Percutaneous closure of small subpulmonic ventricular septal defect with an ADO I PDA occluder in a child

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ABSTRACT

Ventricular septal defects (VSDs) comprise the most common congenital heart defect at birth. The chances of spontaneous closure of VSD depend on the size and location of the defect. Subpulmonic location is an unlikely site for the VSD to close spontaneously and known to have complications such as aortic valve prolapse and regurgitation. Percutaneous closure has become the preferred strategy for small–moderate-sized VSDs located in muscular, perimembranous areas. Subpulmonic location poses concerns due to the close proximity to the aortic valve. Herein, we present a case of percutaneous device closure of a subpulmonic VSD using ADO I occluder device.

Keywords: Acyanotic congenital heart disease, ADO I, percutaneous closure, subpulmonic ventricular septal defect

INTRODUCTION

Ventricular septal defects (VSDs) are the most common congenital heart disease seen at birth. Smaller VSDs, especially perimembranous and muscular types, tend to close spontaneously. However, even they are prone to cause certain complications such as aortic valve prolapse and regurgitation, infective endocarditis, and double-chambered right ventricle. Subpulmonic VSDs because of their proximity to the right aortic cusp have increased chances of aortic regurgitation, so their closure at early stages is advised.[1,2] Percutaneous closure has been well developed in muscular and perimembranous VSDs, however, its use in subpulmonic VSDs has not been well defined. Here, we have used a percutaneous technique for the closure of a subpulmonic VSD with Amplatzer Duct Occluder I (ADO I) Patent Ductus Arteriosus (PDA) device after failure with ADO II and muscular device.

CASE REPORT

A 4-year-old girl was referred for further cardiac evaluation as she was found to have a pansystolic murmur on cardiac examination by her general pediatrician. Her family did not give any history of recurrent respiratory illness or cyanosis, although she had a poor weight gain. She did not have any dysmorphism or history of infective endocarditis in the past. She weighed 10 kg and was 90 cm tall. On cardiac examination, she was found to have a mild cardiomegaly with Grade 4/6 pansystolic murmur best in the left lower sternal border. Her electrocardiogram showed right ventricular (RV) dominance, with no strain, q in lateral leads suggested left ventricular (LV) volume overload with chest radiograph showing mild cardiomegaly with increased pulmonary blood flow, as shown in Figure 1.

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Echocardiogram showed a 3.5-mm subpulmonic VSD with left-to-right shunt, mild enlargement of the left heart with no features of pulmonary arterial hypertension, as shown in Figure 2. There was no aortic valve prolapse or aortic regurgitation. Hence, she was planned for an elective percutaneous closure of VSD.

Catheterization and transcatheter device closure of ventricular septal defect

As per our unit's policy, catheterization and the device closure were performed under intravenous sedation. The standard digital palpation was used to establish the right femoral vein (max: 6 Fr) and right femoral artery (max: 6 Fr). An LV angiogram in the left anterior oblique (60°), cranial (20°), and lateral views could not clearly delineate VSD, although RV was seen filling. A right anterior oblique view showed a small subpulmonic VSD measuring 3.5 mm. Hence, we thought to use either 6-mm muscular VSD device or an ADO II 6-6 to close this defect. A 6-Fr pigtail (shaped to "C" by cutting the end to negotiate the VSD location) was placed in LV, and a straight tip Terumo (0.035") (Terumo™) was used to cross the VSD which was parked in the LPA. Through right femoral venous access, this wire was snared out to exteriorize, as shown in Figure 3a. Through the venous end, a 6-mm muscular VSD device was deployed across the defect. A transthoracic echocardiogram showed protrusion of the device into the left ventricular outflow tract (LVOT) causing mild gradient across, although flow across the defect was sealed, as shown in Figure 4. The findings were no different when ADO II (Abbott™) device was deployed across, as shown in Figure 5. Although not routinely recommended, we wondered if ADO I (Abbott™) can help us in this situation as the family was not keen for surgical closure of VSD. Hence, we deployed an ADO I 8/6 (COCOON) across the defect, as shown in Figure 3b,c with transthoracic echo showing no protrusion in LVOT and no residual shunt. Post device release, LV angiogram showed no residual defect, as shown in Figure 3d, no pullback gradient from LV to the aorta, and no aortic regurgitation on aortic root angiogram. The child withstood the procedure well. Hemostasis was achieved with digital compression and was monitored in the intensive care unit for 12 h with subsequent uneventful stay, and she continued to be in sinus rhythm. At 6 months of follow-up, she gained 4.5 kg at a rate more than previously, and echocardiogram showed a stable position of the device with no residual flow, no aortic regurgitation, or right ventricular

