TRANSCATHETER CLOSURE OF VENTRICULAR SEPTAL DEFECTS IN INFANTS AND CHILDREN

Reyadh A. Elgindy, Hala M. Agha, Hala S. Hamza, Sherief H. Abdel Rahman and Khaled M. Saleh

ABSTRACT

Objectives: The objective of this study was to evaluate the safety and efficacy of the transcatheter approach to correct congenital ventricular septal defects (VSDs) in a prospective clinical trial.

Background: VSD is a common congenital heart disease in children. Surgical closure of VSD is a well-established therapy but requires open-heart surgery with cardiopulmonary bypass. Although the transcatheter approach is associated with significant incidence of complete artioventricular block, it may provide a less invasive alternative.

Methods and results: Implantation of transcatheter devices was attempted in 30 patients (pts.) with congenital VSDs aged 1 to 14years from November 2013 to November 2014 and follow up for one year. The following anatomic types were present: 11 muscular, 19 perimembranous, single in 26, two in one and multiple in three, Mean VSD size was 6.6 mm (range 3–14), fluoroscopy time 51 min (range 13-107). Devices implanted were Amplatzer duct Occulder*I (ADOI)* in12, Amplatzer duct Occulder *II (ADOII)* in8, *Amplatzer muscular VSD* in 5, *PFM* in 3 and *Cribriform septal occluder* in 1 case. Procedure was successful in 25 cases (83.3%). Complications: device embolization in one case (surgical retrieval), unstable device in two pts., minor rhythm disturbances in 2 pts., complete heart block (cAVB) in one pt. NO deaths. All patients were Symptoms free and gaining weight one year after closure.

Conclusion Transcatheter closure of congenital VSDs offers encouraging results.

Recommendation: More experience and long-term follow-up are mandatory to assess safety and effectiveness of this procedure as an alternative to conventional surgery.

Keywords: Ventricular septal defect (VSD) - Interventional therapy - Amplatzer duct occluder II (ADO II) - Congenital heart disease - Follow-up.

INTRODUCTION

The Ventricular septal defect (VSD) is the most common congenital heart defect. These defects are classified according to their location within the septum: perimembranous, muscular and supracristal, with perimembranous VSD being the most common. Indications to VSD closure are symptoms of heart failure, signs of left heart chambers overload, and history of endocarditis. In patients with an overload of left heart chambers, closure is necessary in order to prevent pulmonary arterial hypertension, ventricular dysfunction, arrhythmias, aortic regurgitation, and development of double-chambered right ventricle.

The traditi5onal treatment is surgical repair, which was performed for the first time by Lillehei et al. 2 in 1954. The surgical approach is considered to be the gold standard, but it is associated with morbidity and mortality, patient discomfort, sternotomy, and skin scar.³⁻¹⁴

Percutaneous closure of VSD started in the late 1980s, after devices designed for the closure of atrial septal defects (ASD) or patent ductus arteriosus (PDA) were implanted in the interventricular septum^[4,16,20,21]

Transcatheter VSD closure avoids extracorporeal circulatory support of surgical approach and has the benefit of faster recovery, but both methods carry a potential risk of atrioventricular block (AVB). This occurs at an average rate of 5 % in most series but may be as high as 22 % ^{[8,14,25].} AVB may develop intraoperatively or postoperatively; with transcatheter closure it is more unpredictable and may occur late, months or even years after device implantation. The incidence of AVB also depends on the device used, and more flexible devices may reduce this incidence significantly.

No. 2

The aim of this study to evaluate the feasibility of transcatheter closure of muscular and perimembranous ventricular septal defects (VSDs) in infants and children using different devices and to determine the outcome of (VSDs) closure at pediatric cardiology unit and catheterization laboratory at Cairo University Pediatric Hospital (CUPH) from November 2013 through November 2015.

PATIENTS AND METHODS

We reviewed prospectively patients with VSD who underwent transcatheter VSD closure with 11/2013 to 11/2015.

Inclusion criteria

The following inclusion criteria were used: (i) isolated defects; (ii) congenital VSD; (iii) muscular or perimembranous (iv) haemodynamically significant VSD (demonstrated either by clinical data,echocardiography or angiography); (v) history of infective endocarditis.

