronary intervention, presented 4 weeks after MI with a new onset harsh systolic murmur, bilateral crackles, and worsening heart failure symptoms as Killip class III. A 12 L ECG showed the sinus rhythm and an old anterolateral wall infarct (*Figure 1B*). His TTE revealed mild RV dysfunction and a PIVSD measuring 8–10 mm with a thinned out aneurysmal pouch at the apex. There was a 2nd 3–4 mm PIVSD located superiorly to the larger apical one (*Figure 2B*). The entire LV apex was also dilated and aneurysmal. Moreover, a moderate TR and mitral regurgitation were appreciated. He was admitted and maintained on low-dose nitroglycerin drip and furosemide intravenous as needed for congestion. A multi-disciplinary heart team was held and advised delayed PIVSD open surgical repair if he remained haemodynamically stable. On the 4th week of hospitalization, the patient was a good candidate for surgery; he was still haemodynamically stable with preserved LV ejection fraction (54%) and only moderate renal dysfunction (eGFR 52 mL/min/1.73 m^2). However, he strongly declined due to personal and religious beliefs.

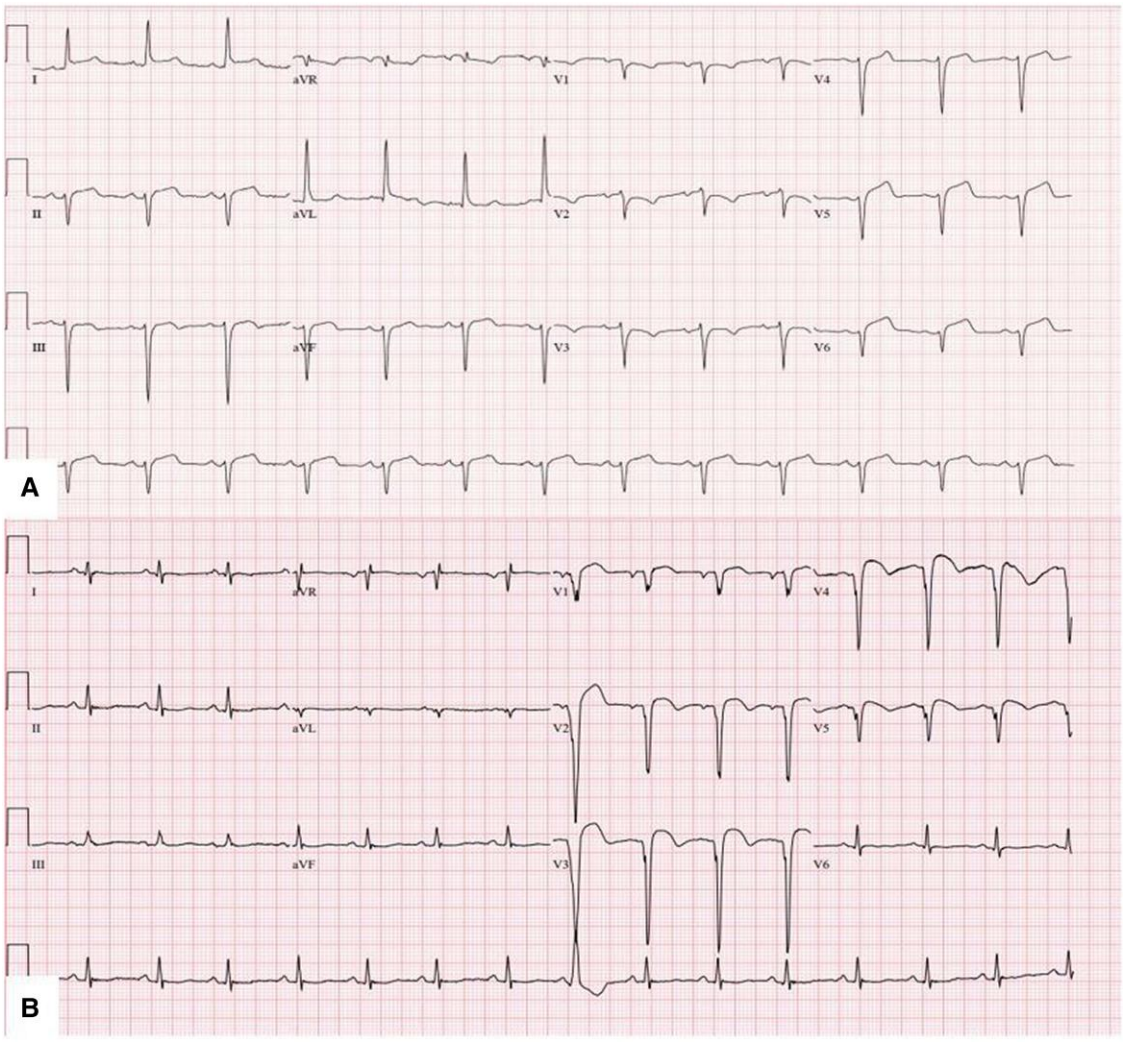


Figure 1 12 L ECG. (*A*) Patient 1 electrocardiogram demonstrating mild ST elevation in the lateral and ST depression in the anteroseptal leads. (*B*) Patient 2 electrocardiogram demonstrating sinus rhythm and old anterolateral wall infarct.

Technique

