International Journal of Cardiology Sciences

ISSN Print: 2664-9020
ISSN Online: 2664-9039
Impact Factor: RJIF 5.42
IJCS 2024; 6(1): 15-20
www.cardiologyjournals.net
Received: 16-11-2023
Accepted: 23-12-2023

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Assessment of right heart size in children after percutaneous closure of secundum atrial septal defect with amplatzer septal occluder

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DOI: https://doi.org/10.33545/26649020.2024.v6.i1a.44

Abstract
Introduction: Any opening between the two atria other than a competent foramen ovale is an interatrial defect, which accounts for 6%–10% of all CHD in children, with a 2:1 female preponderance. Transcatheter secundum ASD closure is adopted worldwide.
Aim of the study: To measure right atrial (RA) and right ventricular (RV) size and quick remodeling to normal in children after percutaneous secundum atrial septal defect repair using the Amplatzer septal occluder.
Methods: The research included 70 children aged 4-14.5 years (7.61±2.5 years). Using 2D TTE, longitudinal, transverse axis, area of RA, RV inflow dimensions at basal one-third and midway between the tricuspid annulus and the apex, short axis, and M-mode RV at end diastolic dimensions were measured (indexed to body surface area). Measurements were taken before, 24 h, 1 month, and 3 months after the procedure and compared to the control group.
Results: A substantial difference (p<0.05) was seen in all measures between patients and controls. A substantial positive link between RAD1, RAD2, RAA, RVD1, RVD2, RVD3, RVD4, and RVD5 with time. Right ventricular transverse dimension (RVD1 and RVD2), RA transverse axis (RAD2), and longitudinal axis dimensions (RAD1 and RVD3) normalized after 3 months, although RAA, RV short axis, and M-mode RV end diastolic dimension decreased slowly. The transverse-to-longitudinal RA axis ratio increased considerably after 3 months of ASDII**.**
Conclusions: Right atrial and right ventricular measures drop fast in the first 24 hours and usually normalise within 3 months. M-mode and short axis RV end diastolic and RAA end systolic dimensions don't show RV alterations. The greater transverse to longitudinal RA axis ratio indicates that Amplatzer septal occluder closure of ASD affects RA geometry.

Keywords: Heart, size, children, percutaneous closure, Secundum, ASD, septal occluder

Introduction
Interatrial septal defects (ASDs) are among the most prevalent congenital heart diseases, representing about 6% to 10% of congenital cardiac anomalies in children, and display a female predominance at a 2:1 ratio [1-2]. These defects involve openings between the atria, excluding a normal foramen ovale. Historically, the patent foramen ovale was first described by Leonardo Da Vinci in 1531, marking the earliest recognition of congenital heart disease [3]. Karl von Rokitansky later provided detailed anatomical descriptions of ASDs in 1875, followed by Ashman's identification of their clinical and radiological features in 1921. The first clinical diagnosis of an ASD was achieved by Bedford and colleagues in 1941 [4]. The development of the interatrial septum is crucial for fetal survival, starting with the formation of the ostium primum early in gestation. The process involves the septum primum and septum secundum, culminating in the creation of the fossa ovalis. Most secundum ASDs are attributed to a deficiency in the septum primum, and these defects are larger and located at the fossa ovalis, distinguishing them from the patent foramen ovale (PFO) [5]. ASDs are classified into four types based on morphologic and physiological features: septum primum defect, septum secundum defect (Which is the most common, comprising 74% of ASDs), sinus venosus, and coronary sinus defects [6]. Ostium primum ASDs, representing 20% of ASDs, are considered mild forms of endocardial cushion defects and are frequently associated with Down syndrome.