Exclusion criteria

The exclusion criteria were as follows: (i) defects associated

with other cardiac lesions needing a surgical approach; (ii) irreversible pulmonary vascular disease greater than 7 Woods U/m^2);(iii) contraindication to antiplatelet therapy; (iv) sepsis; (v) the presence of asubaortic rim as shown by echocardiography in the long axis view less than2 mm in cases of perimembranous VSD.

Data collection

The data was collected prospectively, the data collected included demographic details (date of procedure, age, gender, weight, height), echocardiographic data position. (VSD associated anomalies. aortic. mitral or valve regurgitation, tricuspid subaortic rim in patients with perimembranous VSD as shown in figure 1 and 2), procedural details (access route, sheath size, VSD size on angiography, type and measure of device implanted, residual shunting, fluoroscopic, and procedural times). Furthermore, when closure failed, reasons for failure were recorded



Figure (1): (A) Two-dimensional image of perimembranous ventricular septal defect (pmVSD).



Figure (2): Transoesophagealechocardiography of amidmuscular VSD.

A major complication was defined as an event that resulted in death, long-term sequelae, need for immediate surgery, potentially life-threatening events, persistent arrhythmias needing long-term therapy (6 months) or pacemaker placement, ongoing haemolysis requiring blood transfusion, thrombosis that required thrombolytic therapy, and increased regurgitation needing valvalr device removal or drug therapy.

A minor complication was defined as an event that required drug therapy but was not lifethreatening, with no long-term (6 months) sequelae, and which did not require long-term therapy. The following were also included in this group: device embolization with transcatheter retrieval, haematoma of the groin, cardiac arrhythmias that required cardioversion of drug therapy during the procedure, minor degree trioventricular blocks, and transient loss of peripheral pulse needing only heparin therapy.

Definition of outcomes

Procedural success

Procedural success was defined by device implantation in the appropriate position with no need for surgery (for example due to significant residual shunt or significant valve regurgitation).

Residual shunt

A residual shunt was considered to be present if colour-Doppler flow mapping showed a left to right shunt across the interventricular septum. It was classified as follows; trivial (1 mm colour jet width), small (1–2 mm colour jet width), moderate (2–4 mm colour jet width), or large (more than 4mm colour jet width).

Devices used

The following devices were used: muscular Amplatzer VSD occluder, Amplatzer Duct occluder *I*, Amplatzer Duct occluder *II*, *Cribriform septal occluder* and PFM coil.

Closure protocol

All procedures had been performed in agreement with the ethical guidelines of the Cardiac Catheterization Unit in Pediatric Hospital, Cairo University.

The procedure was done under general endotracheal anesthesia. Access is obtained in the femoral vein, the femoral artery and the right internal jugular vein.

A femoral vein (FV) access was used to approach the closure of a

perimembranousVSD, and an internal jugular vein (IJV) was used for muscular VSD closure. An arteriovenous circuit must be created (internal jugular veinfemoral artery for muscularVSD and femoral vein-femoral artery for perimembranousVSD closure).

Left ventricular angiographies were obtained in axial projections for best evaluation of VSD size and position. addition in to Transesogheal echocardiography views. Left ventriculography (35° left anterior oblique/35° cranial) is performed to image mid-muscular, apical posterior defects. Anterior defects are better seen in 60° left oblique/20° anterior cranial (Figure 1, 2). For perimembranous angiographies VSD were performed using 60° left atrial oblique plus 20° cranial view. An angiogram of the ascending aorta was performed in 50° left atrial oblique view to check for aortic insufficiency.

The VSD is crossed from the left side by using a right Judkins catheter and a soft Guide-wire (0.035", Terumo); the wire was advanced to the pulmonary artery or to the SVC/IVC.

No. 2



Figure (3): Fluoroscopic and angiographic steps in the closure of a muscular VSD. A, LV angiogram demonstrates the presence of a single mid-muscular VSD; B. The wire is across the VSD from the left ventricle; C. The delivery sheath is in the left ventricle from the right internal jugular and the device is being advanced; D. The left sided disc is advanced into the left ventricle. E. LV angiogramto confirm the position of the left ventricular disc; F. The right disc is deployed in the right ventricle; G. The device is released from the delivery cable; H. LV angiogram shows complete closure of the muscular VSD with the Amplatzer muscular VSD device.