The procedure was performed in the catheterization laboratory under general anaesthesia, with TTE and fluoroscopic guidance. The right femoral artery (FA) and right internal jugular vein (IJV) were accessed with 5 F and 14 F introducer sheaths, respectively. Under fluoroscopic guidance, a 5 F JR4 catheter over a 0.035" 260 cm Terumo glidewire in the right FA was manoeuvered to cross the aortic valve and LV. The JR4 catheter was carefully advanced and positioned in the LV apex so as not to disrupt the aneurysmal wall. The catheter was pointed medially to direct the glidewire, gently across the PIVSD, RV moderator band (MB), and tricuspid valve (TV) (*Figure 3A*). The glidewire is expected to preferentially move through the largest defect in the PIVSD and RV MB as it follows the path of the highest shunt flow. The wire position from the LV apex, large septal defect, across the middle segment of the RV MB to the TV was verified using apical and parasternal views. Once the wire position is confirmed, the JR4 catheter is advanced over the wire until it is seen across the RV MB. This was done under fluoroscopic guidance to ensure that a stable wire position is always maintained. The glidewire was parked near the right atrium–superior vena cava junction, where it was snared via the right IJV and externalized to create an arteriovenous (AV) loop (*Figure 3B*). During externalization, the JR4 catheter should be across the PIVSD to prevent inadvertent tissue tears. A clamp is placed on the distal glidewire at the hub of the JR4 catheter to prevent accidental loss of wire control on the arterial side. A 12 F-long patent ductus arteriosus (PDA) delivery sheath was tracked over the venous side of the AV loop into the 14 F introducer sheath. Under fluoroscopic guidance, the 12 F delivery

