Safety and efficacy of percutaneous closure of perimembranous ventricular septal defects in children: Review of the results at Inkosi Albert Luthuli Central Hospital

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ABSTRACT

Background/Hypothesis: Ventricular septal defect (VSD) is the most common congenital cardiac lesion. Surgical closure is the gold standard, but in an isolated perimembranous ventricular septal defect (PMVSD), percutaneous closure is an attractive alternative, particularly in a limited resource setting. Our experience suggests that percutaneous closure of a perimembranous VSD, in the appropriately selected patient, is safe and effective.
Materials and methods: We performed a retrospective chart review of all children that underwent percutaneous closure of a PMVSD at Inkosi Albert Luthuli Central Hospital, from October 2010 until December 2016. Patients that had percutaneous closure of any VSD other than PMVSD, including residual VSD post-surgical closure, were excluded.
Results: Fourty two patients were included in our retrospective analysis, 27 females and 15 males, with a mean age of 6 years 6 months (Range: 2 years 9 months - 15 years 9 months). Mean follow-up was 2 years 3 months. Successful device delivery was achieved in 97.6%. A total of 30 patients (71.4%) had complete closure of their defect. Eleven (26.2%) patients had a residual but haemodynamically insignificant defect. Two patients had mild aortic regurgitation post procedure. Significant early complications included 1 patient with moderate tricuspid regurgitation and 2 patients with device embolisation. In one of these patients, the embolised device was retrieved and replaced with a larger device. In the second patient, surgical retrieval and closure was required. No cases of heart block were recorded.
Conclusions: In our experience, percutaneous closure of a perimembranous ventricular septal defect in a child appears to be safe and effective. SAHeart 2019;16:14-20

INTRODUCTION

Ventricular septal defects (VSDs) account for 20% - 40% of all cardiac lesions in the paediatric population.[1-9] Perimembranous VSDs (PMVSDs) are anatomically related to the aortic and tricuspid valves and are found in the upper part of the ventricular septum. PM VSDs make up 60% - 70% of all VSDs.[2,13]

Closure of a persistent, significant VSD is necessary, as volume overload of the left atrium and ventricle predisposes the patient to ventricular dilatation and dysfunction, arrhythmias, aortic regurgitation, pulmonary arterial hypertension, endocarditis, or a double-chambered right ventricle.[5,10] In addition, patients have a tendency to have frequent respiratory tract infections and failure to thrive where a VSD is left unrepaired.[2]

Surgical closure is the gold standard for repair of the VSD. It is now a routine procedure, with minimal mortality,[2,3,5,6,9,10] and is supported by a large body of data.[2] However, it can be associated with complications, largely associated with the need for cardiopulmonary bypass and sternotomy.[8,9] Such complications include residual VSD and the need for repeat surgery, complete atrioventricular block (CAVB), post pericardiotomy syndrome, wound infection, arrhythmias, neurologic complications following cardiopulmonary bypass, atelectasis, chylothorax, pneumonia, pulmonary hypertensive crisis with cardiac arrest, phrenic nerve palsy, and even death.[2,4,10] In South Africa, and especially in our centre, access to surgery is also dependent on finite resources such as beds in the intensive care setting and available theatre time.

The concept of transcatheter device closure for the treatment of cardiac defects has been recognised since the 1960s, with significant development of the procedure taking place over the last 2 decades.[1,2,10] The development and introduction of the Amplatzer device (St Jude Medical) (first described in 2002),[3] including muscular and perimembranous occluders, has made

percutaneous closure of perimembranous VSDs (a more complex procedure) accessible to an increased number of patients.[2,10] Previously, patients with a distance of at least 5mm between the defect and the aortic valve were eligible for closure of the defect with a muscular occluder. However, the more recent availability of the perimembranous occluder allowed for percutaneous closure of perimembranous defects with only 1 - 2mm between the defect and the aortic valve.[10] There is also literature reporting the use of a patent ductus arteriosus (PDA) occluder device to close VSDs percutaneously.[4,15]

The Paediatric Cardiology Department at Inkosi Albert Luthuli Central Hospital (IALCH) is located in Durban, South Africa. Due to resource limitations, the waiting list for surgery is long. Thus, in this particular setting, percutaneous closure is a very attractive and possibly a more cost-effective option.