Then the wire was snared with Goose Neck Snare and а exteriorized out of the internal jugular vein or femoral vein establishing arteriovenous an circuit. Over the circuit, an appropriate size delivery sheath is advanced from the vein all the way until the tip of the sheath is in the ascending aorta. The dilator is withdrawn, and the sheath is pulled back in the left ventricle.

When the tip of the sheath is placed in the mid cavity of the left ventricle, the dilator and the wire gently removed; а left are ventriculogram is usually repeated, to confirm the position of the long sheath and also to obtain additional information on the position and the size of the VSD (figure 3). According to both angiographic and echocardiographic information, a VSD occluder

1–2 mm larger than the maximum size of the defect is chosen.

The device was attached to the delivery cable, loaded into the plastic loader, introduced and advanced into the sheath. The left disc was deployed in the left ventricular cavity, making sure it is not impinged in mitral valve apparatus, and then the entire system was withdrawn towards the septum, and the central waist and the proximal disc were deployed; a test angiogram was done to verify the correct position of the device.

2D TEE views were used to confirm the position of the two discs on left and right side of the septum, respectively, and the central waist within the septum. The device was then released. A final angiogram is performed approximately 10–15 min afterwards to assess the position of the device and the possible residual shunt (Figure 4).



Figure (4): Left ventricular angiogram: showing mid muscular VSD device after insertion of the device.

Post-operative care and follow-up:

All subjects underwent clinical examination, electrocardiography, chest X-rays, and TTE before discharge and at 1, 6, and 12 months after the procedure and yearly thereafter. Platelet antiaggregation therapy with aspirin 5 mg/kg/day peroral and endocarditis prophylaxis were prescribed for 6 months.

Statistical analysis

Data were analyzed using IBM© SPSS© Statistics version 23 (IBM© Corp., Armonk, NY, USA) and MedCalc© version 15.8 (MedCalc© Software bvba, Ostend, Belgium).

Al-Azhar Journal of Ped.	Vol. 19	<i>No. 2</i>	Jan 2016
The D'Agostino-Pearson was used to examine the norma of numerical data distributi Normally distributed numerical	test SD, lity and ion. rical ical or m	and skewed interquartile data were pr umber (%).	data as median range. Catego- resented as ratio
data were presented as mean	ı ±		

RESULTS

Procedural data

Table (1): General characteristics of all patients and types of devices.

Variable	Mean
Male	17(56.7%)
Female	13(43.3%)
Weight (kg)	16.2kg(6-45)
VSD type	
Perimembranous	19(63.4%)
Muscular	11(36.7%)
VSD Size	
Echo Size TTE	6.7(4-13mm)
Angio Size	6.6(3-14mm)
TEE Size	5.8(4-11mm)
Ratio defect/device	0.89
Device used	
ADOI	12
ADOII	8
Cribriform septal occlude	1
Amplatzer muscular VSD	5
PFM	3
Procedure Time	51.6(13-107)min

The percutaneous femoral venous and arterial approaches were used in all procedures. The size of the VSD measured in the angiogram ranged from 3 to 14

mm(mean 6.6 mm), in the echocardiogram from 4 to 14 mm (mean 6.7 mm). The mean size of the implanted device 8.1mm.

 Table (2): Correlation among VSD size estimations by 2DTTE, 2DTEE, or angiography and the device size

		VSD size by LV angiography	VSD size by TEE	VSD size by TTE	Device size
VSD size by LV angiography	Spearman rho	-	0.651	0.626	0.514
	p-value	-	< 0.001	< 0.001	0.004
VSD size by TEE	Spearman rho	0.651	-	0.826	0.431
	p-value	<0.001	-	<0.0001	0.020
VSD size by TTE	Spearman rho	0.626	0.826	-	0.514
	p-value	<0.001	<0.0001	-	0.004
Device size	Spearman rho	0.514	0.431	0.514	-
	p-value	0.004	0.020	0.004	-

Spearman rank correlation.

Significant correlation between VSD size by angiography ,TEE and TTE.Highly significant correlation between VSD by TTE and TEE.

 Table (3): Procedure outcome.

