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Original Article / Özgün Makale

Single-center results of the use of transcatheter closure for ventricular septal defects

Tek bir merkezin transkateter ventriküler septal defekt kapatma sonuçları

Nazmi Narin,[1] Ali Baykan,[1] Mustafa Argun,[1] Abdullah Özyurt,[1] Özge Pamukçu,[1] Sadettin Sezer,[1] Sertaç H Onan,[1] Gökmen Zarasız,[2] Kazım Üzüm[1]

Departments of [1]Pediatrics, Division of Pediatric Cardiology, [2]Biostatistics and Medical Informatics, Medical Faculty Erciyes University, Kayseri, Turkey

ABSTRACT

Background: This study aims to investigate the safety, efficacy, and mid- to long-term follow-up results of percutaneous closure of ventricular septal defects (VSD).

Methods: The data of 53 patients (30 boys, 23 girls; median age 6.5 years; range 3 to 19 years) who were performed transcatheter closure of VSD between September 2006 and April 2013 in a single center were retrospectively analyzed. Perimembranous (n=42) and muscular (n=11) VSDs with hemodynamic significance which were suitable for transcatheter closure were included in the study. Four different devices were used in the study: the perimembranous Amplatzer VSD occluder, muscular Amplatzer VSD occluder, ductal Amplatzer occluder I, and II.

Results: Median body weight was 21.5 kg (range 13 to 87 kg). The ratio of successful closure was 92%. One patient died due to complete atrioventricular block five days after the procedure and one patient died due to cerebral hemorrhage one day after the procedure.

Conclusion: Transcatheter closure of VSDs is an efficient method in selective cases. However, occurrence of complete atrioventricular block is a major concern in this procedure. Surgery should be considered as the first treatment choice in perimembranous VSD. However, in selected patients, transcatheter approach can be used for defect closure when necessary measures are taken against possible complications.

Keywords: Arrhythmia; catheterization; complication; device; echocardiography; heart defects.

ÖZ

Amaç: Bu çalışmada ventriküler septal defektlerin (VSD) perkütan kapatılmasının güvenilirliği, etkinliği ve orta-uzun dönem takip sonuçları araştırıldı.

Çalışma planı: Eylül 2006 - Nisan 2013 tarihleri arasında tek bir merkezde transkateter VSD kapatılması uygulanan 53 hastanın (30 erkek, 23 kız; ort. yaş 6.5 yıl; dağılım 3-19 yıl) verileri retrospektif olarak incelendi. Çalışmaya transkateter kapatılma için uygun, hemodinamik olarak anlamlı perimembranöz (n=42) ve musküler (n=11) VSD'ler dahil edildi. Çalışmada dört farklı cihaz kullanıldı: perimembranöz Amplatzer VSD oklüder, musküler Amplatzer VSD oklüder, duktal Amplatzer oklüder I ve II.

Bulgular: Ortanca vücut ağırlığı 21.5 kg (dağılım 13-87 kg) idi. Başarılı kapatma oranı %92 idi. Bir hasta işlemden beş gün sonra tam atriyoventriküler blok nedeniyle ve bir hasta da işlemden bir gün sonra beyin kanaması nedeniyle öldü.

Sonuç: Ventriküler septal defektlerin transkateter kapatılması seçilmiş olgularda etkin bir yöntemdir. Bununla birlikte, tam atriyoventriküler blok oluşumu bu işlemde önemli bir sorundur. Perimembranöz VSD'de cerrahi ilk tedavi seçeneği olarak düşünülmelidir. Öte yandan, olası komplikasyonlara karşı gerekli önlemler alındığında, transkateter yaklaşım seçilmiş olgularda defektin kapatılması için kullanılabilir.

Anahtar sözcükler: Aritmi; kateterizasyon; komplikasyon; cihaz; ekokardiyografi; kalp defektleri.

