Evaluating the Impact of Transcatheter Closure of Secundum Atrial Septal Defect on Ventricular Function Using Different Echo-Doppler Modalities Haidy Badei Yosef Mohamad¹, Taher Saeed Abdelkareem²,

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ABSTRACT

Background: Atrial septal defect (ASD) closure has become an established therapy. Novel echocardiographic methods have been developed to quantify global as well as regional ventricular function and are used for diagnostic and prognostic evaluation of various cardiovascular diseases.

Objective: To assess the impact of transcatheter closure of secundum atrial septal defects on the left and right ventricular function using conventional and newer echo-Doppler modalities.

Patients and methods: This study included 40 subjects with secundum ASD. Cases were assessed by transesophageal echo pre ASD closure to detect suitability for transcatheter ASD closure, then selected patients were evaluated by transthoracic echo-Doppler using conventional and recent modalities before closure, one month and three months after closure.

Results: Post ASD closure there were increase in the Left ventricle (LV) dimensions and volumes, increase in LVEF, and reduction of LV-GLS compared to pre closure. The RV volumes, RV-GLS and RVEF were decreased post closure. Both LV Tei index and RV Tei index showed significant reduction post ASD closure.

Conclusion: Transcatheter closure of secundum atrial septal defect leads to structural and functional changes in both ventricles in the form of: (a) reduction in RV dimensions and volumes with apparent deterioration of the RV systolic function, (b) increase in dimensions and volumes of the LV with apparent improvement of LV systolic function, and (c) apparent improvement of both left and right ventricular diastolic function post ASD closure.

Keywords: Transcatheter closure, Secundum atrial septal defect, Left and right ventricular function, New echo-Doppler modalities.

INTRODUCTION

Atrial septal defect (ASD) is one of the most frequent (5%–10%) congenital heart diseases (CHD) in adults^[1]. Anomalous pulmonary venous connection, persistent left superior vena cava (SVC), pulmonary valve stenosis, and mitral valve prolapse are most lesions that are in common association with secundum ASD. Holt–Oram syndrome may be also an association with ASD ^[2].

Ostium secundum ASD is the most common form of ASD and most often occurs as the result of a true deficiency of septum primum tissue^[3]. Ostium secundum defects represent 80% of ASDs^[2]. ASD flow is predominantly left to right, but with isometric strain transient right-to-left shunts are occuring. During diastole the bulk of the shunt flow has been occuring^[4]. The shunt volume depends on RV/LV compliance, defect size, and LA/RA pressure^[2].

ASD causes right ventricular (RV) dilatation and RV volume overload, the size of the left ventricle is affected by increased RV size and it causes a significant change in LV shape and geometry, LV becomes smaller in size, with a reduction in LV strock volume (SV) and impaired LV diastolic filling. Inter- and intraventricular dyssynchrony are caused by RV dilatation and volume overload, which impairs LV performance and distensibility^[5].

The primary diagnostic method to assess ASD is transthoracic echocardiography (TTE) including location, size and ASD hemodynamics. Right ventricular dilatation is the most echocardiographic finding associated with a left-to-right shunt^[6]. Twodimensional transthoracic echocardiography (2D TTE), 2D transesophageal echocardiography (2D TEE), and three-dimensional (3D) TEE are required for interatrial septum (IAS) assessment^[7].

Shunt lesions are needing accurate assessment and (3D) echocardiography is used for the diagnosis, management and follow-up of these patients^[8], as the Teichholz and Quinones methods for calculating LV volumes from LV linear dimensions are no longer recommended for clinical use, so LV volumes now are measured using 2DE or 3DE^[9]. ASD closure causes ameliorating symptoms and improving functional capacity^[3].

The presence of right heart dilatation caused by significant left-right shunt is indicating ASD closure regardless of symptoms, while no indication for ASD closure with small defects unless there is evidence of right-left shunting causing significant hypoxaemia and/ or paradoxical embolism^[10].

Nowadays, the first choice for secundum defect closure is the device closure for most patients when feasible from morphology (includes stretched diameter = 38 mm and sufficient rim of 5 mm except towards the aorta)^[11]. Since 2001, Amplatzer septal occluder (ASO) device is FDA approved for ASD closure and has the widest usage^[12]. Device erosion is the most serious complication would be, which is associated with oversizing the device or rim deficiency^[13].

The aim of this study was to assess the impact of transcatheter closure of secundum atrial septal defects on the left and right ventricular function using conventional and newer echo-Doppler modalities.

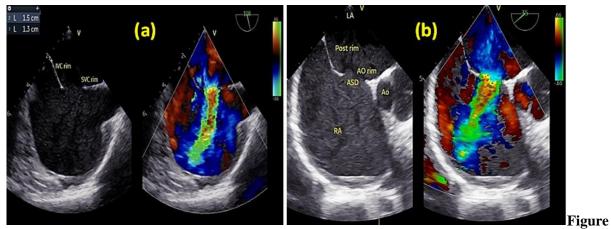
PATIENTS AND METHODS Study design

Forty patients with ASD after their informed consent were subjected to thorough medical history and physical examination with demographic data including age and sex; and the dominant most annoying symptoms to the patient with special emphasis on palpitation and dyspnea, during the period from October 2021 till May 2023.

Interatrial communication other than secundum ASD, associated other congenital heart diseases, significant valvular heart disease, previous surgical or percutaneous cardiac interventions, patients with rhythm other than sinus rhythm, documented IHD, other systemic diseases that may affect ventricular function, and poor echocardiographic window were excluded. Patients were assessed by ECG, transesophygeal echocardiography (TEE) and transthoracic echocardiography (TTE).