Procedure success		
Successful procedure	25	83.3
Unsuccessful procedure	4	13.3
Cause of failure		
Unstable device	2/4	50.0
Device embolization to right PA	1/4	25.0
Complete Heart block after insertion	1/4	25.0
(removed)		
Immediate complications		
Transient bradycardia	1/30	3.3
Heart block	1/30	3.3

Al-Azhar Journal of Ped.	Vol. 19	<i>No. 2</i>	Jan 2016
--------------------------	---------	--------------	----------

The procedure was successfully performed in 25 pts (83.3%). The procedure failed in 4 subjects due to the following reasons: unstable of the device in two patients, device emolization in one case and complete heart block in one case reported in table 3.

Table (4): Kaplan-Meier a	nalysis for time to complete VSD clo	osure	
Time after procedure (days)	Proportion with residual flow across VSD	SE	
0	0.48	0.10	
1	0.44	0.10	
90	0.28	0.09	
180	0.00	0.00	
Time to complete VSD closure	Value	95% CI	
Mean (days)	64.8	33.4 to 96.3	
Median (days)	0	0 to 90	

r

Significant shunt reduction was detected in all patients after successful VSD closure and only minor residual shunts were documented shown in Table 4. There was no increase in aortic or tricuspid valve regurgitation (TR) in any patient after procedure.

Table (5): LVEDD and LVESD z-scores before and 6 months after the procedure

Variabla	Before procedure		6 months after procedure				
v ariable	Mean	SD	Mean	SD	t	df	p- value
LVEDD z- score	1.75	1.54	1.42	1.19	2.004	29	0.054
LVEDS z- score	1.16	0.89	1.01	0.70	0.984	29	0.333



Figure (4): Kaplan-Meier curve for time to freedom from symptoms.

The success of the procedure is also reflected in the decrease of the LVEDD post procedure, as well as the improvement of clinical symptoms in those patients with symptoms related to the presence of the VSD prior to percutaneous closure as shown in table 5 and figure 4.

DISCUSSION

Ventricular septal defects are the most common congenital heart defects, with the perimembranous VSD as the most common subtype ^{[22].} Spontaneous closure occurs in 40-60 % of patients, mostly in preschoolers ^[6]. In patients with signs of LV volume overload only from left to right shunting from a increase VSD. an of right ventricular or pulmonary artery pressure has to be excluded. These patients may require defect closure to prevent ventricular dilatation dysfunction. and arrhythmias.

aortic or significant tricuspid regurgitation, or endocarditis ^{[17].}

Surgical closure has been the standard method of VSD closure. However, although it is generally a safe procedure, it does have some potential risks: complete AVB in 1-5 % of the patients, significant residual VSD in 1-5 % with the necessity to reoperate in 2 %, and perioperative death in 0.5 %. Furthermore sternotomy. general anesthesia and cardiopulmonary bypass may initiate significant co-morbidities, such as infection, tachyarrhythmias, and neurologic complications [12,18,22].

cardiologists Pediatric have been interested in closing VSDs interventionally for several years. Moderate success was achieved initially large deliverv with devices. where and systems residual shunting, induction of regurgitation of the valves (tricuspid and aortic valve), as well arrhythmia as and hemorrhage were dangers ^[15,27]. A transcatheter approach for the treatment of congenital heart diseases is appreciated by patients and their parents because of its psychological impact lesser (absence of a skin scar), shorter hospital time, less pain and discomfort. and avoidance of admission to an intensive care unit [8]. Closure of ASD, PDA, muscular VSD, and perimembranous VSD using transcatheter devices such Amplatzer as greatly occluders has been improved and widely reported ^{[5,9,13,23].} Therefore, in the current transcatheter closure era, of perimembranous VSD provides a valuable alternative to surgery. In our experience, the results of transcatheter VSD closure with the different devices are satisfactory: the procedure was successfully performed in 25 of 30 patients (83.3 %), confirming the results reported in other published studies for interventional VSD closure [1,3,10,24].

No deaths or haemolysis was reported directly after implantation or during follow-up. No vascular complication such as thrombosis, dissection, or aneurysm occurred. No evidence of infective endocarditis during follow up.