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Correspondence: Mustafa Argun, M.D. Erciyes Üniversitesi Tıp Fakültesi Çocuk Hastalıkları Anabilim Dalı, Çocuk Kardiyolojisi Bilim Dalı, 38039 Talas, Kayseri, Turkey.
Tel: +90 352 - 207 66 66 e-mail: dr.margun@hotmail.com

Ventricular septal defects (VSDs) are the most common form (approximately 20%) of congenital heart disease when not taking into account bicuspid aortic valves.[1] Eighty percent of these defects are perimembranous and involve the membranous septum and the adjacent area of the muscular septum.[2] The usual treatment for VSDs is open heart surgery, which was first carried out by Lillehei et al.[3] in 1955. The open surgical approach has been widely accepted with minimal mortality, but it still carries the potential risks of complete heart block, a residual shunt, postpericardiotomy syndrome, wound infection, and neurological sequelae associated with cardiopulmonary bypass (CPB).[4,5] Various devices have been used as alternatives to surgery beginning with the first report of transcatheter VSD closure by Lock et al.[6] in 1988. Moreover, the transcatheter closure of congenital VSDs has become more effective with the development of the Amplatzer™ VSD occluder (St. Jude Medical Inc., St. Paul, MN, USA)[7] because there is no scarring, less pain, shorter hospital stays, and lower costs with this method than with traditional open heart surgery.[8] However, the possibility of complete atrioventricular block (AVB) is a major concern with this procedure.[9] In this study, we report the mid- and long-term results of 53 patients who underwent VSD closure with various devices.

PATIENTS AND METHODS

The data of 53 patients (30 boys, 23 girls; median age 6.5 years; range 3 to 19 years; median weight 21.5 kg; range 13 to 87 kg) who underwent transcatheter closure of a VSD between September 2006 and April 2013 at a single center was analyzed retrospectively. In addition, the clinical and electrocardiographic findings along with the conventional two-dimensional and color Doppler transthoracic echocardiographic (TTE) parameters were recorded.

Only those patients with a muscular or perimembranous VSD with a hemodynamically significant left to right shunt that was detected via a clinical examination and echocardiography were included in the study. Those with the following characteristics were excluded: *(i)* body weight <8 kg and age <3 years old, *(ii)* a non-aneurysmal perimembranous VSD (PMVSD) with a subaortic rim of <2 mm, *(iii)* pulmonary systolic pressure of >70 mmHg as identified by TTE, *(iv)* a right to left shunt, *(v)* sepsis, *(vi)* a defect associated with complex heart lesions *(vii)* a contraindication to antiplatelet therapy.

The four devices used in the study were the Amplatzer™ muscular VSD occluder (MVSDO), the Amplatzer™ PMVSDO, the Amplatzer™ duct occluder I (ADO I), and the Amplatzer™ duct occluder II (ADO II). The MVSDO was used in 11 patients with an MVSD. The PMVSDO was preferred for the 32 patients with a PMVSD. The ADO I device was used in the nine patients with an aneurysmatic PMVSD because it was suitable for the shape of the defect, and the ADO II device was used in one patient.

The catheterization procedure was carried out routinely under general anesthesia, and the patients were given antibiotics intravenously before the procedure. In addition, an electrocardiogram monitor was used throughout the procedure. The patients also initially received heparin 100 IU/kg, and additional doses were given when necessary to maintain an activated clotting time (ACT) of >200 seconds. The preferred vascular access sites were the right femoral vein and the left femoral artery. Left ventriculography in the left anterior oblique view was used to show the location and size of the VSD and then the procedure was performed with fluoroscopic and transesophageal echocardiographic (TEE) guidance. The size of the VSD size was measured on the right ventricular side, and a ventricular septal aneurysm was present in 15 of the patients with a PMVSD (35%). In these cases, the size was also measured on the right ventricular side and at the narrowest part. The appropriate device size was chosen to be at least 1 to 2 mm larger than the VSD size as measured at the largest diastolic phase by color Doppler TEE or ventriculography. A 4 or 5 French (F) right Judkins catheter or a partially cut pigtail catheter was then passed through the VSD. Afterwards, a hydrophilic guidewire was passed across the defect into the right ventricle and on into the pulmonary artery or superior vena cava (SVC) where it was snared and withdrawn from the femoral vein. Thus, an arteriovenous loop was established. Subsequently, a long sheath (6-12F) was advanced into the left ventricle via the arteriovenous circuit. An occluder was then inserted into the delivery sheath and positioned on the VSD. If the device position was satisfactory according to the fluoroscopy and TEE, the device was released. After the procedure, the position of the occluder was assessed via TEE and left ventriculography. The arterial approach was used in only two patients with PMVSDs. All of the patients were followed up with continuous electrocardiography (ECG) monitoring during the first 24 hours after the procedure and were discharged within five days of the procedure. Acetylsalicylic acid (5 mg/kg/daily) was administered to all patients for six months, and physical examinations, ECG, and TTE were performed at the postprocedural first, third, sixth, and 12th months