In contrast, ostium secundum ASDs result from atrial septal tissue deficiency at the fossa ovalis area and are predominant among ASDs. Sinus venosus ASDs, accounting for about 5% of cases, involve abnormal connections between the right pulmonary veins and the superior or inferior vena cava. The rarest type, coronary sinus ASDs, emerge from the unroofing of the coronary sinus into the left atrium, constituting less than 1% of cases [7]. The secundum type of ASD is the most prevalent form of ASD in adults, with a reported incidence of about 75% of all ASD cases and a prevalence of around 2 per 1,000 live births [8]. The etiology of ASD includes both sporadic occurrences and familial inheritance patterns, with mutations in several genes implicated in its pathogenesis [9]. Patho-physiologically, ASDs affect cardiac hemodynamics based on the size of the defect and the compliance of the ventricles, significantly influenced by postnatal changes in pulmonary blood flow and pressure [10]. Small ASDs might remain asymptomatic and could potentially close spontaneously, while large defects might cause symptoms such as shortness of breath, exercise intolerance, and arrhythmias in adulthood due to the increased shunt volume [11]. Diagnosis and management strategies for ASDs include physical examinations, chest X-rays, electrocardiography, and echocardiography to assess the defect's size and impact. Advanced imaging techniques like CT and MRI may be employed for detailed anatomical assessments, especially in complex cases or when echocardiographic findings are inconclusive [12]. Transcatheter device closure has become a standard treatment for secundum ASDs, with specific indications based on the defect's hemodynamic significance and anatomic suitability [13]. The aim of study is to assess the dimensions of the right atrium and ventricle prior to and subsequent to percutaneous closure of ASDII using the Amplatzer septal occlude and to assess the rate at which right atrial and ventricular remodelling returns to normal size following ASDII closure at 24 hours, one month, and three months, in comparison to the control group.

Methods

A prospective cohort study was conducted at Ibn Al-Bitar Cardiac Center in Baghdad, Iraq, from October 2018 to February 2020, to evaluate the outcomes of percutaneous closure of secundum atrial septal defects (ASDII) in children. The study enrolled 70 children diagnosed with secundum ASD, comprising 34 males and 36 females who visited the outpatient clinic for Congenital and Structural Heart Diseases. These patients underwent clinical evaluation and transthoracic echocardiography (TTE) before receiving percutaneous closure with an Amplatzer Septal Occluder (ASO). Demographic data (age, sex), anthropometric measures (height, weight, body surface area [BSA]), clinical status, and echocardiographic parameters were collected for each patient. Prior to the procedure, parents and patients were informed about the transcatheter closure, and preoperative preparations included blood tests (complete blood count, renal function tests, viral screening), arranging one pint of whole blood per patient, and prescribing oral aspirin at a dosage of 3-5mg/kg/day starting 24 hours before catheterization. Inclusion criteria specified patients with secundum ASD featuring sufficient rims (≥ 5 mm) except for the aortic rim, a total interatrial septum length greater than the device's left atrial (LA) disk, a significant left-to-right shunt (Qp/Qs ratio ≥ 1.5:1), and evidence of right ventricular volume overload. Exclusion criteria ruled out patients with multiple deficient rims, mild or significant residual atrial shunt, association with other congenital heart diseases (CHD), chronic diseases, clinical instability or abnormal heart rhythms, antiplatelet contraindications, irreversible pulmonary vascular disease, or a history of nitinol allergy. The patient cohort ranged in age from 4 to 14.5 years, with heights between 96 and 165 cm, weights from 12 to 65 kg, and BSA from 0.58 to 1.52 m². Echocardiographic diagnosis and measurements of ASDII used a Vivid E9 with XDclear system, with defect sizes ranging from 6 to 30 mm and Qp/Qs ratios from 1.43 to 2.89. Echocardiography, performed by the same investigator, included assessments of right atrial (RA) parameters and right ventricular (RV) dimensions using specific views and techniques, as well as the pulmonary to systemic blood flow ratio (QP/QS). A control group of 30 healthy children, matched by gender, age, weight, height, and BSA to the study group, was also evaluated. They had structurally normal hearts with an assumed Qp:Qs ratio of 1. Statistical analyses were conducted using IBM SPSS Statistics 23, with significance levels set at $p< 0.05$, $p< 0.01$, and $p< 0.001$ for statistical, highly significant, and extremely significant differences, respectively.