The aim of this study was to determine the safety and efficacy of percutaneous closure of perimembranous ventricular septal defects in the cohort of children treated in this unit.

PATIENTS AND METHODS

We performed a retrospective chart review of all children that underwent percutaneous closure of PM VSD at our unit from October 2010 until December 2016. Patients that had percutaneous closure of any VSD other than PMVSD, including residual VSD post-surgical closure, were excluded. Ethics approval was obtained from the Biomedical Research Ethics Committee of the University of KwaZulu-Natal.

All patients with PMVSD were assessed clinically and with transthoracic echocardiography (TTE). Patients that presented with evidence of a significant left-to-right shunt, based on clinical and echocardiographic criteria, were considered for VSD closure. A left-to-right shunt was considered significant clinically when there was cardiomegaly on chest X-ray, failure to thrive or frequent respiratory tract infections. Echocardiographic criteria for a significant left-to-right shunt included left atrial dilatation (LA:Ao ratio >1.4) and/or LV EDD measurement with a Z-score of more than +2SD for age. Patients with a weight under 10kg, with defects considered too large or too close to the aortic valve, were referred for surgery. In the same period, 184 patients with VSD underwent surgical closure at our centre.

Catheterisation standard procedure

Informed consent was obtained prior to cardiac catheterisation from the patient's parent and/or guardian. Prophylactic antibiotics (cefazolin 25mg/kg/dose) were administered 1 hour before the procedure and this was followed up by 2 doses post procedure. The procedure, to date, has been performed under general anaesthesia. After puncture of the femoral vessels, heparin 100IU/kg was given (max 5000IU) and the activated clotting time (ACT) was monitored regularly throughout the procedure. We aimed to keep the ACT at between 200 and 300 seconds during the procedure.

Fluoroscopy, transoesophageal as well as transthoracic echocardiography were used. Characteristics of the VSD and relating aorta were assessed using standard right and left cardiac catheterisation, standard left ventriculography and angiography, as well as transoesophageal echo. A device 1 - 2mm larger than the VSD diameter was selected.[2,10]

The VSD was crossed with an appropriate catheter from the arterial side – usually a Judkins Right coronary catheter with an angled Terumo wire. If the strategy was to close the VSD retrogradely, as may be the case with some of the Amplatzer Duct Occluder (ADO) devices (ADO II) (St Jude Medical) and Amplatzer Muscular VSD occluders (St Jude Medical), the wire and catheter were placed in the pulmonary artery. However, if the plan was to deliver the device through the vein, then an arteriovenous circuit was created. An exchange length wire, usually a Noodle Wire (St Jude Medical) was passed through the catheter. A snare was used to capture the wire through an appropriate catheter placed in the femoral vein. The snared wire was then exteriorised from the femoral vein, creating an arteriovenous loop. More recently, an alternate method with the VSD crossed from the venous side directly, has been employed where possible. An appropriate delivery sheath was then introduced from the venous side and passed through the VSD over the guidewire and ideally placed in the LV or alternatively in the aorta. The guidewire was then removed and the occluder attached to a delivery wire, which was then passed through the sheath and deployed over the defect. The position of the device was assessed using echocardiography and fluoroscopy. The aortic and tricuspid valves, in particular, were carefully assessed for worsening regurgitation. When the device position was considered to be stable and satisfactory, it was released.

All patients were transferred back to the ward post procedure, and were monitored overnight. A repeat 12-lead ECG, chest X-ray, echocardiogram and urine analysis (to check for signs of haemolysis) were done on the day after the procedure and were analysed on the same day. Aspirin 3 - 5mg/kg daily was started after the procedure and was continued for 6 months post device closure. After discharge, patients were followed up in our clinic at 4 - 6 weeks post procedure, and then 6 monthly to yearly thereafter if stable. At each follow-up visit, a clinical assessment, chest X-ray, ECG and transthoracic echo was done. ECGs were evaluated at the time of the visit and the standard

procedure is to record rate, rhythm, QRS axis, PR interval and signs of atrial enlargement or ventricular hypertrophy and any arrhythmias for each ECG done.