and yearly thereafter. Furthermore, 24-hour Holter monitoring was performed three months after the procedure.

Shapiro-Wilk's and Levene's tests were used to assess the normality and changes in homogeneity of the data. Values were expressed as frequencies and percentages, mean ± standard deviation (SD), or median and 25^{th}-75^{th} percentiles due to the normality and variable type of the data. To compare the defects, device sizes and defects/device sizes were used, and for all statistical analyses, a p value of <0.05 was considered to be statistically significant. All analyses and calculations were done using the SPSS for Windows, Version 15.0 software program (SPSS Inc., Chicago, IL, USA).

RESULTS

The median end-diastolic VSD diameter for the patients according to TEE was 4.3 mm (range 2.8-8 mm), and the median Qp/Qs ratio was 1.8 (range 1.4-3.5). In addition, the median mean pulmonary artery pressure (mPAP) was 24 mmHg (range 18-52 mmHg). Follow-up was carried out until April 2013, and there was a median follow-up period of 32 months (range 1-79 months). The baseline data is given in Table 1.

Furthermore, one patient underwent a pulmonary banding operation, two had mild pulmonary stenosis, two had mild mitral regurgitation, one had patent ductus arteriosus, one had a VSD with a residual shunt and had previously surgery for this. Additionally, an Amplatzer™ Septal Occluder was used to close an atrial septal defect (ASD) in another patient. The VSD closure was successful in 49 patients (92%), but it failed in four others. In one patient, the device was retrieved because we were unable to achieve a good device position via ADO I, and transient complete atrioventricular block (AVB) occurred in another. Therefore, we ended the procedure, and normal sinus rhythm returned after one hour. In another patient, the device was embolized in the femoral artery during the PMVSD closure via ADO I, and in the fourth patient, the device was embolized in the left pulmonary artery during the PMVSD closure using PMVSDO. The embolized devices were then captured by a snare catheter and retracted into the delivery system.

Table 1. Baseline characteristics of the 53 patients who underwent transcatheter ventricular septal defect closure

	n	%	Median	Range
Gender				
Female	23	43		
Male	30	67		
Age (years)			6.5	3-19
Age distribution in years				
<5	15	28		
5-10	24	45		
>10	14	27		
Weight (kg)			21.5	13-87
Defect type				
Perimembranous ventricular septal defect	42			
Muscular ventricular septal defect	11			
Successful closure rate	49/53	92		
Device type				
Perimembranous amplatzer VSD occluder	32	60		
Muscular amplatzer VSD occluder	11	21		
Amplatzer occluder I	9	17		
Amplatzer occluder II	1	2		
Defect measurement in (mm)			4.3	2.8-8
Device diameter			6	4-10
Device/defect measurement rate				1.4
Catheterization				
Qp/Qs			1.8	1.4-3.5
Pulmonary arterial mean pressure (mmHg)			24	18-52

VSD: Ventricular septal defect.