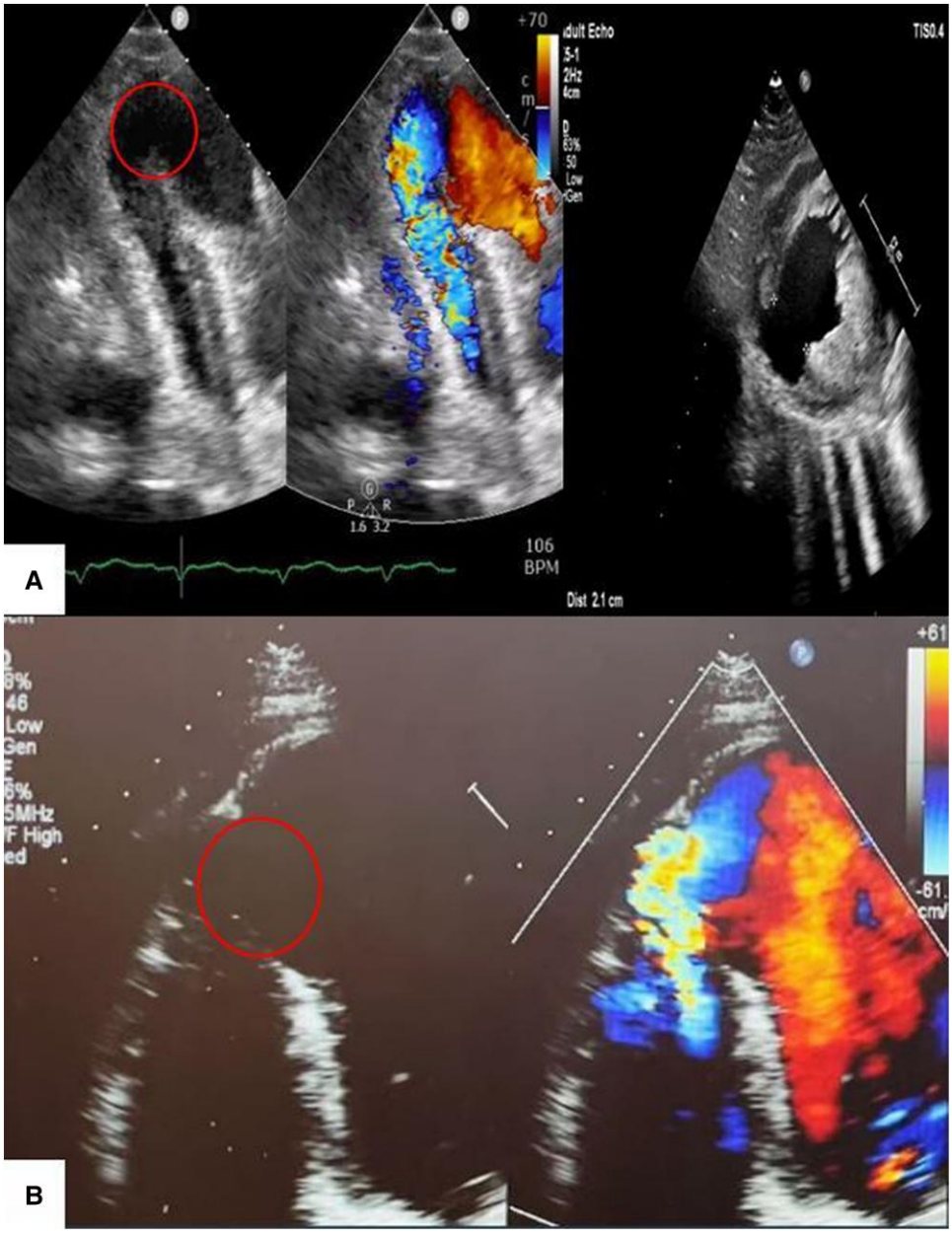


Figure 2 Preoperative transthoracic echocardiography. (*A*) Patient 1 apical four-chamber with RV focused and parasternal short-axis views demonstrating apical post-infarction ventricular septal defect (PIVSD) (circled). (*B*) Patient 2 apical four-chamber view demonstrating PIVSD (circled).

sheath was advanced until its dilator is seen kissing the JR4 catheter. At this point, gentle traction on the glidewire is created and another clamp was placed on the wire at the hub of the 12 F sheath to create a tight AV loop. This allowed the 12 F delivery sheath to be gently advanced across the RV MB and PIVSD, as the JR4 catheter is simultaneously pulled out from the FA access. The 12F dilator tip was positioned in the LV apex and fixed by hand, as the 12F sheath is unscrewed from the dilator hub and slowly advanced over the dilator until its tip is in the LV apex. During this process, adequate tension on the wire assembly is needed to keep the wire curved smoothly in the LV apex, as to prevent the sharp edge of the sheath and the dilator tip from inadvertent puncture of the LV posterior and aneurysmal wall (*Figure 3C*). The dilator was removed once the position of the sheath tip is confirmed by TTE, and the glidewire was pulled back towards the arterial sheath and

