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Original article

Perventricular device closure of isolated muscular ventricular septal defect in infants: A single centre experience☆

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ABSTRACT

Objectives: To evaluate prospective single centre experience of mid-term safety and efficacy of perventricular device closure of isolated large muscular ventricular septal defect (mVSD) in high-risk infants.

Background: Surgical closures of large mVSD in infants represent a challenge with significant morbidity.

Methods: Between August 2008–2010, perventricular closure was attempted in 24 infants of 6.01 ± 2.37 months age and 4.27 ± 0.56 kg weight under TEE guidance.

Results: The device was successfully deployed in 21/24 infants. Size of mVSD was 8.42 ± 1.46 mm (6.1–12 mm). Mean procedure time was 28.8 ± 11.7 min. The closure rate was 84% immediately and 100% at 6 months. Four patients suffered major complications: 2-died, 1-esophageal perforation, 1-persistent CHB. At 26.23 ± 6.63 months follow-up two patients were symptomatic: 1-required device retrieval, 1-died of severe gastroenteritis.

Conclusion: Perventricular device closure of isolated mVSD appears feasible option at mid-term follow-up and may either substitute or complement the conventional surgical technique in selected cases depending on institutional paediatric cardiac surgery performance.

1. Introduction

Muscular ventricular septal defects (VSDs) account for approximately 10–15% of all VSDs.[1] Conventionally, children with persistent small mVSD are subjected to percutaneous device closure at preschool age and the infants with large mVSD are managed surgically.[2–4] Sometimes, the conventional approach is complicated by the need for

Abbreviations: mVSD, muscular ventricular septal defect; RV, right ventricle; TV, tricuspid valve; LV, left ventricle.

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ventriculotomy[5–7] or palliative pulmonary artery (PA) banding, inability to locate the defect during ventriculotomy and occasionally, by LV dysfunction.[4] The percutaneous closure of mVSD is an alternative technique with encouraging results in children.[8,9] However, its application in infants is limited due to relatively large size of the device-delivery-apparatus, which may cause hemodynamic instability and rhythm disturbances.[10] This underscores the need for a hybrid approach where the access obtained by sternotomy or epigastric incision allows deployment of closure device into the beating-heart under echocardiographic and/or fluoroscopic guidance. The short-term results of perventricular VSD device closure as a sole procedure for isolated VSDs and as an additional procedure for VSDs in complex congenital heart disease (CHD) are promising in several, mixed, small cohorts of patients.[11–18] We aim to report our experience with perventricular device closure of isolated large mVSD in a relatively large cohort of high-risk infants.

2. Materials and methods

2.1. Inclusion criteria

Our patient population consists of infants with symptomatic large mVSD and left to right shunt. The suitability criteria for perventricular closure of mVSD included: (1) symptoms of persistent heart failure including failure to thrive despite optimal medical management, (2) body weight less than 5 kg, which is considered inappropriately low for standard percutaneous closure, (3) isolated large muscular VSD with left to right shunt and significant pulmonary arterial hypertension (4) presence of at least 5 mm muscular rim separating the defect from semilunar valves and atrioventricular valve tension apparatus and (5) hemodynamically insignificant left to right shunt from additional mVSD(s), when present.

The patients with the following criteria were considered unsuitable for the procedure: (1) additional CHD except small patent ductus arteriosus (PDA) and atrial septal defect (ASD), (2) any major debilitating illness making the infant unfit for cardiac surgery.

The hospital ethics committee approved the study protocol. A written consent was obtained from both the parents who were informed and explained about the nature, the advantages and the disadvantages of the procedure in comparison with open surgical repair using cardiopulmonary bypass (CPB). The clinical history and examination, investigational reports, case notes, echocardiography reports, procedure-related notes and post-procedural findings were recorded in the departmental database.

2.2. Anaesthesia and monitoring

Twenty-four infants who met the suitability criteria were brought to the operating room (#1–18) or cardiac catheterization laboratory (#19–24) in a post-absorptive state. Stand-by CPB machine was available for every procedure. The procedure was performed under general anaesthesia after adequate pre-oxygenation. The infants were orotracheally ventilated with pressure-controlled, volume-guaranteed artificial ventilation. ECG, pulse oximetry, invasive arterial pressure, central venous pressure, temperature and urine output were continuously monitored throughout the procedure.