No. 2

One significant early complication occurred in our study with device embolization. In 10 y boy VSD perimembranous with measuring 1 cm x 0.9 cm using Muscular VSD device 10x6 the device empolyzed to RPA and failed trancatheter retrvial and surgical retrieval of the device with closure of VSD and the patient had no sequlae. In our study unstability of the device in two cases and removal of the device. One case female patient 1year and 7months old with perimembranous VSD measuring 8mm using ADOI 10/8 and the other case was female patient 4y and 3months old with perimembranous VSD measuring 8.5mm using ADOI 10/8 after realsing of the whole delivery system and confirming device position, device was not safely situated in position with excessive movements and instability due to large aneurysmal pouch so the decision was taken to quit the procedure and for another trial using PFM VSD coil.

In the current study occurrence of complete AV block (cAVB) in one case male patient 3y old with amidmuscular VSD 4mm on top of previously closed muscular defect using ADOI long sheath 6 fr failure to pass the VSD and occurrence of complete heart block and the procedure was aborted.

One significant complication occurred (1/30 patients, 3.2 %) in a 2.2-year-old girl (12.6 kg) with a perimembranous VSD (4 mm), a 4/4 mm ADO II was used to close the VSD. After release from the delivery system а device embolization in the RV occurred. It was possible to retrieve the device percutaneously: it was recaptured in the right ventricle using a goose-neck snare and an 8-F long sheath. The VSD was closed by surgery.no significant hemolysis with the need for retrieval of the device occurred arrhythmic complications (minor) occurred in two patients a 2-yearold girl (mVSD) developed.

Transient bradycardia after device implantation that lasted 5 min and adrenaline was administration. One patient (boy, 7 years, pmVSD) had AV rhythm (80/min) for 24 h postimplantation; this resolved spontaneously.

A trivial residual shunt was present in 10 patients (32.2 %) at

the end of the procedure. At discharge only three patients (10.3 %) had a tiny residual shunt, and the number was reduced to only two patients at follow-up.

One significant early complication occurred in our study with device embolization. This complication was transient and the patient had sequelae. no Retrospectively this event could have been prevented with appropriate sizing because the device may have been too small: 10/6 mm ADO II in a 10 mm sized defect result As ิล of that experience we now use the defect size and add 0.5-1 mm in order to determine the appropriate device size. Implantation of the ADO II has been described with the approach from either the LV or RV side due to the high flexibility of the device. Deployment of the LV disk first followed by passage through the defect and release of the RV disk at the end makes an appropriate distance to the aortic valve in our experience more realistic.

Statistical analysis showed that no variable predicted the occurrence of early complications. The incidence of major complications reported in the literature ranges between 0 and 8.6 % ^{[3, 11].} No significant valve regurgitation occurred in our series. Vol. 19

One serious concern of VSD closure is still the occurrence of complete AV block (cAVB). This complication may require pacemaker implantation, and it seems to be more frequent in young patients ^{[7].} The cAVB after surgical closure has been reported to occur in around 5.7 % of interventionally treated patients, but late occurrence 3-5 years after implantation also has been reported [3, 10]. Various mechanisms may be involved: mechanical compression, tissue, or inflammatory reaction followed by scar formation in the conduction tissue ^{[2].} It might be an advantage of the ADO II device that it may keep the incidence of cAVB low, due to either the flexibility of the device low profile the of the or implantation process. There was no incidence of cAVB in our study group with a median follow-up of 41 (range 0.6-67) months.

Ventricular septal defects in selected patients may be closed percutaneously using an ADO II device, as an off- label therapy. It appears that ADO II may be the preferable device for the closure of defects of moderate size (2-5 mm), especially in infants and small children, because of its better profile and trackability. The incidence of complications is acceptably possible low. Its superiority in terms of less AVB

development needs to be proven with longer follow-up periods, as late development has been described for other devices ^{[25].}