Devices

Devices used included ADO I and II, as well as the Amplatzer Membranous and Muscular VSD occluders (St Jude Medical) – see Figure 1. Selection of the device was based on the anatomy of the VSD and the distance from the aorta.

RESULTS

Patients

Forty two patients were included in our retrospective analysis. In 41, the indication for PM VSD closure was a significant left-to-right shunt. One had closure because of a preceding episode of infective endocarditis with significant tricuspid regurgitation (TR) due to a vegetation.

Twenty seven patients (64.3%) were female and the mean age for our cohort was 6 years 6 months (Range: 2 years 9 months - 15 years 9 months). The lowest weight was 11.4kg, with a median weight of 20.05kg. Table I delineates patient data with the relevant ranges.

Associated syndromes were Pena Shokeir syndrome in 1 patient and Down's syndrome in 2. One patient had moderate mitral regurgitation (MR) prior to PMVSD closure due to Double orifice mitral valve. The MR remained unchanged post PMVSD closure.

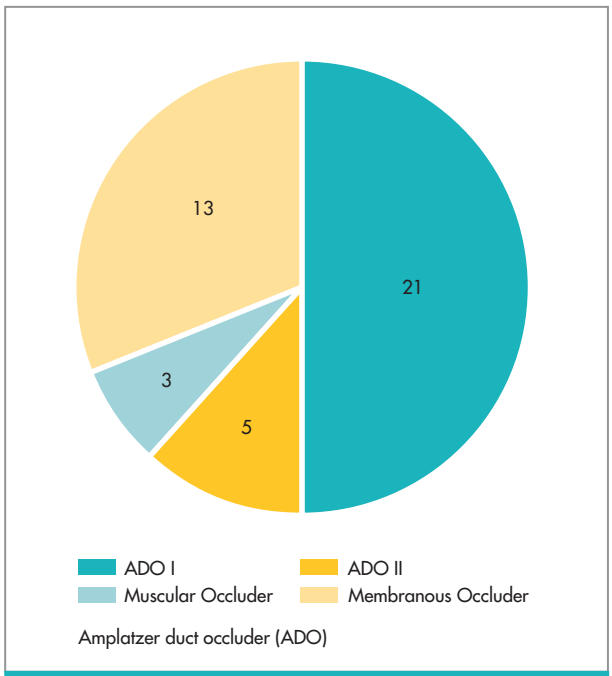

FIGURE 1: Devices used.

Cardiac catheterisation

Femoral venous and arterial access was used in all 42 patients. In 31 patients (73.8%) an arteriovenous loop was created to deliver the device. The ADO I device was the most common device used for percutaneous closure in 21 of the 42 patients (50%), followed by a membranous occluder, ADO II and muscular occluder (Table II). We used angiography and echocardiography in all patients to determine a stable device position before release. Three patients underwent combined procedures, 2 patent ductus arteriosus (PDA) closure with a coil, and 1 patient closure of a secundum atrial septal defect (ASD).

More than 1 ventricular septal defect was present in 10 patients. In each case the larger defect was closed. A total of 30 patients (71.4%) had complete closure of their defect. Eleven (26.2%) patients had a residual but haemodynamically insignificant defect. Of these, 8 patients were noted to have more than one VSD at the time of percutaneous closure.

Fluoroscopy time, procedural time (from time of induction of anaesthesia), dose of radiation, and amount of contrast used are summarised in Table III.

TABLE I: Patient data.

Variable	Value
Male	15 (35.7%)
Female	27 (64.3%)
Age (years), mean (range)	6.6 (2.8 - 15.8)
Weight (kg), mean (range)	20.4 (11.4 - 41.6)
Height (cm), mean (range)	114.8 (87 - 163)
Body Surface Area (m^2), mean (range)	0.80 (0.52 - 1.37)

TABLE III: Procedural data as median (range).

Procedure time (min)	130 (55 - 360)
Fluoroscopy time (min)	29.05 (8.13 - 169.40)
Radiation dose (uGym2)	3862.65 (270.62 - 22029.20)
Contrast used (ml)	80.0 (28.0 - 170.00)
Contrast/kg body weight (ml/kg)	4.35 (1.37 - 7.83)

TABLE 11: Overview of device used, size, method and outcome.