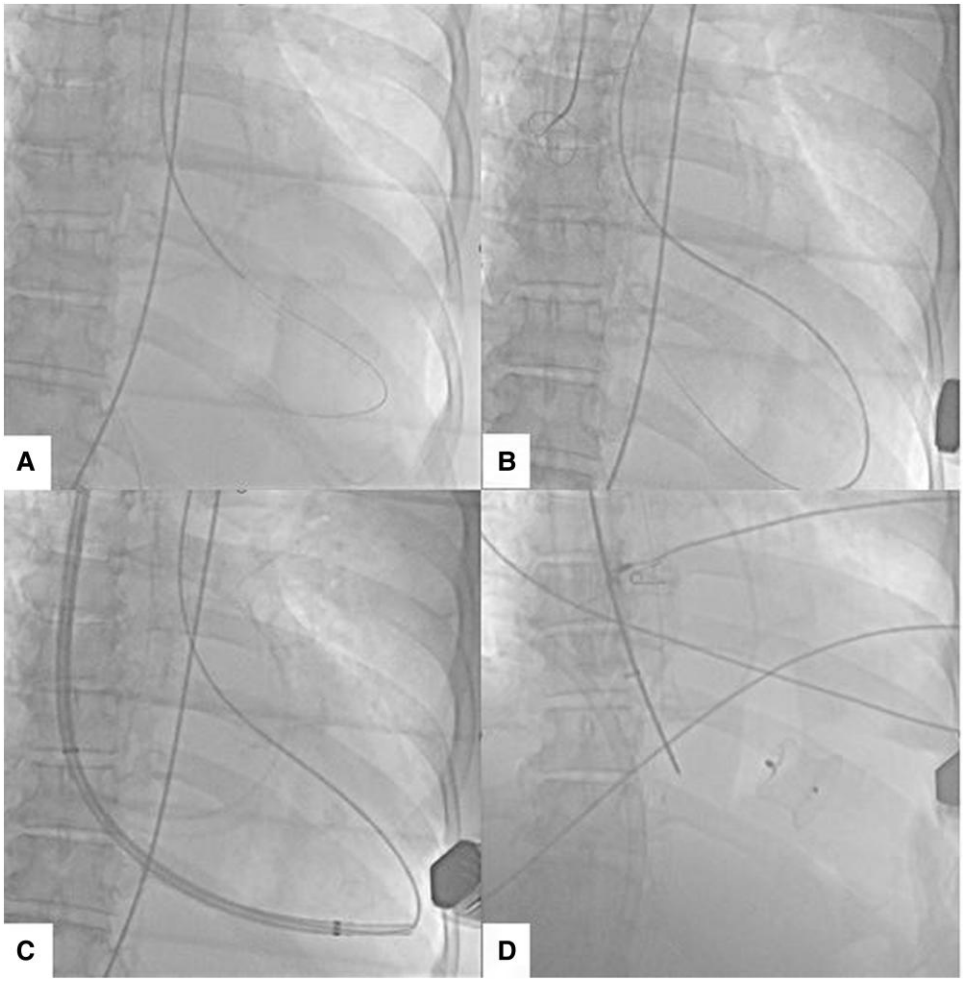


Figure 3 Cine images of the procedural steps—cine images of the procedural steps. (*A*) Direct crossing of the aortic valve, LV, post-infarction ventricular septal defect, and RV. (*B*) Snaring of glide wire, (*C*) positioning of the system in the LV, and (*D*) deployment of the personal digital assistance occluder device.