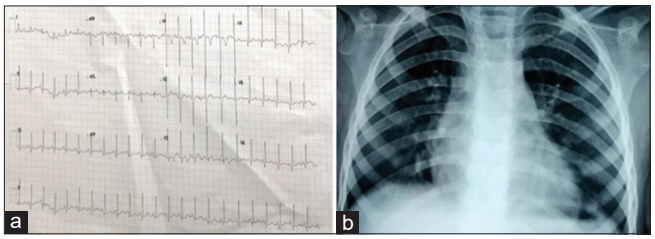

Figure 1: (a) Electrocardiogram showing right ventricular dominance with left ventricular volume overload, (b) chest X-ray showing mild cardiomegaly with increased pulmonary blood flow

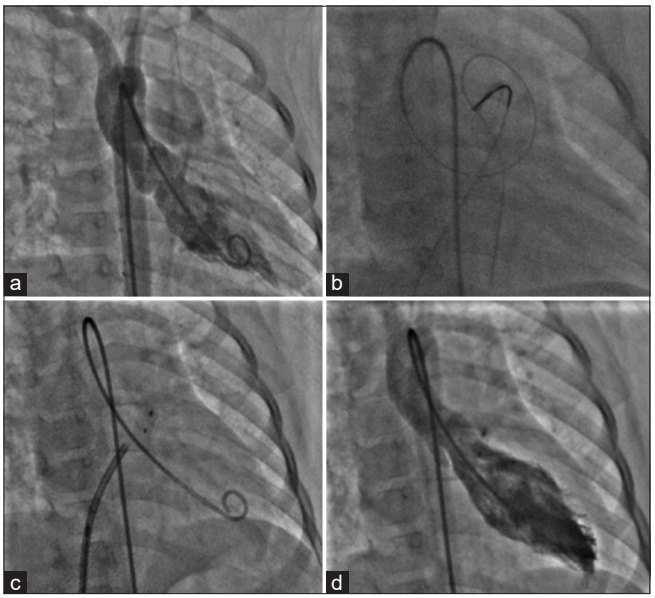

Figure 3: Catheterization images showing (a) left ventricular angiogram in right anterior oblique view contrast filling left ventricular and right ventricular through subpulmonic ventricular septal defect, (b) formation of arteriovenous loop across the ventricular septal defect, (c) deployment of ADO I device across the ventricular septal defect, (d) post device deployment left ventricular angiogram showing proper position of the ventricular septal defect device and no residual flow across the device

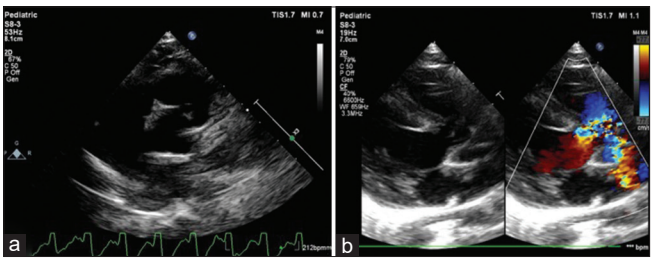

Figure 2 Echocardiogram showing (a) subpulmonic ventricular septal defect of size 3.5 mm, (b) color Doppler showing turbulent jet across the defect