Unfortunately, two patients died (3.8%) due to severe complications. In a four-year-old boy whose VSD had been closed with a 6 mm PMVSDO, complete AVB occurred four days after the procedure. This condition had not been observed during the two-day follow-up period in the hospital, so the patient had been discharged. He had syncope four days after transcatheter closure. He also had bradycardia with a heart rate of 36 beats/min. Electrocardiography revealed the complete AVB, and even though a transient transvenous pacemaker was implanted immediately in the patient, he did not survive. The other patient was a three-year-old girl whose 4 mm VSD was closed with a 6 mm PMVSDO. On the second day, she suddenly lost consciousness, and a cerebrovascular aneurysm and cerebral hemorrhage were detected via computed tomography (CT). The patient was not under the prophylactic heparin treatment; therefore, this intracranial hemorrhage was thought to be due to the cerebrovascular aneurysm. Despite all of our efforts, she also did not survive.

In one patient, a rupture of the chordae tendineae caused mitral insufficiency, but the patient remained asymptomatic. In another patient, right bundle branch block (RBBB) developed following the procedure, but in the other patients, the BBBs were tolerated well after the VSD closure. All of the complications are summarized in Table 2.

The complete closure rate was almost perfect at 98%, but one patient experienced a mild residual defect after the procedure. During the procedure, no deaths, cardiac perforations, or cerebrovascular events occurred, and no femoral artery damage, femoral artery thrombosis, inguinal hematomas, or brachial palsy-like complications were noted. Moreover, during the follow-up period, no additional complications, such as late AVB, were encountered.

Table 2. Major and minor complications associated with the 53 patients who underwent transcatheter ventricular septal defect closure

	n	%
Major complications	2	3.8
Death due to complete atrioventricular block	1	1.9
Death due to a cerebral hemorrhage	1	1.9
Minor complications	5	9.4
Transient complete atrioventricular block	1	1.9
New valvular regurgitation less than two grade	1	1.9
Device embolization with transcatheter removal	2	3.8
Right bundle branch block	1	1.9

Our statistical analysis showed that the mean defect size of the 49 patients who had successful VSD closure was 4.3±0.2 mm, whereas the mean defect size of the four whose attempted VSD closure failed was 6.7±0.7 mm. The mean age of the seven patients who developed procedure-related complications was 4.8±0.9 years, and their mean body weight was 17.1±2.6 kg. The mean age of the patients without complications was 8.4±3.1 years, and their mean body weight was 28.7±3.1 kg. The defect size of the two patients who did not survive was 4 mm and the device size was 6 mm, whereas the defect size of the patients without complications was 4.6±1.5 mm and their device size was 6±1.5 mm. All of the complications occurred in the PMVSD cases (7/42; 16%), but no complications occurred in the MVSD cases (0/11; 0%) (p=0.322). Furthermore, all four of the unsuccessful VSD closure cases also involved patients with PMVSD, but there were no statistically significant differences with regard to these results.

DISCUSSION

Many VSDs are small and asymptomatic and close spontaneously over time.[10] In those that need closure, surgical or transcatheter closure should be carried out in order to prevent ventricular dilatation and dysfunction, aortic regurgitation, pulmonary arterial hypertension (HT), endocarditis, and arrhythmias.[11,12] In the last decade, the percutaneous approach for the closure of VSDs was developed by interventional pediatric cardiologists,[6] and this procedure has been the focus of a number of studies, and of which included the major complications associated with this procedure.[13-15]

In this report, a single-center experience involving the transcatheter closure of VSDs was presented. Other studies have reported a successful closure rate for this procedure of between 91 and 100%.[13-15] In our series, the success rate for implantation was 92%, and we saw only one procedural complete AVB, which was transient. However, Zhou et al.[16] reported two cases of complete AVB in their study. In addition, device embolizations occurred in two of their cases, and the device was removed percutaneously in a catheterization laboratory. In both of these cases the embolization could have been caused by the small size of the devices. Moreover, Holzer et al.[13] reported two device embolization cases in 75 MVSD patients in their study.