Results

The study group included 73 children aged 4-14.5 years (mean± SD: 7.61±2.5 years), heights 96-165 cm (mean±SD: 125.15±15.63 cm), weights 12-65 kg (mean±SD: 24.3±10.01 kg), and BSAs 0.58-1.52 m2. TTE readings for ASDII varied from 6 to 30 mm (mean±SD: 12.99±5.01 mm), IAS size from 24 to 44 mm (mean±SD: 33.62±4.2 mm), and Qp/Qs from 1.43 to 2.89 (mean±SD: 1.87±0.37). All patients utilised one ASO with diameters from 10 to 36 mm (mean±SD: 17.29±5.62). One case was excluded due to significant mild to moderate residual shunt and two patients due to concomitant significant valvular pulmonary stenosis, but 70 children (34 males and 36 females) were instantly closed with no residual shunt. There were no severe issues during and after the treatment. The research group was matched for gender, age, body weight, height, and body surface area with 30 healthy youngsters (15 males and 15 girls). Ages: 4.5-14.5 years (mean±SD: 8.59±2.75 years), heights: 102.5-162 cm, weights: 16.5-51.5 kg, BSA: 0.68-1.52 m2, IAS size: 22-41 mm (mean±SD: 29.6±9.36 mm). ASD II size, ASO size, IAS size, Qp, Qs, Qp/Qs obtained before the procedure were significantly different between the total study group and total control, as shown in table (1) and between female patients and female control, as shown in table (2). Table (3) shows a significant difference in all parameters between male patient and male control, but table (4) shows no significant difference between male patient and female patients before the treatment.

Table 1: Comparison of demographic and atrial septal defects indexes between total patients and total control groups before ASDII closure.

Total Number			
Parameter	**Patient (n=70) Mean±SD**	**Control (n=30) Mean±SD**	**Sig. Value (P-value)**
Age (years)	7.61±2.5	8.59±2.75	0.182
Height (cm)	125.15±15.63	128.08±16.77	0.127
Weight (Kg)	24.3±10.01	28.6±9.36	0.467
BSA(M^2)	0.91±0.23	0.99±0.23	0.342
ASD II Size (mm)	12.99±5.01	0.00±0.00	0.0001*
IAS Size (mm)	33.62±4.2	29.6±4.7	0.0001*
ASO Size	17.29±5.62	0.00±0.00	0.0001*
Qp	6377.6±2343.97	1.00±0.00	0.0001*
Qs	3456.9±1153.31	1.00±0.00	0.0001*
Qp/Qs	1.87±0.37	1.00±0.00	0.0007*

*p< 0.05 considered significant. BSA: Body surface area, ASDII: Secundum atrial septal defect, ASO: Atrial septal occluder, Qs: Systemic blood flow, Qp: Pulmonary blood flow, IAS: Interatrial septum.

Table 2: Comparison of demographic and atrial septal defects indexes between female patients and female control groups before ASDII closure.

Female			
Parameter	**Patient (n=36) Mean±SD**	**Control (n=15) Mean±SD**	**Sig. Value (P-value)**
Age (years)	7.90±2.56	7.17±2.65	0.3628
Height (cm)	126.90±17.34	120.63±17.16	0.2437
Weight (Kg)	24.62±10.1	24.37±8.64	0.9335
BSA(M^2)	0.92±0.24	0.89±0.22	0.6790
ASD II Size (mm)	12.94±4.96	0.00±0.00	0.0001*
IAS Size (mm)	33.86±3.93	28.20±4.41	0.0001*
ASO Size	17.05±5.27	0.00±0.00	0.0001*
Qp	6169.1±2351.11	1.00±0.00	0.0001*
Qs	3522.9±1217.75	1.00±0.00	0.0001*
Qp/Qs	1.74±0.29	1.00±0.00	0.0007*

* *P*< 0.05 considered significant. BSA: Body surface area, ASDII: Secundum atrial septal defect, ASO: Atrial septal occluder, Qs: Systemic blood flow, Qp: Pulmonary blood flow, IAS: Interatrial septum.

Table 3: Comparison of demographic and atrial septal defects indexes between male patients and male control groups before ASDII closure.