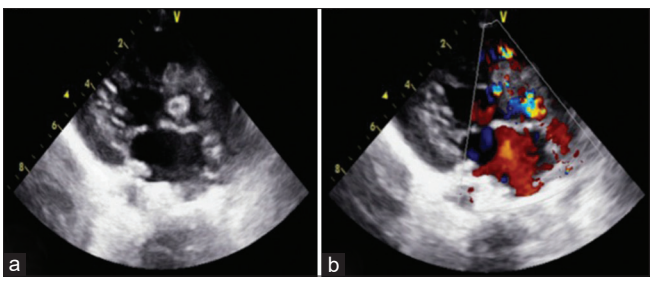

Figure 4: (a) Echocardiogram showing ventricular septal defect muscular device protruding into the left ventricular outflow tract. (b) Color Doppler showing turbulence across the left ventricular outflow tract

outflow tract obstruction, as shown in Figure 6, and she continued to be in sinus rhythm.

DISCUSSION

VSDs are one of the most common forms of congenital heart defects accounting for approximately 20% of defects in isolation. VSDs may occur anywhere within the ventricular septum, and 70%–80% of defects are perimembranous (also called membranous or infracristal) in location, 5%–20% being muscular in nature.[3] Conal septal VSDs account for 5%–8% of all VSDs, which involve a deficiency in the outlet or infundibulum septum, and the nomenclature for these defects is redundant and often confusing, using terms such as supracristal, conoventricular, outlet, doubly committed subarterial, juxta-arterial, and subpulmonary. Percutaneous closure of VSD is the first-line approach for closure of muscular VSDs.[4] Percutaneous closure is also helpful in some perimembranous VSDs with suitable anatomy.[5] A meta-analysis comparing percutaneous closure versus surgical closure in perimembranous VSD shows that percutaneous closure is associated with similar procedural success rate (relative risk [RR]: 1.00, confidence interval [CI]: 0.99–1.00; P = 0.67) without increased risk of significant valvular regurgitation (RR: 0.70, CI: 0.26–1.86; P = 0.47) or heart block (RR: 0.99, CI: 0.46–2.14; P = 0.98), lesser need for blood transfusion (RR: 0.02, CI: 0.00–0.05; P < 0.001), and duration of hospital stay (standard mean difference 22.17 days, CI: 23.12–21.23; P < 0.001) compared with surgical closure.[6] Conal septal VSDs imply an absence of fibrous continuity between the aortic and pulmonary valves and can lead to prolapsing of the right coronary cusp of the aortic valve and significant aortic valve disruption over time. As such, there is a lower threshold for recommending surgical VSD closure at an early age for these types of VSD. However, subpulmonic VSDs are not amenable to transcatheter closure since there is no supporting tissue between the margins of the defect and the atrioventricular valve tissue. In a case series by Mingbaio *et al.*, 49 patients underwent intracristal VSD closure with a success rate of 94%. No deaths, vascular complications, hemolysis, heart block, or pericardial tamponade occurred. Trivial to mild aortic regurgitation is seen in five patients. Follow-up of these patients showed a stable position of the device in all patients.[7] In another series consisting of 56 patients, 48 patients underwent procedure successfully, two patients had aortic regurgitation of which one required surgery and one patient had residual shunt which required a second occluder. Complete heart block did not occur in any of the patients, two patients had incomplete left bundle branch block, which disappeared after 3 months to 1 year of follow-up, and one had complete right bundle branch block, which remained after 3 years of follow-up.[8]

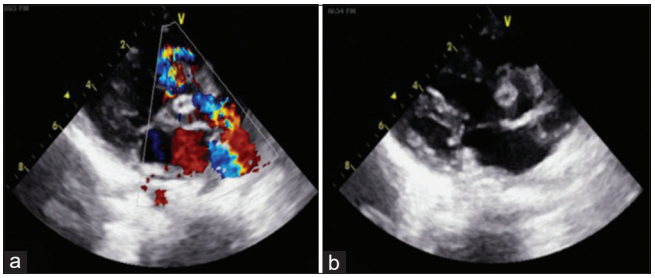

Figure 5: (a) Echocardiogram showing ADO II device across protruding into the left ventricular outflow tract, (b) color Doppler showing turbulence across the left ventricular outflow tract because of protrusion of device into the left ventricular outflow tract