2.3. Perventricular access

The heart was approached through median sternotomy ($n = 23$) or minimally invasive subxiphoid incision ($n = 3$). Under continuous transesophageal echocardiographic (TEE) guidance, the optimal site for right ventricular (RV) puncture was chosen by indenting the RV free wall with forceps-held cotton gauze. The puncture site was considered optimal, if it was away from the papillary muscle and remained at an adequate distance from the septum so as to allow perpendicular access to the defect (Figs. 1 and 2). After securing purse-string suture with 6-0 polypropylene around the optimal puncture site, a 20G needle, Jelco (Smith Medical, UK), was introduced into the RV, perpendicular to the mVSD. A 0.025-inch straight-tipped guide wire, Terumo (Terumo Corporation: Tokyo, Japan), was introduced through the needle and maneuvered across the defect into the LV (Fig. 3). Unfractionated heparin (50 units/kg bolus) was administered intravenously. The needle was exchanged with a short 5F sheath (Terumo Corporation: Tokyo, Japan), over the wire. To avoid injury to RV free wall and inadvertent perforation during subsequent manipulation, the tip of the sheath was parked just across the RV puncture and the partially withdrawn dilator was left inside it, 5–10 mm beyond its tip. Customized delivery system including puncture needle may be used as well.[19] The 5F sheath was exchanged with a short 8 or 10F sheath (Cordis Corporation; Miami, FL, USA) over 0.038-inch angled guide wire (Cordis Corporation; Miami, FL, USA). The sheath was positioned across the defect with its tip in the LV cavity (Fig. 4).

2.4. Selection of closure device

The defect size was measured in 4-chamber and long axis TEE views. The larger of the two values was considered for device

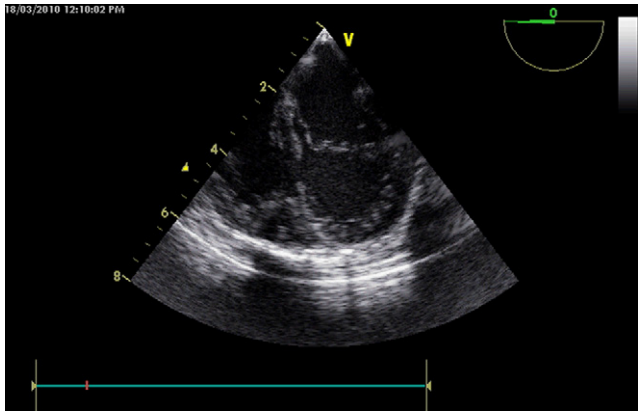

Fig. 1 – Transesophageal echocardiographic four chamber view of large mid-mVSD.

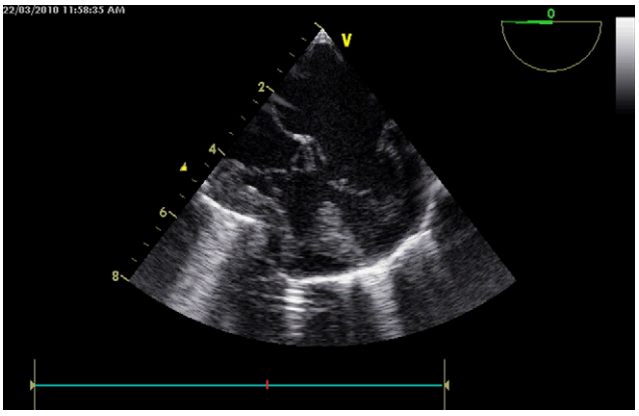

Fig. 2 – RV free wall indentation with a forceps tip, away from the anterior papillary muscle of the TV.

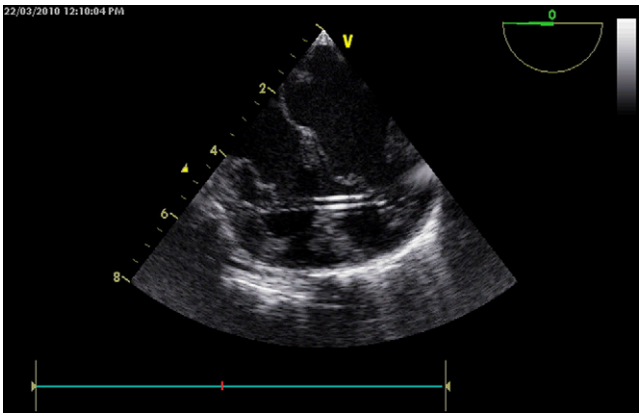

Fig. 4 – An introducer sheath is passed over a dilator and positioned into the LV cavity.