Male			
Parameter	**Patient (n=34) Mean±SD**	**Control (n=15) Mean±SD**	**Sig. Value (P-value)**
Age (years)	7.32±2.44	10.00±2.84	0.0006*
Height (cm)	123.4±13.91	135.53±16.37	0.0105*
Weight (Kg)	23.98±9.92	32.83±10.08	0.0062*
BSA(M^2)	0.89±0.22	1.10±0.24	0.0044*
ASD II Size (mm)	13.05±5.04	0.00±0.00	0.0001*
IAS Size (mm)	33.38±4.47	31.0±4.99	0.0001*
ASO Size	17.53±5.97	0.00±0.00	0.0001*
Qp	6598.42±2351.05	±0.00 1.00	0.0001*
Qs	3386.96±1094.8	±0.00 1.00	0.0001*
Qp/Qs	1.96±0.44	±0.00 1.00	0.0007*

* *P*< 0.05 considered significant. BSA: Body surface area, ASDII: Secundum atrial septal defect, ASO: Atrial septal occluder, Qs: Systemic blood flow, Qp: Pulmonary blood flow, IAS: Interatrial septum.

Table 4: Comparison of demographic and atrial septal defects indexes between male patients and female patient's groups before ASDII closure.

Patients			
Parameter	**Patient Male (n=34) Mean±SD**	**Patient Female (n=36) Mean±SD**	**Sig. Value (P-value)**
Age (years)	7.32±2.44	7.90±2.56	0.3359
Height (cm)	123.4±13.91	126.90±17.34	0.3566
Weight (Kg)	23.98±9.92	24.62±10.1	0.7901
BSA(M^2)	0.89±0.22	0.92±0.24	0.5881
ASD II Size (mm)	12.7±5.71	13.60±4.40	0.4612
IAS Size (mm)	33.38±4.47	33.86±3.93	0.635
ASO Size	17.53±5.97	17.05±5.27	0.546
Qp	6598.42±2351.05	6169.1±2351.11	0.3020
Qs	3386.96±1094.8	3522.9±1217.75	0.6851
Qp/Qs	1.96±0.44	1.74±0.29	0.1220

* *p*< 0.05 considered significant. BSA: Body surface area, ASDII: Secundum atrial septal defect, ASO: Atrial septal occluder, Qs: Systemic blood flow, Qp: Pulmonary blood flow, IAS: Interatrial septum.

The mean RA indexed measurements before and after percutaneous defect closure and statistical analysis. Prior to the surgery, the study group had considerably greater RA readings than the control group ($p< 0.0001$). 24 hours after defect closure, the mean values of all evaluated parameters reduced considerably compared to pre-procedure levels; 1 month later, only RAD1 achieved control group normalised size. At 3 months, mean values of RAD2, RAA, and even normalised size RAD1 decreased dramatically, with RAD2 reaching control group size but RAA staying significantly higher than controls (table 5).

Table 5: The mean values of right atrial measurements between study and control groups before and after 24h, 1month and 3 months of ASDII closure.

Parameter		Numbers	Mean	Std. Deviation	Std. Error Mean	Sig.
RAD1/ before	Patient	70	37.7571	5.97443	0.71408	0.000*
	Control	30	31.7800	4.82882	0.88162	
RAD1/ 24 h	Patient	70	36.0571	5.80825	0.69422	0.001*
	Control	30	31.7800	4.82882	0.88162	
RAD1/ 1mo.	Patient	70	33.5429	5.59939	0.66926	0.137
	Control	30	31.7800	4.82882	0.88162	
RAD1/ 3mo.	Patient	70	31.4714	5.29895	0.63335	0.785
	Control	30	31.7800	4.82882	0.88162	
RAD2/ before	Patient	70	33.3571	6.08166	0.72690	0.000*
	Control	30	27.8067	4.63353	0.84596	
RAD2/ 24 h	Patient	70	31.7000	5.84919	0.69911	0.002*
	Control	30	27.8067	4.63353	0.84596	
RAD2/ 1m	Patient	70	30.3429	5.68212	0.67914	0.034*
	Control	30	27.8067	4.63353	0.84596	
RAD2/ 3m	Patient	70	29.3571	5.29160	0.63247	0.167
	Control	30	27.8067	4.63353	0.84596	
RAA/before	Patient	70	13.7671	3.31548	0.39628	0.000*
	Control	30	8.0723	1.73787	0.31729	
RAA/24 h	Patient	70	11.3857	3.27791	0.39179	0.000*
	Control	30	8.0723	1.73787	0.31729	
RAA/1 mo.	Patient	70	10.0400	3.21696	0.38450	0.002*
	Control	30	8.0723	1.73787	0.31729	
RAA/3 mo.	Patient	70	9.3800	2.98604	.3569 0	0.028*
	Control	30	8.0723	1.73787	.31729 0	

*$p< 0.05$ considered significant, RA: right atrium, RAD1: longitudinal right atrial dimension, RAD2: transversal right atrial dimension and RAA: right atrial area.