In two other studies, significant early complications occurred in two patients (3.8%), and complete AVB developed in approximately 1% of the patients who

underwent surgical repair for the VSDs.[17,18] In the study by Tucker et al.,[18] they determined that Down syndrome (DS) was the most significant risk factor for postsurgical complete AVB. Additionally, some studies have reported an incidence rate for complete AVB of between 0 and 5.7% for patients who undergo transcatheter device closure of VSDs.[13-15] In our study, permanent complete AVB only was seen in one case (1.9%) with a PMVSD, and this did not occur until four days after the procedure. After the death of this patient, we monitored each patient for five days after they underwent non-aneurysmal PMVSD closure.

Complete AVB is a major complication that can occur both during and after transcatheter closure of a PMVSD, and it develops because of the close proximity of the PMVSD to the conduction system.[16] In these cases, the device may interfere with atrioventricular conduction via direct compression trauma, or it may provoke an inflammatory response or cause scarring in the conduction tissue. Particularly in patients who are under three years old, the immaturity of their conduction system means the risk of complete AVB development is much higher. Zuo et al.[9] found that complete AVB is more common (18.2%) in this age group, with the risk for patients over three years of age being just 4.7%. However, our statistical analysis did not reveal any variable that could predict the occurrence of complete AVB in our patients.

In our series, the median follow-up was 32 months, and no late complete AVB was noted. However, complete AVB usually appears soon after surgical repair. In patients treated percutaneously, the occurrence of complete AVB is quite unpredictable, and it is usually seen later.[11] Because of this, careful monitoring of heart rhythm is essential during follow-up, especially in those subjects treated for a PMVSD. It is hard to estimate when complete AVB will occur in the late term; therefore, Holter monitoring is vitally important in the postprocedural first, third, and sixth months. It is also necessary to teach parents how to count the pulse rate so that they can seek immediate care at a third-level health center in cases of syncope and presyncope. This training is especially valuable for the early management of events before the development of irreversible changes. Some authors[9,11] have suggested that a course of steroids should be used to treat AVB, but we believe that the best course of action is prompt device removal for patients who develop complete AVB during the procedure.

A cerebral hemorrhage was seen on CT 24 hours after the procedure in a three-year-old girl in our study, and Yang et al.[19] reported one death due to an unexplained cerebral hemorrhage. Like Yang et al.,[19] we believe that this situation might have been caused by a cerebrovascular malformation.

Mild mitral insufficiency developed in one of our patients due to chordae tendineae rupture, and Yang et al.[19] reported three cases of new-onset mitral insufficiency that required surgery.

The mean defect size in the four patients for whom the VSD closure failed in our study was larger than that of the patients who experienced successful closure. Furthermore, the mean age and weight of the patients with complications were less than those for the patients without complications, and these expected results were in compliance with the literature.[9] The reason for the statistical insignificance in our study seemed to stem from the limited number of patients since we could find no association between the development of complications and the defect size or defect/device ratio.

Conclusion

Transcatheter closure of VSDs is an efficient method in select cases. However, the occurrence of complete AVB is a major concern with this procedure. In suitably selected patients, this procedure can be used for closing the defect when necessary measures are taken to guard against possible complications. Since the complication rate of VSD closure is high, precautionary indications of closure should be taken seriously, and educating the parents of these patients with regard to the occurrence of complete AVB is crucial. We believe that surgical treatment is preferable for most patients, especially when a hemodynamically important PMVSD is involved, and the decision between surgery and transcather closure needs to be made quickly. However, our results indicate that the surgical option should be prioritized over the VSD closure approach, especially in perimembranous cases.