ID	Weight*	Device Used	Size	Method	Outcome	Early complications	Late Complications
1	22.90	Amplatzer Duct occluder II	5x4	AV Loop	Residual VSD	Moderate TR	
2	27.10	Muscular occluder	10	AV Loop	Residual VSD		
3	21.70	Membranous occluder	8	AV Loop	Closed		
4**	14.70	Muscular occluder	14	AV Loop	Closed		
5	14.80	Membranous occluder	8	AV Loop	Closed		
6	22.40	Membranous occluder	9	AV Loop	Closed		
7	18.70	Amplatzer Duct occluder I	8x6	AV Loop	Closed		Moderate AR
8	34.40	Muscular occluder	8	AV Loop	Residual VSD		
9**	11.70	Amplatzer Duct occluder II	4x4	Arterial	Residual VSD		
10	30.40	Amplatzer Duct occluder II	12x10	Arterial	Closed		
11	23.40	Membranous occluder	8	AV Loop	Closed		
12	20.80	Amplatzer Duct occluder I	10/8	AV Loop	Closed		
13	23.00	Membranous occluder	10	AV Loop	Closed		
14	19.20	Amplatzer Duct occluder I	10/8	AV Loop	Closed		
15	20.90	Membranous occluder	6	AV Loop	Closed		
16	31.00	Membranous occluder	6	AV Loop	Closed		
17	26.60	Membranous occluder	11	AV Loop	Closed		
18	11.60	Amplatzer Duct occluder II	6/4	AV Loop	Closed		
19	15.00	Membranous occluder	9	AV Loop	Closed		
20	28.00	Membranous occluder	7	AV Loop	Closed		
21	19.50	Amplatzer Duct occluder I	10/8	AV Loop	Closed	Device embolisation	
22	17.50	Membranous occluder	10	AV Loop	Closed		
23	24.00	Amplatzer Duct occluder I	10/8	AV Loop	Residual VSD		
24	16.30	Amplatzer Duct occluder I	12/10	AV Loop	Closed		
25**	21.00	Membranous occluder	7	AV Loop	Closed		
26	19.70	Amplatzer Duct occluder I	12/10	AV Loop	Closed		
27	21.40	Amplatzer Duct occluder I	8/6	AV Loop	Closed		
28	32.00	Membranous occluder	8	AV Loop	Residual VSD		
29	41.60	Amplatzer Duct occluder I	8/6	AV Loop	Residual VSD		
30	16.60	Amplatzer Duct occluder I	10/8	AV Loop	Closed		
31	22.00	Amplatzer Duct occluder I	8/6	AV Loop	Closed		
32	16.20	Amplatzer Duct occluder I	8/6	AV Loop	Closed		
33	20.40	Amplatzer Duct occluder II	5/6	Arterial	Closed		
34	14.10	Amplatzer Duct occluder I	10/8	AV Loop	Residual VSD		
35	21.20	Amplatzer Duct occluder I	8/6	Venous	Residual VSD		
36	11.40	Amplatzer Duct occluder I	8/6	Venous	Closed		
37	13.20	Amplatzer Duct occluder I	8/6	Venous	Residual VSD		
38**	17.00	Amplatzer Duct occluder I	10/8	Venous	Closed		
39	13.80	Amplatzer Duct occluder I	8/6	Venous	Closed		
40	12.70	Amplatzer Duct occluder I	8/6	Venous	Residual VSD		
41	12.80	Amplatzer Duct occluder I	8//6	Venous	Failed	Device embolisation	
42	13.00	Amplatzer Duct occluder I	8/6	AV Loop	Closed		

**Weight in kilograms, **Follow-up <1 year, AV = arteriovenous, Residual VSD = haemodynamically insignificant residual VSD.*

Complications

One patient had elevated ST segments on electrocardiogram (ECG) after the sheath was placed across the VSD. There were no changes in blood pressure or heart rate and no pericardial effusion on the TOE. A coronary angiogram was done that demonstrated normal coronaries and the cause for this change in ST segments remains unclear. The patient's subsequent ECGs demonstrated sinus rhythm with normal ST segments.

Another patient had a transient nodal rhythm after the sheath was placed across the VSD. This resolved spontaneously without any intervention or change in haemodynamic parameters.