For the study group after 24h, 1 month, and 3 months, there were no differences in the mean RAD2/RAD1 ratio values before and after the procedure, although these values remained higher than in controls. Table (11) shows a significant change ($p< 0.05$) in the mean RAD2/RAD1 ratio in the study group after 3 months of the operation.

Table 6: Right atrial transverse (RAD2) to longitudinal axis (RAD1) ratio in children before and after secundum atrial septal defect closure and in control groups.

Ratio	Subject	No.	Mean	Std. Deviation	Std. Error Mean	Sig.
RAD2 / RAD1 Before	Patient	70	0.8906	0.14138	0.01690	0.528
	Control	30	0.8740	0.03650	0.00666	
RAD2 / RAD1 after 24 hrs.	Patient	70	0.8864	0.14170	0.01694	0.637
	Control	30	0.8740	0.03650	0.00666	
RAD2 / RAD1 after 1 month	Patient	70	0.9137	0.15285	0.01827	0.164
	Control	30	0.8740	0.03650	0.00666	
RAD2 / RAD1 after 3months	Patient	70	0.9441	0.16368	0.01956	0.023*
	Control	30	0.8740	0.03650	0.00666	

*$p< 0.05$ considered significant, RA: right atrium, RAD1: longitudinal right atrial dimension, RAD2: transversal right atrial dimension.

Before percutaneous closure of ASDII, all RV indexed measurements (RVD1, RVD2, RVD3, RVD4 and RVD5) in the patients group were significantly higher than in the control groups. The mean values of all RV indexed parameters after 24h, 1month and 3 months decreased significantly as compared to pre-procedure values, and RVD1, RVD2 and RVD3 were reached normalized size of control group after 3 months of closure, while both RVD4 and RVD5 significantly higher than control as shown in table (7).

Table 7: The mean values of right ventricular measurements between study and control groups before and after 24h, 1month and 3 months of ASDII closure.

Parameter		Numbers	Mean	Std. Deviation	Sig.
RVD1/ before	Patient	70	32.5857	3.74290	0.000**
	Control	30	25.7667	2.61538	
RVD1 / 24 h	Patient	70	29.9571	3.37693	0.001*
	Control	30	25.7667	2.61538	
RVD1 / 1 month	Patient	70	27.3000	3.11496	0.032*
	Control	30	25.7667	2.61538	
RVD1 / 3 months	Patient	70	25.4429	2.98220	0.058
	Control	30	25.7667	2.61538	
RVD2 / before	Patient	70	29.6857	3.33256	0.000**
	Control	30	20.9333	2.20397	
RVD2 / 24 h	Patient	70	26.5143	3.05664	0.001*
	Control	30	20.9333	2.20397	
RVD2 / 1 month	Patient	70	24.0429	2.85774	0.014*
	Control	30	20.9333	2.20397	
RVD2 / 3 months	Patient	70	21.7857	2.57819	0.061
	Control	30	20.9333	2.20397	
RVD3 / before	Patient	70	45.9857	5.97046	0.000**
	Control	30	32.6767	4.85462	
RVD3 / 24 h	Patient	70	42.5857	5.79978	0.001*
	Control	30	32.6767	4.85462	
RVD3 / 1 month	Patient	70	38.4857	5.35876	0.029*
	Control	30	32.6767	4.85462	
RVD3 / 3 months	Patient	70	33.9571	5.03322	0.068
	Control	30	32.6767	4.85462	
RVD4/ before	Patient	70	24.2429	3.38237	0.000**
	Control	30	19.2333	2.31462	
RVD4 / 24 h	Patient	70	23.5143	3.13247	0.001*
	Control	30	19.2333	2.31462	
RVD4 / 1 month	Patient	70	22.5571	2.90359	0.006*
	Control	30	19.2333	2.31462	
RVD4 / 3 months	Patient	70	21.0500	2.73922	0.022*
	Control	30	19.2333	2.31462	
RVD5/ before	Patient	70	23.0286	3.27774	0.000**
	Control	30	17.4667	2.11316	
RVD5 / 24 h	Patient	70	22.4143	3.00871	0.0001*
	Control	30	17.4667	2.11316	
RVD5 / 1 month	Patient	70	20.5143	2.87981	0.008*
	Control	30	17.4667	2.11316	
RVD5 / 3 months	Patient	70	19.9857	2.61091	0.005*
	Control	30	17.4667	2.11316	