just inside the 12 F sheath, to allow de-airing and haemostasis before device insertion. The AV loop was maintained to protect the LV apex from inadvertent sheath puncture. A PDA occluder device sized 26 × 24 mm was used, which is almost twice the size of the largest diameter of the MB in the RV at 15 mm measured in the apical four-chamber view (*Figure 4*). Gentle traction in the glidewire was applied as the PDA device is advanced to the sheath tip with a quick push to partially extrude the device forming a small ball shape for a relatively atraumatic sheath tip before the AV loop was broken. Using the apical 4-chamber RV focused view, the sheath and device assembly were pulled slowly, close to the PIVSD, and then, the left wing of the PDA device was opened fully as the assembly is pulled further to abut the RV MB. Once the device is seen and felt abutting the RV MB, the entire body of the device was fully deployed to enmesh with the RV MB and takes a slight apple core shape that represents an adequately positioned device (*Figure 3D*). Colour doppler interrogation immediately showed a marked decrease in the left to right shunt (*Figure 5*). The device was temporarily left attached to observe possible complications, for safety, and prevention of inadvertent embolization. Heparin drip was adjusted to maintain an activated clotting time of 300–350 s to minimize thrombus formation and was removed 24 h post procedure at the same time the device was detached.

Outcomes and follow-up

Immediately post procedures, both cases had sudden improvement of systolic blood pressures and cardiac output with lowered pulmonary artery pressures. The first case was weaned off from vasopressors. TTEs showed small residual shunts, improved RV function, and resolution of TR. However, the first case passed away, 4 days post procedure due

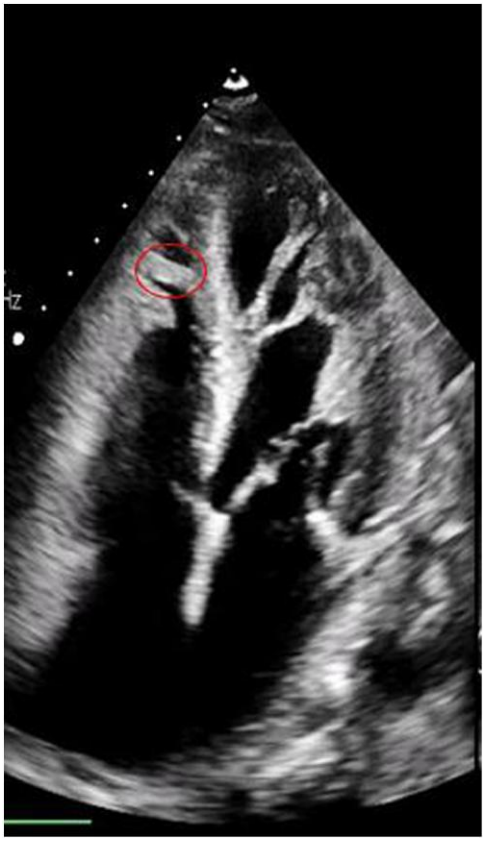


Figure 4 Transthoracic echocardiography—apical four-chamber view demonstrating the RV MB (circled).

to intractable monomorphic ventricular tachycardia from infarction and septic shock secondary to hospital-acquired pneumonia. The PDA occluder was seen intact at its position even after cardiopulmonary resuscitation. The second case was discharged and improved. He was maintained on dual-antiplatelet therapy for a year and de-escalated to clopidogrel thereafter. On 1 year follow-up, he has good functional capacity with intact PDA occluder and small residual shunt flow (*Figure 6A*). Up to the 4th year of follow-up, he has the same findings but with mild LV dysfunction secondary to neo LV-RV aneurysm (*Figure 6B*).

Discussion

To our knowledge, this is the first transcatheter treatment report of apical PIVSD by direct externalization and enmeshment to the right ventricular MB using an occluder device. A technique was coined from the innovator of the technique and one of the authors.