No. 2

REFERENCES

- 1. Arora R, Trehan V, Kumar A (2003) Transcatheter closure of congenital ventricular septal defects. Experience with various devices. J Interv Cardiol 16:83-91
- Bass JL, Gruenstein D (2012) Transcatheter closure of the peri- membranous ventricular septal defectpreclinical trial of a new Amplatzer device. Catheter Cardiovasc Interv 79(7):1153-1160. doi:10.1002/ccd.23367 Epub 2011 Dec 12
- 3. Bass JL, Kalra GS, Arora R et al (2003) Initial human experience with the Amplatzer perimembranous ventricular septal occluder device. Cathet Cardiovasc Intervent 58:238-245
- 4. Benton JP, Barker KS (1992) Transcatheter closure of ventricular septal defect: anonsurgical approach to the care of the patient with acute ventricular septal rupture. Heart Lung 4:356-364
- Bol-Raap G, Weerheim J, Kappetein AP, Witsenburg M, Bogers AJ (2003) Follow-up after surgical closure of congenital ventricular septal defects. Eur J Cardiothorac Surg 24:511-515
- Butera G, Chessa M, Piazza L, Negura D, Micheletti A, Carmi- nati M (2006) Percutaneous closure of ventricular septal defects. Expert Rev Cardiovasc Ther. 4(5):671-680 Review

- Butera G, Carminati M, Chessa M et al (2006) Percutaneous closure of ventricular septal defects in children aged b 12: early and midterm results. Eur Heart J 27(23):2889-2895
- Butera G, Carminati M, Chessa M, Piazza L, Micheletti A, Ne- gura DG, Abella R, Giamberti A, Frigiola A (2007) Transcatheter closure of perimembranous ventricular septal defects: early and long-term results. J Am Coll Cardiol 50(12):1189-1195
- Chessa M, Carminati M, Butera G et al (2002) Early and late complications associated with transcatheter occlusion of secun- dum atrial septal defect. J Am Coll Cardiol 39:1061-1065
- Hijazi ZM, Hakim F, Al Fadley F, Abdelhamid J, Cao QL (2000) Transcatheter closure of single muscular ventricular septal defects using the Amplatzer muscular VSD occluder: initial results and technical considerations. Catheter Cardiovasc Inter- vent 49:167-172
- 11. Hijazi ZM, Hakim F, Haweleh AA et al (2002) Catheter closure of perimembranous ventricular septal defects using the new Amplatzer membranous VSD occluder: initial clinical experience. Cathet Cardiovasc Interv 56:508-515
- 12. Hobbins SM, Izukawa T, Radford DJ, Williams WG, Trusler GA (1979) Conduction disturbances after surgical correction of ventricular septal defect by the atrial approach. Br Heart J 41:289-293
- Holzer R, Balzer D, Qi-Ling C, Lock K, Hijazi ZM, Amplatzer Muscular Ventricular Septal Defect Investigators (2004) Device closure of

muscular ventricular septal defects using the Amplat- zer muscular ventricular septal defect occluder. J Am Coll Car- diol 43:1257-1263

- 14. Holzer R, de Giovanni J, Walsh KP et al (2006) Transcatheter closure of perimem-branous ventricular septal defects using the amplatzer membranous VSD occluder: immediate and midterm results of an international registry. Catheter Cardiovasc Interv 68:620-628
- 15. Janorkar S, Goh T, Wilkinson J (1999) Transcatheter closure of ventricular septal defects using the Rashkind device: initial experience. Catheter Cardiovasc Interv 1:43-48
- 16. Kalra GS, Verma PK, Dhall A et al (1999) Transcatheter device closure of ventricular septal defects: Immediate results and intermediateterm follow-up. Am Heart J 2(Pt 1):339-344
- 17. Kidd L, Discroll DJ, Gersony WH et al (1993) Second natural history study of congenital heart defects: results of treatment of patients with ventricular septal defects. Circulation 87(Suppl I): 138-151
- Kirklin JW, Barrat-Boyes BG (1993) Ventricular septal defect. In: Kouchoukos NT, Blacksgowe EH, Doty DB, Hawley FL, Karp RB (eds) Cardiac surgery, 3rd edn. Churchill Livingstone, New York, pp 850-910
- 19. Koneti NR, Sreeram N, Penumatsa RR, Arramraj SK, Karunakar V, Trieschmann U (2012) Transcatheter retrograde closure of perimembranous ventricular septal defects in children with the Amplatzer duct occluder II device. J Am Coll Cardiol. 60(23):2421-2422.