*$p< 0.05$ considered significant, RVD1: basal diameter, RVD2: Transverse halfway between the tricuspid annulus and the apex, RVD3: longitudinal RV dimension, RVD4: transverse dimension assessed in the parasternal short axis view at the level of the mitral valve, and RVD5: in M-mode echocardiography from the parasternal long axis view at the level of the mitral valve chordae tendineae.

Discussions

This study, conducted at Ibn Al-Bitar Cardiac Center, Baghdad, aimed to evaluate the efficacy of transcatheter

closure of secundum atrial septal defects (ASDII) in a cohort of 70 children (34 males and 36 females). The transcatheter approach, utilizing the Amplatzer Septal Occluder (ASO), is celebrated for its safety, minimization of clinical sequelae, reduced hospital stays, quicker recovery, and favorable long-term outcomes compared to surgical closure, which involves cardiopulmonary bypass, thoracotomy, and leaves a surgical scar [14]. Successful outcomes hinge on careful patient selection and the proficiency of the operator. The study revealed complete closure of ASD in all cases, with no residual shunts or trivial shunts disappearing within 24 hours post-procedure. This aligns with findings from Omeish A *et al*., highlighting the procedure's effectiveness [14]. Hemodynamic assessments confirmed significant left-to-right interatrial shunting, with the Qp/Qs ratio ranging from 1.43 to 2.89 (mean± SD: 1.87±0.37), indicating the presence of significant shunting necessitating closure. Statistical analysis showed positive correlations between patient age, height, weight, BSA, and the size of ASDII, as well as the Qp/Qs ratio, underscoring the impact of patient characteristics on ASD dimensions and hemodynamic effects [15]. Notably, there was no significant correlation between sex and the size of the interatrial septum. Echocardiographic parameters assessed included right ventricular (RV) end-diastolic diameters, right atrial (RA) end-systolic diameters, and the transverse to longitudinal RA axis ratio at end-systole, serving as indicators of cardiac geometry changes post-closure. Significant reductions in these parameters were observed at 24 hours, 1 month, and 3 months' post-procedure, consistent with the literature on the topic [16-17]. These changes suggest effective reversal of the cardiac overload and geometric alterations caused by the ASD. Further analysis showed a rapid decrease in RA longitudinal axis and a more gradual reduction in the transverse axis, leading to changes in the RAD2/RAD1 ratio, indicative of alterations in RA geometry post-ASD closure [18]. Despite the normalization of several dimensions within 3 months, the right atrial surface area (RAA) remained significantly larger compared to controls, suggesting a slower normalization process for RAA, particularly in older patients [19]. RV dimensions were meticulously evaluated, showing all indexed RV measurements were significantly higher than those of the control group pre-procedure. Following ASD closure, there was a significant decrease in these dimensions, with RVD1, RVD2, and RVD3 reaching the normalized size of the control group within 3 months. However, RVD4 and RVD5 remained elevated, indicating persistent alterations in RV geometry [20-21].

Conclusion

Transcatheter closure of secundum atrial septal defects (ASDII) is a preferred alternative to surgery in pediatric patients, offering significant benefits including rapid normalization of right atrial and ventricular dimensions within three months, indicative of favorable cardiac remodeling. This remodeling extends beyond the immediate correction of paradoxical septal motion, as evidenced by the significant decrease in right atrial areas, reflecting the positive impact of correcting the left-to-right shunt. However, traditional M-mode and short axis measurements fall short of accurately depicting these changes, with the Amplatzer Septal Occluder (ASO) notably influencing right atrial geometry and potentially altering the RA axis ratios due to septal stiffening.

Conflict of Interest
Not available

Financial Support
Not available

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How to Cite This Article
Kareem HM, Al-Enbari A. Assessment of right heart size in children after percutaneous closure of secundum atrial septal defect with amplatzer septal occluder. International Journal of Cardiology Sciences. 2024; 6(1): xx-xx