Declaration of conflicting interests

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REFERENCES

1. McDaniel NL, Gutgesell HP. Ventricular septal defects. In: Allen HD, Driscoll DJ, Shaddy RE, Feltes TF, editors.

Moss and Adams' Heart Disease in Infants, Children, and Adolescents. 7th ed. Philadelphia: Lippincott Williams & Wilkins, a Wolter Kluwer business; 2008. p. 667-82.
2. Thanopoulos BV, Rigby ML, Karanasios E, Stefanadis C, Blom N, Ottenkamp J, et al. Transcatheter closure of perimembranous ventricular septal defects in infants and children using the Amplatzer perimembranous ventricular septal defect occluder. Am J Cardiol 2007;99:984-9.
3. Lillehei CW, Cohen M, Warden HE, Ziegler NR, Varco RL. The results of direct vision closure of ventricular septal defects in eight patients by means of controlled cross circulation. Surg Gynecol Obstet 1955;101:446-66.
4. Fu YC, Bass J, Amin Z, Radtke W, Cheatham JP, Hellenbrand WE, et al. Transcatheter closure of perimembranous ventricular septal defects using the new Amplatzer membranous VSD occluder: results of the U.S. phase I trial. J Am Coll Cardiol 2006;47:319-25.
5. Şahin MA, Güler A, Cingöz F, Günay C, Yokuşoğlu M. Ventriküler septal defekt tamir ameliyatında kordal ayrıştırma yöntemi. Turk Gogus Kalp Dama 2012;20:636-7.
6. Lock JE, Block PC, McKay RG, Baim DS, Keane JF. Transcatheter closure of ventricular septal defects. Circulation 1988;78:361-8.
7. Arora R, Trehan V, Kumar A, Kalra GS, Nigam M. Transcatheter closure of congenital ventricular septal defects: experience with various devices. J Interv Cardiol 2003;16:83-91.
8. Fu YC. Transcatheter device closure of muscular ventricular septal defect. Pediatr Neonatol 2011;52:3-4.
9. Zuo J, Xie J, Yi W, Yang J, Zhang J, Li J, et al. Results of transcatheter closure of perimembranous ventricular septal defect. Am J Cardiol 2010;106:1034-7.
10. Mercan AŞ, Akay T, Aşlamacı S, Tokel K, Varan B, Saygılı A. İnfant ventriküler septal defekt cerrahisinde risk faktörleri. Turk Gogus Kalp Dama 2001;9:149-52.
11. Butera G, Carminati M, Chessa M, Piazza L, Micheletti A, Negura DG, et al. Transcatheter closure of perimembranous ventricular septal defects: early and long-term results. J Am Coll Cardiol 2007;50:1189-95.
12. Bol-Raap G, Weerheim J, Kappetein AP, Witsenburg M, Bogers AJ. Follow-up after surgical closure of congenital ventricular septal defect. Eur J Cardiothorac Surg 2003;24:511-5.
13. Holzer R, Balzer D, Cao QL, Lock K, Hijazi ZM. Device closure of muscular ventricular septal defects using the Amplatzer muscular ventricular septal defect occluder: immediate and mid-term results of a U.S. registry. J Am Coll Cardiol 2004;43:1257-63.
14. Carminati M, Butera G, Chessa M, Drago M, Negura D, Piazza L. Transcatheter closure of congenital ventricular septal defect with Amplatzer septal occluders. Am J Cardiol 2005;96:52-58.
15. Handke R, Jux C, Schranz D, Schneider M. Transcatheter closure of perimembranous ventricular septal defects using umbrella devices. Catheter Cardiovasc Interv 2006;68:936-41.
16. Zhou T, Shen XQ, Zhou SH, Fang ZF, Hu XQ, Zhao YS, et al. Atrioventricular block: a serious complication in and after transcatheter closure of perimembranous ventricular septal defects. Clin Cardiol 2008;31:368-71.
17. Andersen HØ, de Leval MR, Tsang VT, Elliott MJ, Anderson RH, Cook AC. Is complete heart block after surgical closure of ventricular septum defects still an issue? Ann Thorac Surg 2006;82:948-56.
18. Tucker EM, Pyles LA, Bass JL, Moller JH. Permanent pacemaker for atrioventricular conduction block after operative repair of perimembranous ventricular septal defect. J Am Coll Cardiol 2007;50:1196-200.
19. Yang J, Yang L, Wan Y, Zuo J, Zhang J, Chen W, et al. Transcatheter device closure of perimembranous ventricular septal defects: mid-term outcomes. Eur Heart J 2010;31:2238-45.