selection. The muscular device occluder (Cardi-O-Fix, Starway Medi Tech, Inc., Beijing, China), 1–2 mm larger than the VSD size and rim extending 7 mm from the central disk on either side was selected. The Starway Cardi-O-Fix VSD Occluder is a self-expandable, double disc implantable device made up of Nitinol wire mesh. The two discs are linked together by a short connecting waist corresponding to the size of the VSD. In order to increase its closing ability, the discs and the waist are filled with polyester fabric. The polyester fabric is securely sewn to each disc by surgical sutures.

2.5. *Deployment of device*

The device, pre-soaked in blood, was screwed on to the cable and loaded inside a 6–8F loader. The device was advanced into the short sheath and the LV disk opened in mid-LV by gently retracting the sheath over the cable (Fig. 5). The device was deployed across the defect under continuous TEE guidance conventionally (Fig. 6). TEE imaging in multiple planes was undertaken to confirm appropriate device placement and assess residual shunt, if any. In addition, any new valvular obstruction or regurgitation was also looked for.

2.6. *Post-procedural care and follow-up*

Post-procedurally; aspirin (5 mg/kg) was given for 12 months. Oral diuretic (furosemide, 1 mg/kg) was also prescribed for the first 3–6 months. Relevant clinical and echocardiographic details (including residual shunt and pulmonary artery (PA) pressure estimated by tricuspid regurgitation jet velocity) were obtained during follow-up visits scheduled at 1, 3, 6, 12, 18 and 24 months, post-discharge. Based on the jet width detected on transthoracic color Doppler study, the residual shunt was classified as trivial (<1 mm), small (1–2 mm), moderate (2–4 mm), or large (>4 mm).

3. Results

Table 1 summarizes the results of the study.

3.1. *Demographics*

From August 2008 to August 2010, 24 symptomatic infants with mVSD detected on TTE, were found suitable for perventricular device closure at our institute. All infants had either

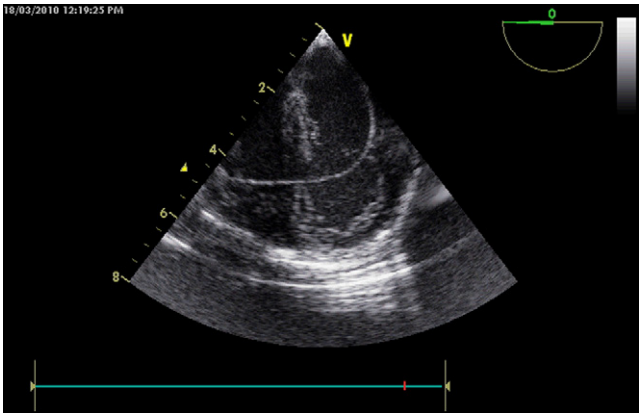

Fig. 3 – A guide wire manipulated into the LV.

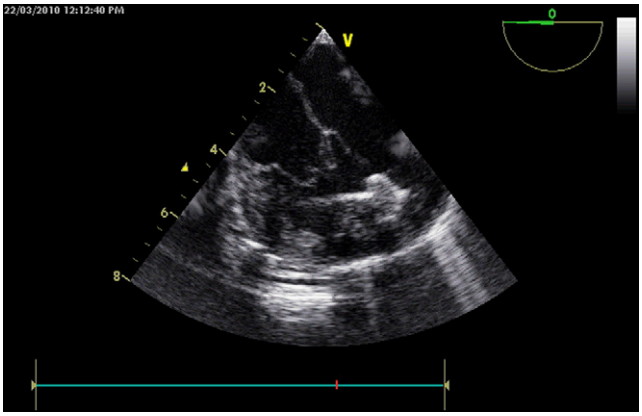

Fig. 5 – The LV disk of the device has been opened.