Only one patient had transient pulse loss post percutaneous VSD closure. Enoxaparin was administered and the pulse recovered within a few hours. There were no incidences of bleeding or haematoma reported.

Significant complications were documented in 3 patients. One had moderate tricuspid regurgitation post device closure, and 2 had device embolisation. In one of these patients, the embolised device (ADO II) was retrieved and replaced with a larger device (ADO I). In the second patient, device embolisation (ADOI) was detected on clinical review the morning following the procedure, and attempts at retrieval in the cardiac catheterisation laboratory were unsuccessful. This patient underwent surgical retrieval of the occluder from the left pulmonary artery followed by VSD closure. The patient had an uneventful post-operative course and was discharged within 8 days of the initial percutaneous procedure. The index case with moderate TR post procedure had complete resolution of the TR at the latest follow-up visit.

Two patients had mild aortic regurgitation (AR) post procedure. In 1 of these patients there appears to have been progression of the AR after 5 years, with some concern that the device was impinging on the right coronary cusp – a finding not appreciated immediately post procedure. The other patient has had no progression of the initial AR. Both patients remain under close follow up.

No cases of heart block or significant arrhythmias were recorded.

Hospital stay post procedure

Thirty one patients were discharged the day following the procedure (range 1 day - 11 days, median 1 day) and 92.7% (38/41) of the cohort were discharged within 3 days of the procedure. In that group of patients, the reason for later discharge related to difficulty with transportation. One patient stayed for 8 days post procedure, as she was from a very rural part of the country with limited access to healthcare. Another patient was found to have bilateral hydronephrosis on routine screening of the kidneys post procedure, and was kept in hospital for 11 days post PMVSD closure for a renal diagnostic work up.

Follow up

All patients attended at least 1 post-procedural follow-up visit and the mean follow-up time was 2 years 3 months (n=42).

In terms of arrhythmias, 1 patient had a wandering atrial pacemaker noted on her first follow-up ECG, and 1 patient had a first degree heart block (PR interval 0.24 s). Both of these subsequently resolved.

Several patients with mild to moderate MR and TR prior to PM VSD closure showed resolution of their valve regurgitation on follow-up. The patient that had previous infective endocarditis (IE) and a vegetation on the tricuspid valve causing severe TR, had persistent TR post PMVSD closure.

Four patients were lost to follow-up before 1 year. At last post-procedural visit (range 1 - 16 weeks (n = 4)), 1 patient had residual flow through the device and the remaining 3 had complete closure of their VSDs.

DISCUSSION

Studies reviewing percutaneous closure of PMVSDs report successful closure rates of 90% - 100%,[2,6,10] rates that are comparable with those achieved by surgical closure.[3,9]

Reported major complication rates for percutaneous closure are 0% - 8.6%.[15] The main complication for consideration was that of complete atrioventricular node block (CAVB) (0% - 5.7%), a complication also associated with surgical repair of PMVSD (1% - 5%).[2,9,10] CAVB is associated with both percutaneous and surgical repairs of this defect, due to the anatomical association between the conduction system and the defect.[2,10,12] The AV node is found in the posterior upper membranous ventricular septum and branches into the left and right bundle in the posterior lower margin.[4] Postulated mechanisms of interference include direct compression or inflammation of, or scar tissue formation within the conduction tissue.[2] In percutaneous device closures, CAVB tends to occur more frequently,[7] at a later stage, and in a less predictable fashion[10] when compared to surgery. Young age at time of implantation, specifically age less than 6 years, has been identified as the primary risk factor in some studies.[10] Similarly, various studies reported use of the Amplatzer PMVSD occluder as a common factor in patients presenting with early and late

CAVB.[10,16,17] After these reports and further case reports of late CAVB associated with this device, a decision was made in our centre, in 2013, to discontinue use of the membranous occluder for percutaneous VSD closure.

No cases of CAVB were reported in our review. Late CAVB has been described 3 - 5 years post device closure[15] and 10 of the patients included in our review had not completed 3 years of follow-up visits. Due to the relatively small cohort size, it is impossible to speculate whether device size or type influenced the absence of CAVB.