The RV MB is present in majority of people and is the most apical muscular trabecula that bridges the ventricular cavity walls and extends from the interventricular septum to the base of the anterior papillary muscle of the TV. Based on its morphology, direct location to the PIVSD,[8] and the absence of apical rims used in traditional septal anchoring, the idea of enmeshing an occluder device within this cardiac structure is formed (*Figure 7*). The size of a PDA occluder device, 1.5–2× the widest diameter of the RV MB, allowed a secure fit and compression of the muscle fibres of the MB, together with the large left wing of the device which prevented most of the left to right shunt. During enmeshment and device deployment, concerns of oversizing and damage to the MB[9] leading to possible Purkinje-mediated arrhythmias such as short-coupled premature ventricular contractions, ventricular tachycardia with left bundle branch block in left superior axis morphology, and idiopathic ventricular fibrillation[10] were anticipated but did not occur. Similar to a case report of a hybrid approach in apical PISVD that involved sandwiching the septum, RV MB, and RV apex, no incidence of arrythmia was reported.[11]

The major advantage of the technique is its independence from the friable septal landing zones, defects that lack apical rims, and its capacity to address a large and complex PIVSD as long as the shunt flows caudal to the RV MB. Imaging guidance with TTE was preferred because of the anterior location of the RV apex and RV MB. In contrast to other reports of hybrid approach and TCT, cardiac computed tomography scan or magnetic resonance imaging for preoperative planning[12] and the traditional transoesophageal echocardiogram with general anaesthesia for procedural guidance[13,14] were utilized. The potential of conscious sedation to avoid further cardiovascular collapse in a cooperative patient during this procedure is yet to be determined.

The possible drawback in the long term is the expansion of a preexisting LV aneurysm commonly associated with PIVSD and the creation of a neo LV–RV aneurysm that can potentially cause heart failure, thromboembolism, and ventricular arrhythmias which is probably the same mechanism seen in large LV aneurysms.[15] The immediate improvement in both patients and the other with a good long-term outcome highlights its value as a possible bridge or definitive treatment in a subset of patients with apical PIVSD.

Conclusions

In large, complex, apical PIVSD with no apical rims, the technique allows for exclusion of the defect and vestigialization of the right ventricular apex. An immediate and dramatic haemodynamic improvement can be achieved.

Lead author biography

Dr. Bryan Rene F. Toledano graduated from the University of Santo Tomas, where he obtained his degree in Medicine. He completed his Internal Medicine residency at the same institution and went on to pursue a fellowship in Adult Cardiology at the Philippine Heart Center. Additionally, he served as an Interventional Clinical Research fellow at The Medical City Pasig, Philippines. Dr. Toledano's research focuses on studying rare and unique cardiovascular conditions, with a particular emphasis on developing treatment approaches that address the challenges encountered in developing