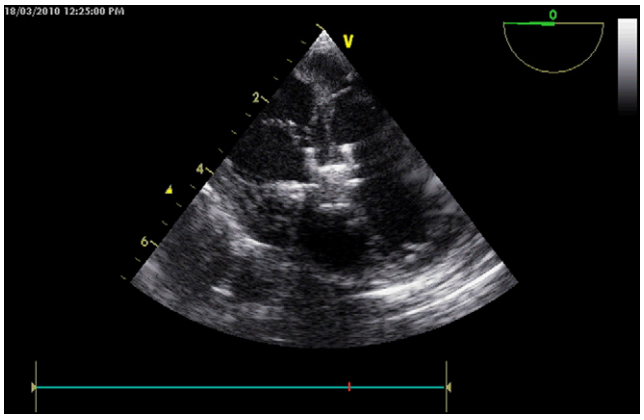

Fig. 6 – The device deployed across the defect, not unscrewed from cable.

persistent failure to thrive or clinical signs of cardiac failure. Their mean age was 6.01 ± 2.37 mon (range 2–12 mon) and the mean weight was 4.27 ± 0.56 kg (range 2.7–4.9 kg). All but 3 infants were malnourished. Eighteen infants had grade III–IV protein energy malnutrition (PEM) and 3 infants had grade II PEM.[20]

3.2. *Pre-procedural echocardiography*

The location of the defect was mid-muscular in 12, distal septal in 5 and anterior muscular septal in 5 patients. Two infants had posterior septal defect. The mean echocardiographic size of the mVSD was 8.42 ± 1.46 mm (range 6.1–12 mm) in its longest dimension. Fig. 7 shows the scattered diagram of partial linear relationship between VSD size and defect to device ratio. Two patients had additional small muscular VSDs and five had PDA. One patient had bicuspid aortic valve without significant aortic stenosis. Two other patients had small ostium secundum ASD. One patient had undergone successful balloon coarctoplasty, 1 week prior to the procedure. Prior to device closure, the mean estimated PA pressure was 72.05 ± 10.20 mmHg. All infants had normal LV systolic function at presentation. In 5 patients, surgical ligation of associated PDA was accomplished successfully during perventricular VSD closure.

3.3. *Acute procedural and echocardiographic outcomes*

Successful perventricular device closure was accomplished in 21 out of 24 infants with intention to treat. Three infants, among the first 18 who were intervened in the operating room, required surgical closure of the mVSD. The last 6 infants underwent successful perventricular device closure in the catheterization laboratory. Excluding the surgical preparation time, the mean procedure time was 28.8 ± 11.7 min. The mean size of successfully deployed mVSD occluder was 11.5 ± 1.93 mm (range 8–16 mm). Fig. 7 shows the scattered diagram of partial linear relationship between VSD size and defect to device ratio. Since the sheaths were gradually upgraded from smaller to larger size instead of direct use of large-size sheath, we did not notice any sustained arrhythmia or mechanical LV compression. Immediately after the mVSD device closure, 11 infants had no residual shunt; trivial (intradevice foaming) residual shunt was detected in the remaining 10. Immediately post-device closure, estimated PA pressure dropped to mean 56 ± 8.4 mmHg.

3.4. *Surgical conversion*

Three infants failed perventricular device closure of mVSD and required immediate surgical conversion. One patient with mid-mVSD extending into the posterior septum developed small LV perforation during the introduction of the sheath over the Terumo wire. In another infant with a similar location of mVSD, the defect could not be crossed with the guide wire even after repeated attempts through different RV puncture sites. The third infant had a large residual shunt

Table 1 – Clinical, echocardiography and procedural details of study patients.

Patient demographics		Echocardiography data		Procedure analysis	
Patients	24	VSD location	Mid-muscular 12, distal septum 05, anterior muscular 05, posterior muscular 02	VSD occluder size Device/defect ratio	11.5 ± 1.93 (8–16 mm) 1.35 ± 0.14
Sex	Male 10 Female 14	VSD Size (mm)	8.42 ± 1.46 (6.1–12)	Procedure success	Device deployment 21 (complete closure 11, trivial shunt 10); surgical closure 03
Age (months)	6.01 ± 2.37 (2–12)	Preprocedure PA Pressure (mmHg)	72.05 ± 10.2	Post-procedure PA pressure (mmHg)	56 ± 8.4
Weight (kg)	4.27 ± 0.56 (2.7–4.9)	Additional defect	Small mVSDs 02, PDA 05, bicuspid AV 01, small OS ASD 02	Procedure time (minutes)	28.8 ± 11
PEM status	Grade III–IV: 18 Grade II: 3	Additional procedure	Balloon coarctoplasty 01, PDA ligation 05	Median ICCU stay (days)	3

Abbreviations: mVSD – muscular ventricular septal defect; PA – pulmonary artery; PDA – patent ductus arteriosus; OS ASD – ostium secundum atrial septal defect; AV – aortic valve; PEM – protein energy malnutrition.