In a study by Oses, et al., patients were followed up for 4 years. Late onset of CAVB was not identified and QRS intervals remained constant. Of concern was that the PR interval increases with time. This finding has been flagged for ongoing monitoring and follow-up.[11] In that particular study, the Amplatzer VSD occluder was used and there was no incidence of CAVB in the patients with a prolonged PR interval after 4 years of follow up.[11]

In our cohort, 1 patient was noted to have a prolonged PR interval 2 years 7 months post procedure (PR interval of 240ms at age 15 years). Fortunately, this has resolved, and his PR interval has remained at 0.2 seconds. No other significant rhythm abnormalities were reported in our review.

Other recognised complications of percutaneous perimembranous VSD closure include other rhythm abnormalities (atrial fibrillation, bundle branch blocks), device embolisation, and vascular complications (for example, femoral arterial thrombosis).[2,11] New valve regurgitation or significant residual shunts have not generally been identified as problematic in some studies;[2] however, aortic and/or tricuspid insufficiency and residual shunts have been cited as potential complications in other reviews.[7,9] Other studies have identified serious complications such as perihepatic bleeding, cardiac perforation, rupture of tricuspid valve chordae tendineae, and procedure-related death.[2,4] The surgical approach is associated with a higher complication rate. However, many of these are minor (i.e. complications not requiring treatment, including small pneumothoraces or small pleural effusions).[3,9]

For the patient, percutaneous VSD closure has considerable benefit, including less psychological impact due to absence of a surgical scar, no need for intensive care admission, less pain, and shorter time in hospital.[2,10]

When comparing percutaneous vs. surgical closure, it is important to note the difference in the patients selected for each treatment option. The patient profiles are significantly different and this should be considered when interpreting outcomes and complications of these 2 groups of patients. Patients suitable for device closure tend to be older (with correspondingly higher weights), with smaller VSDs, and no associated cardiac defects that require surgical intervention.[9]

Complications included device embolisation in 2 patients. Retrospectively, both instances of embolisation could have been prevented with a more appropriately sized device, as previously reported.[10,14,15]

Valve regurgitation is a complication noted in various reports.[2,7,9,10-16] If significant AR occurs during the procedure, the procedure is usually abandoned (unless the device can be repositioned to abolish AR) and the patient referred for surgical closure. None of our patients had new onset AR during the procedure. However, 2 of our patients had new onset AR the day after the procedure, both classified as mild. Some reports mention trivial to mild post-procedural AR occurring in up to 3.0% of patients[2,7,16] and suggest that there is no progression of this regurgitation during follow-up.[2,14,16] In 1 patient from our cohort, the AR remained mild and asymptomatic. However, in the other, there has been apparent progression at 5 years and 11 months post procedure. This may suggest that mild valve regurgitation could progress in patients where the device is in very close proximity to the aortic valve; any degree of neo-AR is, therefore, unacceptable.

One patient developed moderate TR the day after the procedure and was evaluated with TTE for any chordal injury. This was excluded, and, on follow-up, the TR resolved.

Closure rates post percutaneous closure vary according to what definition is used and a distinction is made between complete closure and successful closure.[11,14] At the time of our study, 71.42% (30/42) of patients were classified as having complete closure. Complete closure is reported to be 71% - 100%.[2,5,8,10,15,16] Eleven patients had a residual but haemodynamically insignificant shunt. Spontaneous closure is still a possibility, as is readmission for closure of remaining defects percutaneously, should this be indicated. One patient included in the group with residual defects had a small leak through the device initially, but was subsequently lost to follow-up.

Studies that report on successful percutaneous closure define this as device placement in the correct position, with no significant complications (valve regurgitation) or a significant shunt that requires surgical closure.[16] If we use that as a measure of outcome in our review, the success rate is 97.6% (41/42), similar to other reports.[2,5,8,10,15,16]

Limitations of the study include the limited follow-up period, the relatively small number of patients, and the significant number of patients who had defaulted follow-up.

CONCLUSION

Percutaneous closure of perimembranous ventricular septal defects in children appears to be safe and effective. Evaluation over a longer period of time is, however, still necessary. Improvement in device design may be important in preventing complications related to conduction defects and valve regurgitation.

Conflict of interest: none declared.

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