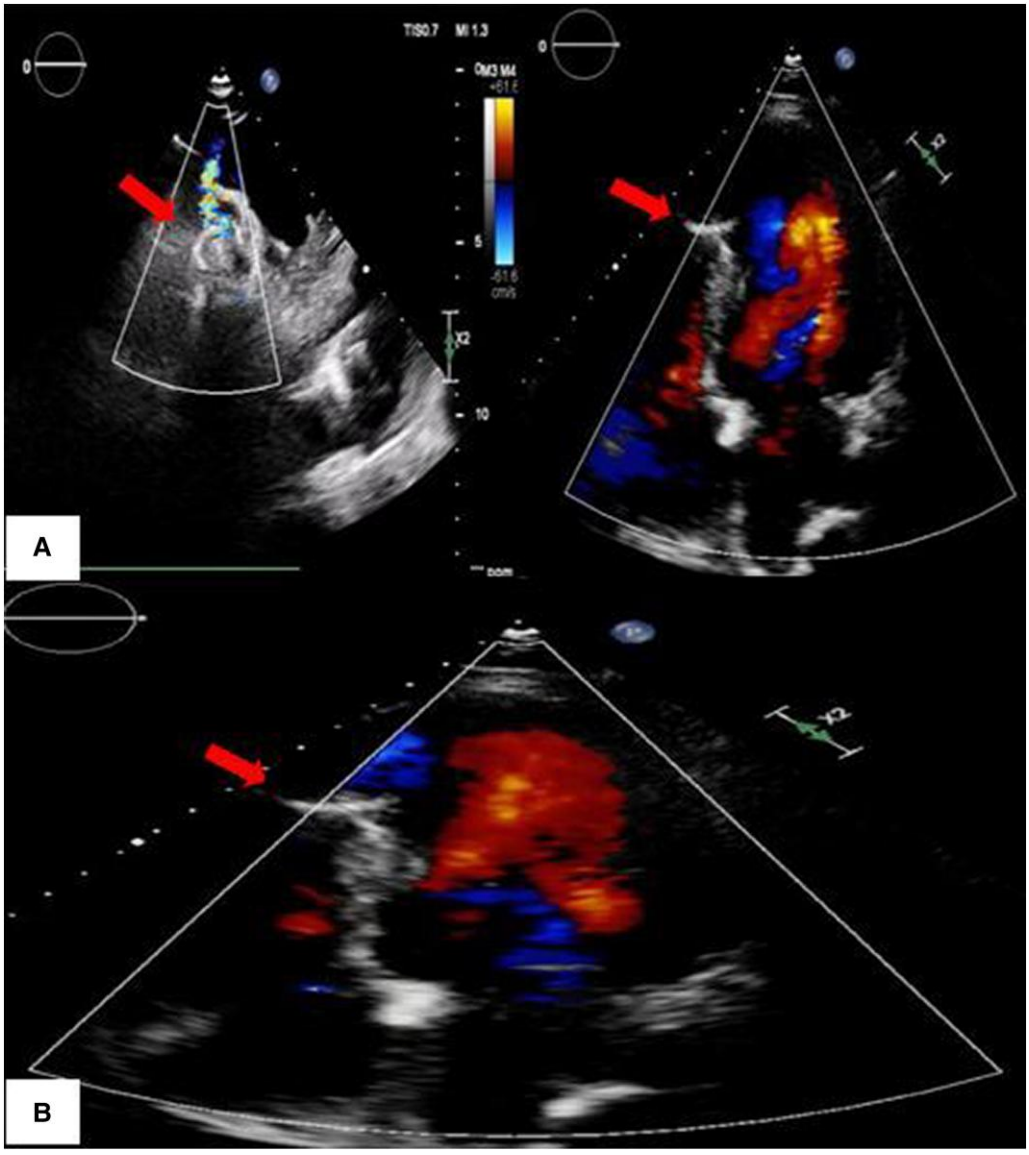


Figure 5 Immediate post-procedure, transthoracic echocardiography. (*A*) Patient 1, (*B*) Patient 2, four-chamber view demonstrating small residual shunt and patent ductus arteriosus occluder device positioned in the RV moderator band (arrow).