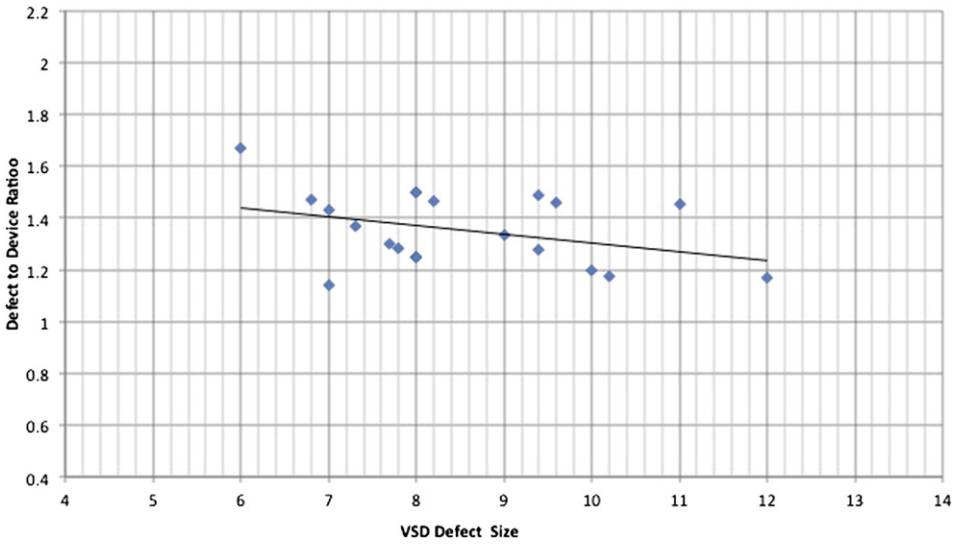

Fig. 7 – Scattered diagram of relationship between VSD size and defect to device size ratio.

after device deployment across the defect, before its release. The defect appeared malaligned and was not amenable to closure with conventionally designed device. Surgical patch closure was undertaken successfully in all of them.

3.5. *In-hospital course*

The median duration of the stay in intensive care unit was 3 days and the median duration of artificial ventilation was 16 h. All except two infants were discharged on the 5th post-operative day. None had LV systolic dysfunction at discharge. Two patients died during in-hospital stay from suspected ventricular tachyarrhythmia and hospital-acquired, fulminant bronchopneumonia leading to septicemia and multi-organ failure, respectively.

3.6. *Complications*

3.6.1. *Deaths*

Three patients died including two infants who died in the hospital, post-procedurally. One infant, who died suddenly during breast-feeding on 3rd post-procedural day, had documented hypokalemia (serum K^+: 2.8 mEq/L), which could have resulted in torsade de pointes and ventricular fibrillation. Alternatively, aspiration during breast-feeding could have been the cause. The defect size was 8.2 mm and 10 mm device was deployed. Echocardiography done during and after resuscitation showed the device lying in-situ and thereby, ruled out device-induced obstruction/leak. A neonate weighing 2.7 kg suffered from hospital-acquired fulminant bronchopneumonia on 3rd post-procedural day. Blood culture was positive for *Klebsiella*. There was no echocardiographic evidence of vegetation on the device. Anti-*Klebsiella* antibiotics were started; device retrieval was not contemplated. The baby eventually expired on 18th day due to septicemia and multi-organ failure. Six months after the device closure, an infant died of acute gastroenteritis complicated by severe dehydration leading to renal dysfunction.

3.6.2. *Complete heart block and LVOT obstruction*

Two patients developed complete heart block (CHB) following device deployment. As a protocol, epicardial wires were left in-situ for up to 1 week; post-procedurally, in all patients. One patient with late (5th day) onset CHB regained sinus rhythm, 48 h later, on oral prednisolone (2 mg/kg/d), which was discontinued after 1 week. There was no evidence of any conduction disturbance on Holter, 6 months later. Another infant developed early onset (12 h after device deployment) persistent CHB unresponsive to oral steroid therapy and necessitating permanent pacemaker implantation. In a 12 mm mid-muscular defect, a 14 mm device with 7 mm rim all around it was deployed. Since the heart block developed post-procedurally, we did not retrieve the device. On echocardiography undertaken at 18 months follow-up, she was found to have developed LV outflow tract obstruction (peak aortic velocity 4.1 m/sec). Inappropriately large-sized device protruding into the outflow tract was surgically removed followed by patch closure of the defect.