doi:10.1016/j.jacc.2012.08.1004 Epub 2012 Nov 7

- 20. Latiff HA, Alwi M, Kandhavel G et al (1999) Transcatheter closure of multiple muscular ventricular septal defects using Gianturco coils. Ann Thorac Surg 4:1400-1401
- 21. Lock JE, Block PC, McKay RG et al (1988) Transcatheter closure of ventricular septal defects. Circulation 2:361-368
- Mavroudis C, Backer CL, Jacobs JP (2003) Ventricular septal defect. Pediatric cardiac surgery, 3rd edn. Mosby, Philadelphia, pp 298-320
- 23. Pass RH, Hijazi Z, Hsu DT, Lewis V, Hellenbrand WE (2004) Multi-

center USA Amplatzer patent ductus arteriosus occlusion device trial. J Am Coll Cardiol 44:513-519

No. 2

- 24. Pedra AC, Pedra SRF, Esteves CA et al (2004) Percutaneous closure of peri-membranous ventricular septal defects with the Amplatzer device: technical and morphological considerations. Cathet Cardiovasc Interv 61:403-410
- 25. Predescu D, Chaturvedi RR, Friedberg MK, Benson LN, Ozawa A, Lee KJ (2008) Complete heart block associated with device closure of perimembranous ventricular septal defects. J Thorac Cardiovasc Surg 136:1223-1228

الملخص العربى

يمثل عيب الحاجز البطيني حوالي 20% من عيوب القلب الخلقية، حوالي 70% منهم يقع في المنطقة المحيطة بالحاجز الغشائي و 15% في الحاجز العضلي كما أن مؤشرات غلق العيب تشتمل علي تضخم غرف القلب في الجانب الأيسر، علامات فشل القلب وكذلك حدوث التهاب الشغاف المعدية.

- **الهدف من الرسالة:** 1- تقويم كفاءة وملائمة وكذلك سلامة غلق عيب الحاجز البطينى عن طريق استخدام أجهزه متعددة عن طريق القسطرة القلبية.
 - 2- متابعة الحالات التي تم غلقها لتحديد المضاعفات ونتائج الغلق.
- 3- مقارنة بين الأنواع المختلفة لتخطيط صدي القلب سواء ثنائي الأبعاد عبر الصدر، ثنائي الأبعاد عبر المرئ وكذلك عبر تصوير الأوعية خلال القسطرة القلبية في تقويم عيب الحاجز بين البطينين ذو الأهمية الديناميكية وتحديد اذا ما كان ذلك يؤثر علي نوع التدخل والعلاج.

طرق البحث

اشتملت الدراسة على 30 حالة بها عيب في الحاجز بين البطينين محولة الي وحدة قلب الأطفال بقسم الاطفال بمستشفي الاطفال التخصصي جامعة القاهرة.

النتائج:

استغرقت الدراسة سنتين في الفترة من أول نوفمبر 2013 حتى نهاية نوفمبر 2015 واشتملت الدراسة علي 30 حالة، 17 من الذكور و13 من الإناث وتتراوح أعمار هم من سنة الي 14 سنة، وأوزانهم بين 6 إلي 45 كيلو جرام وقد أجريت القسطرة القلبية لكل الحالات لغلق العيب عن طريق الجهاز المخصص لغلق العيب وقد نجح الغلق في 25 حالة بنسبة 83,3% وكانت أكثر الأعراض حدوثا هي عدوي الصدر المتكررة في 25 حالة بنسبة 83,3% وأقلها حدوثا هي التهاب الشغاف المعدية في حاله واحده بنسبة 3,3%.

وقد أظهرت الدراسة نجاح غلق عيب الحاجز البطيني عن طريق القسطرة القلبية بنسبه مشجعة وعدم حدوث مضاعفات كبري خلال متابعه المرضى لمدة عام كامل بعد الغلق.

الاستنتاجات والتوصيات:

غلق عيب الحاجز البطيني عن طريق القسطرة القلبيه طريقه فاعلة وآمنة ولها مميزات عديدة مقارنه بالتدخل الجراحي.

رجوع قياسيات عضله غرفة القلب اليسري وكذلك وظائف القلب إلى المعدل الطبيعي في جميع الحالات التي تم غلقها بنجاح.

الاختيار الدقيق للمرضى المناسبين لغلق عيب الحاجز البطيني يقلل من حدوث المضاعفات وبخاصة المرتبطة بكهربية القلب.