Closure of subpulmonic defects would be careful that the rim of the device should not impinge on the aortic cusps. In our case, the use of muscular and ADO II caused protrusion into the LVOT causing gradients across the LVOT. Hence, we proceeded with the ADO I device which gave us a good position without any gradient across LVOT or aortic regurgitation. Although the length of the devices is more or less the same with different devices, the orientation of the LVOT in relation with VSD varies(in three-dimensional space), making the ADO 1

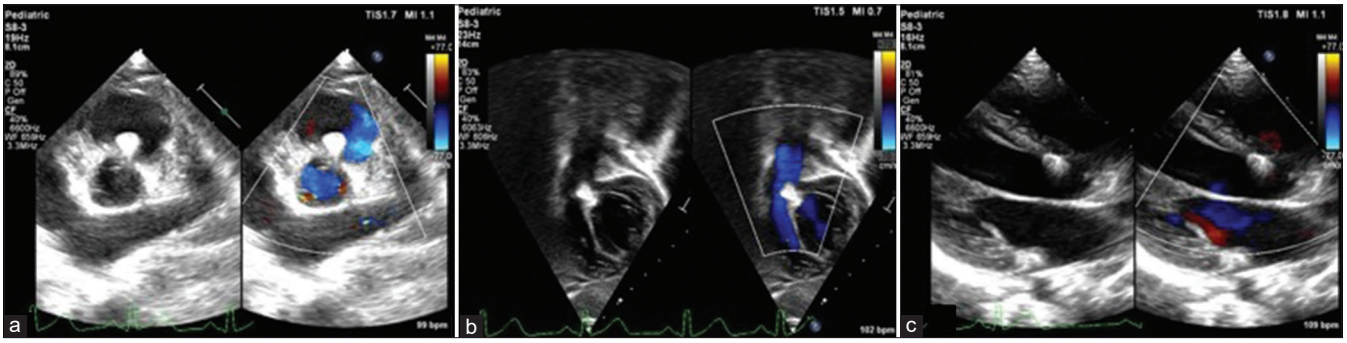

Figure 6: Echocardiogram showing (a) parasternal long axis with right ventricular outflow tract view showing device across the ventricular septal defect with no flow across, (b) apical five-chamber view showing the right ventricular outflow tract and device with no flow across the device, (c) parasternal long-axis view showing device across the ventricular septal defect with no flow across

to align differently and favorably in the given subject. This is somewhat similar to our experience of PDA closure with ADO I, wherein the orientation/alignment of the device (PA disc) determines the turbulence and flow acceleration in LPA. The use of ADO I should be limited to the cases where the use of ADO II or muscular devices causes LVOT obstruction and only be considered as off-label indications. ADO II or muscular device should be the first choice for subpulmonary VSD closure depending on the anatomy. Intraprocedural echocardiography and LV to aorta pullback (for gradient) of pigtail under fluoroscopy are essential steps to rule out LVOT obstruction.

Learning points

1. Percutaneous closure of subpulmonic VSD, although technically challenging, is possible
2. Proper assessment of aortic valve and LVOT pre and before releasing the device is the most crucial part
3. In a child, the length of the device is equally important to avoid LVOT obstruction post device closure
4. Although not routinely used, ADO I can be a safe and good alternate in selected cases of subpulmonic VSD device closure. However, it is an unproven method and needs to be used with caution.

CONCLUSION

To the best of our knowledge, this is the first report in which subpulmonic VSD closed with ADO 1 device in a child of 10 kg. The assessment of aortic valve and LVOT is important for the interventional cardiologists before proceeding to a catheter laboratory for percutaneous closure of subpulmonic VSD.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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