3.6.3. *Esophageal tear*

Esophageal tear resulted from the use of pediatric (instead of neonatal) transesophageal probe in an infant weighing more than 4 kg. The baby recovered completely with conservative management.

3.7. *Follow-up*

All 22 infants, who went home, were asymptomatic at 1 and 3 months of follow-up. At 6 months, all drugs except aspirin were stopped. On repeat echocardiography at 6 months, 8 out of 10 infants with trivial residual shunt had no residual shunt. Trivial shunt persisted in the remaining 2 infants. Six months after the procedure, 1 infant died of severe gastroenteritis.

One infant who developed intra-procedural heart block and required permanent pacemaker prior to discharge presented with device-related LV outflow tract obstruction at 18 months. The child underwent surgical device removal followed by patch closure of the defect. After mean 26.23 ± 6.63 mon, all but two infants remained asymptomatic with adequate weight gain and near complete regression of pulmonary hypertension. Two infants required hospitalization for non-cardiac illness.

4. Discussion

Conventional surgical closure of VSD in small, low weight infants is challenging. Kitagawa et al[4] reported 10% rate of reoperation for residual defects after surgical closure of mVSD in 22 infants using right atriotomy with or without left ventriculotomy approach. Wollenek et al reported similar reoperation rate after left ventriculotomy approach with mortality rate as high as 16%.[21] They concluded that pulmonary artery banding is a better option for infants with significant mVSD. In another series of 130 patients (61 infants) with isolated mVSD, the post-surgical repair mortality was 7.7%.[2] Stellin et al[7] described surgical closure of apical VSD in four infants through right apical ventriculotomy as a better approach. However, it still required CPB and circulatory arrest. On the other hand, retrospective analysis of surgical closure of isolated VSDs by Scully et al has had excellent results with extremely low incidence of adverse events.[22]

Initial intraoperative device closure procedures that required both CPB and circulatory arrest, had high mortality and failure rates ranging from 14 to 25%.[23,24] Since the first perventricular device closure of mVSD in animals, and later in humans by Amin and colleagues,[25–28] nearly 35 cases of perventricular device closure of isolated mVSD in infants with short-term follow-up have been reported in several small series[11–18,29] (Table 2). In a retrospective study, Bacha et al[11] first reported, the safety and efficacy of perventricular device closure in 6 children (20 days–3 years; 3–20 kg). This study was instrumental in establishing the feasibility of hybrid procedure in infants and small children. Crossland et al confirmed that perventricular device closure of isolated large mVSD is safe and effective in 8 infants (median age – 14 (2–41) weeks; median weight – 4 (3–6.6) kg) at short-term follow-up.[13] Gan et al reported similar results in 8 patients (mean age 2.5 ± 3.6 years, mean weight of 10.9 ± 7.6 kg) with post-procedural hospital stay of 7.9 ± 2.2 days.[14] In their experience on mVSD device closure, Karim et al undertook successful perventricular device closure in 8 out of 20 infants.[15] Recently, Bendaly et al confirmed the feasibility of perventricular device closure in 6 infants (median age 4.6 (1.7–24.6) months; median weight 5.7 (4–13) kg) and reaffirmed its safety at 2 years post-intervention.[16]

We have presented a single center prospective experience on the feasibility and mid-term outcome of perventricular device closure of isolated, large, mid-muscular VSD in a relatively large cohort of high-risk, malnourished infants. Our population is comparatively younger (mean age: 6.01 ± 2.37 mon), with lower body weight (mean weight: 4.27 ± 0.56 kg) and sicker (PEM grade II–IV in 80%) than that in the previously published reports. In an analysis of more than 14,000 neonates undergoing surgery for CHD, low body weight remained a strong predictor of higher mortality.[30] The multivariate analysis confirmed that low body weight, longer CPB and aortic cross-clamp times as well as longer duration of circulatory arrest were high-risk predictors of in-hospital mortality. Lower body weight was associated with higher hospital mortality in a meta-analysis of seven series.[31] Risk correlation of lower weight has similar implication during percutaneous intervention, as well. Holzer et al concluded that low body weight, at the time of procedure, significantly correlates with an increased risk of procedure as well as device-related complications ($p - 0.007$). The median weight of patients who had unsuccessful percutaneous procedures was 6.5 kg as against 9.4 kg in the patients who had a successful procedure.[10] By virtue of their age, weight, primary cardiac illness and nutritional status, our patients were among the substantially high-risk candidates for surgical as well as perventricular device closure.