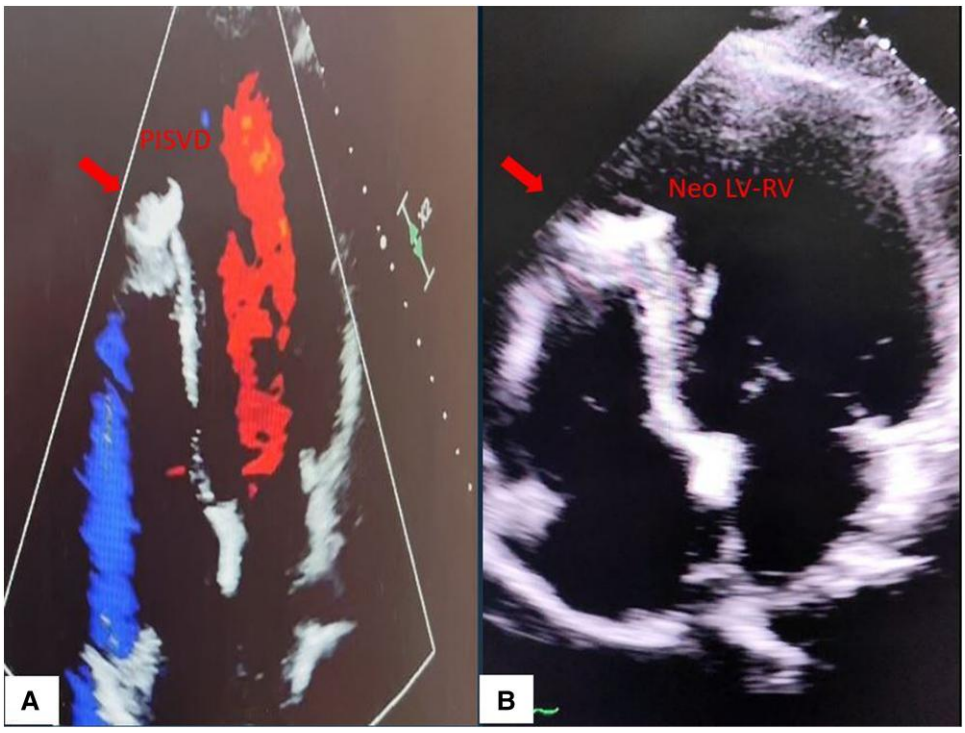


Figure 6 Follow-up post-procedure. (*A*) Patient 2 after 1 year, four-chamber view demonstrating small residual shunt and intact patent ductus arteriosus (PDA) occluder device (arrow). (*B*) Four-chamber view demonstrating intact PDA occluder device (arrow) and presence of neo LV–RV aneurysm (neo LV–RV).

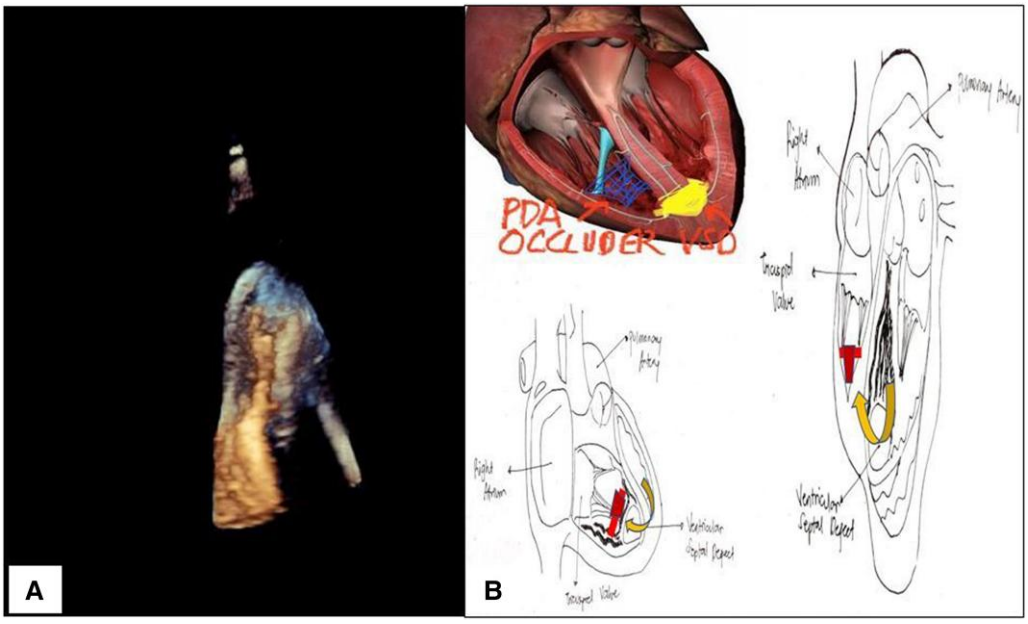


Figure 7 Procedural technique. (*A*) 3D-rendered image and (*B*) illustration demonstrating the patent ductus arteriosus device enmeshed with RV moderator band.

countries. Recognizing the need for specialized skills, he has plans to pursue further training in structural and peripheral vascular interventions.

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Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient/guardian in line with COPE guidelines.

Conflict of interest: none declared.

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Data availability

The data underlying this article are available in the article.

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