Perventricular device closure was deemed successful when the occluder could be placed in the correct position, which was possible in 21 of 24 patients (87.5%). None of our infants required PA banding. Three patients required immediate surgical intervention due to inability to advance the sheath across the posterior muscular defect in one, LV perforation in the other and presence of a large residual shunt in the third that had a malaligned muscular defect. After this continuous TEE-guided-experience in the operating room, it was considered prudent to have an additional imaging modality like fluoroscopy to improve the efficacy and safety of perventricular device closure of VSD. We also believe that certain anatomical variants of muscular defects like high posterior mVSD, distal apical mVSD and malaligned mVSD may not be currently considered suitable for perventricular device closure and necessitate the development of some specifically designed hardware. Having been unable to cross high posterior muscular VSD with conventional RV access, Bacha and colleagues[11] successfully closed it by switching over to peratrial approach in one of their patients. In a multicenter retrospective analysis reported by Ina Michel-Behnke et al, device placement was successful in 23 out of 26 (88.5%) procedures including perventricular approach in 20. Device removal was necessary in three due to arrhythmia, malpositioning or additional defects.[17]

4.1. *Complications*

We observed certain avoidable complications like perioperative CHB, late LVOTO, esophageal tear and infection. Some of them were due to very large defect requiring bulky device and defects close to or extending into the inlet, posterior or apical septum, which we may consider as not suitable for device closure, as yet. Also we emphasize vigilant perioperative care for the prevention of infection and arrhythmia and judicious use of transesophageal probes by skilled and experienced imaging expert to avoid potentially fatal esophageal tear.

After successful device closure, two infants died post-procedurally, in-hospital, due to suspected ventricular arrhythmia in one and septicemia leading to multi-organ failure in the other. Two patients developed CHB after device

Table 2 – Clinical studies involving perventricular device closure of mVSDs.

Study	Patients (CPB required)	Age (mean)	Weight (mean kg)	Procedural difficulties or complications	Additional procedure/ outcome	Follow-up (months)
Bacha et al[11,12]	11 (9)	14 d–3 yrs	3–20	Peratrial approach after failed PV approach: 1 Difficult deployment of RV disc: 1	Mediastinitis: 1	3–23 (12)
Crossland et al[13]	8 (1)	2–41 wks (14)	3–6.6 (4)	Incomplete RV disc opening: 1 (device reposioning on CPB)	Mild residual shunt at device edge	0.5–66 (7.2 wks)
Gan et al[14]	8 (3)	0.5–11 yrs (2.5 ± 3.6)	5.5–27.5 (10.9 ± 7.6)	Incomplete RV disc expansion: 1 (surgical patch closure)	–	6
Karim et al[15]	8 (4)	18 days–4.9 mon	3.2–7.3	Transient EMD: 1 Device migration: 1 Wire perforation: 1	Mediastinitis: 1 (died of sepsis after 10 months)	0.4–6.8 yrs (3.8)
Bendaly et al[16]	6 (1)	1.7–24.6 mon (9.8 ± 9.1)	4–13 (7.2 ± 3.7)	Device embolization: 1 (Surgical patch closure)	LV pseudoaneurysm: 1 (coil embolization)	39.8 ± 25.2
Ina Michel-Behnke et al[17]	17 (2)	0.8–60.1 mon	3.2–16	CPR, rescue CPB: 1 Device repositioning: 2 Device removal: 1 Pericardial effusion: 1	2 died, one due to LV dysfunction and another with complex CHD died of unknown etiology	12 days–47 mon
Present Study	24 (3)	6.01 ± 2.37 mon	4.27 ± 0.56	Patch closure: 3 CHB: 2, one transient, one required pacemaker Fulminant bronchopneumonia: 1 Esophageal perforation: 1 Fatal ventricular tachyarrhythmia: 1	Patient with pacemaker developed significant LVOT obstruction after 18 months. One died of acute gastroenteritis after 6 months	26.2 ± 6.6

Abbreviations: PV – perventricular; mVSD – muscular ventricular septal defect; RV – right ventricle; LV – left ventricle; LVOT – left ventricular outflow tract; EMD – electromechanical dissociation; CPR – cardiopulmonary resuscitation; CPB – cardiopulmonary bypass; CHD – congenital heart disease; CHB – complete heart block.

closure. Early onset CHB was most likely due to oversized-device-induced trauma to the conduction system in an infant with a large defect. Late onset CHB could have been due to edema around the conduction system, which may result from procedural manipulation and device homing. Although the latter may be transient, oral steroid therapy may help it recover. Conduction blocks are well-described complications in transcatheter closure of perimembranous VSD (pmVSD) as well as mVSD particularly in patients with low body weight. Dragos et al has reported CHB requiring permanent pacemaker in 22.2%. Late onset CHB was a significant complication in their population particularly in patients less than 10 kg or those having a higher device:defect ratio.[32] Holzer et al reported arrhythmic events in 20% including conduction blocks (RBBB) in 2.6%.[31] Serraf et al reported 3% incidence of CHB in 130 children after surgical closure of isolated multiple ventricular septal defects.[2]

Though not observed in this series, other procedure-related difficulties or complications associated with perventricular device closure (Table 2) include, device embolization,[16] LV pseudoaneurysm,[17] and unexpanded RV disc protruding into pericardium.[11,29] Incomplete RV disc expansion with screw or disc protruding into the pericardium is the most common another complication reported in literature[11–14] however sometimes can be avoided by selecting duct occluder. As observed in our study, Bacha et al experienced similar difficulty in crossing the posterior defect with conventional RV access, but they closed the defect with peratrial technique.[11] Another potential complication requiring immediate surgical closure is device migration or embolization.[14,17,18] Karim et al had guide wire perforation in one of their patient. There are some uncommon but late complications include LV pseudoaneurysm (possibly due to sheath or dilator-related free wall injury),[17] LVOTO due to disc protrusion etc. Air embolism, hemolysis and tricuspid regurgitation may occur, very rarely.

4.2. *Hybrid intervention*

After the initial experience in the operating theatre on 18 patients, we undertook mVSD device closure in the catheterization laboratory with surgical preparedness in subsequent 6 infants. We observed that combined fluoroscopy and TEE guidance significantly helped guide wire manipulation across the mVSD, sheath advancement and device deployment as compared to TEE guidance, alone. Hence a hybrid cardiac catheterization lab within a surgical theatre would be an ideal setup to perform such technically challenging procedures. A hybrid technique does not require CPB or full sternotomy. Other advantages include avoidance of transection of the moderator band or other RV muscle bundles, immediate confirmation of adequate closure, and avoidance of ventricular incisions. Difficult deployment of RV disk in distal or apical VSD can be dealt with by a simple pledgetted stitch placed on the beating-heart or using the duct occluder until custom-made device is available.[14,15,18]

With advent of hybrid technique the indications for surgery are likely to be redefined in selected patients. It appears that palliative PA banding may not be indicated any more in infants with large muscular VSD. One-stage repair can be offered to neonates with aortic coarctation and muscular VSD. Similarly, large muscular VSD can be closed using a perventricular technique at the time of PA band takedown. In addition to the primary closure of muscular VSDs,[11–13] the perventricular technique appears to be excellent for device closure of perimembranous VSD in the operating room, particularly in small babies, who are high-risk candidates for closure in the catheterization laboratory.[33,34]

Limitation: our study involves prospectively followed cohort of infants subjected to perventricular mVSD device closure. There is no randomized comparison with surgical closure arm. The duration of follow-up is medium term and therefore this study is unable to address incidence of ventricular arrhythmias, ventricular dysfunction, pseudoaneurysm and sudden deaths, which have been reported late after surgical closure.

5. Conclusion

In selected high-risk infants, perventricular device closure of isolated mVSD is effective and may either substitute or complement the conventional surgical closure depending on the performance of institutional pediatric cardiac surgery program. The procedural safety can certainly be improved with more precautions for preventable complications. Until specifically designed hardware is available, very large defect or defects extending into inlet, posterior or apical septum are not suitable for perventricular closure.

Conflicts of interest

All authors have none to declare.

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