Transesophageal echocardiography (TEE) was performed before ASD closure at multiple levels with different angles to assess ASD morphology and its suitability for closure under TEE guidance (**Figure 1**).

Transthoracic echo (TTE) was performed before ASD closure in the standard views in both supine and left lateral position using GE vingmed, ultrasound AS, Horton, Norway (N95) equipped with TDI and STE technology, using a multi frequency M5Sc-D (1.4-4.6 MHz) phased-array probe with simultaneous display of ECG physio-signal. All acquisitions were recorded and digitally stored as echo images and loops for subsequent off-line analysis using EchoPAC, soft ware version 2010.



1: 2D TEE mid esophageal bicaval (a), and short axis views (b), guided by color flow Doppler showing ASD rims. Ao: aorta, ASD: atrial septal defect, IVC: inferior vena cava, RA: right atrium, SVC: superior vena cava.

Transcatheter closure of ASD was performed to all cases elegible for this procedure then transthoracic echo-Doppler examination was repeated one and three months after closure with assessment of the same prameters measured pre-closure.

Assessment of both left and right ventricular function were taken from standard views (including RV-focused apical 4-Ch view), as recommended by the update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging for Cardiac Chamber Quantification by Echocardiography in Adults^[9].

Measured LV echo-Doppler parameters included LV end-diastolic and end-systolic dimensions (LVEDD and LVESD respectively), left ventricular percent fractional shortening (PFS) and M-mode measured LV ejection fraction (EF "m-mode"), mitral annular systolic velocity (M ann-Sa), LV global longitudinal strain (LV-GLS), 4-D LV end-diastolic volume (4D-LVEDV), 4D LV end-systolic volume (4D LV-ESV), 4D LV stroke volume (4D-LVSV), and 4D LV ejection fraction (4D- LVEF), transmitral early diastolic velocity (MV-E vel), mitral annular early diastolic velocity (M ann-Ea), ratio of to transmitral early diastolic flow velocity to mitral annular early diastolic velocity, (MV-E/Ea) and LV-Tei index, which reflects both systolic and diastolic LV function (measured as the ratio of the time spent in isovolumetric activity divided by the time spent in ventricular ejection^[14]).

Measured RV echo-Doppler parameters included: Basal, mid and longitudinal right ventricular diameters (**Basal RVD, Mid RVD and Long RVD**) while displaying the largest basal RV diameter and thus avoiding foreshortening, proximal and distal right ventricular outflow tract diameters (**RVOT**_{prox} and **RVOT**_{dist} respectively), tricuspid annular plane systolic excursion (**TAPSE**), RV fractional area change (**RV-FAC**) calculated as RV end diastolic area minus RV end-systolic area divided by RV end diastolic area, RV systolic pressure (**RVSP**) calculated from tricuspid regurgitation velocity applying modified Bernoulli equation^[15] with adding estimated RA pressure according to IVC collapsibility index, tricuspid annular systolic velocity (**T ann-Sa**), RV global longitudinal strain (**RV-GLS**), 4D-RV end-diastolic volume (**4D-RVEDV**), 4D-RV end-systolic volume (**4D-RVESV**), 4D-RV stroke volume (**4D-RVSV**), and 4D-RV ejection fraction (**4D-RVEF**) with normal value set at 45%^[16], transtricuspid early diastolic velocity (**TV-E vel**), tricuspid annular early diastolic velocity (**T ann-Ea**), ratio of to transtricuspid early diastolic flow velocity to tricuspid annular early diastolic velocity (**TV E/Ea**), and **RV-Tei** index that reflects both systolic and diastolic RV function.

Statistical analysis: Numerical variables were expressed as mean \pm SD. <u>ANOVA test</u> was used to compare the corresponding quantitative measures obtained during the 3 echo examinations (Pre-ASD closure, one-month post ASD closure and 3 months post ASD closure). The SPSS 23.0 software (SPSS Inc., Chicago, IL, USA) was used for statistical analysis, and p-values < 0.05 were considered statistically significant.

Ethical Approval:

This study was ethically approved by the Institutional Review Board of the Faculty of Medicine, Benha University. Written informed consent was obtained from all participants. This study was executed according to the code of ethics of the World Medical Association (Declaration of Helsinki) for studies on humans.

RESULTS

The current study included 40 ASD patients, 7 males and 33 females. The mean value (\pm SD) of age of the study cases was 26.27 \pm 11.24 years; 27 patients had dyspnea, 24 patients had palpitation, and 11 patients had dyspnea and palpitation.

There was significant progressive reduction in QRS duration from pre ASD closure to one month then 3 months post ASD closure as shown in **Table (1)**.

Electrocardiogram (ECG) finding	Pre ASD closure	-	Three months post ASD closure	<i>p-value</i> Pre vs. 1 month post ASD closure	vs.	<i>p-value</i> One month vs. 3 months post ASD closure
QRS duration (sec)	0.11±0.02	0.10±0.03	0.09 ± 0.02	<0.001**	<0.001**	<0.001**

Table 1: Comparison of ORS duration between	pre ASD closure, one month and 3 months post closure

Left ventricular(LV) Echo-Doppler data:

Comparison of LV echo-Doppler data bofore ASD closure versus one month and three months post closure and between one month vs 3 months post ASD closure are demonstrated in **Table (2)**.

There were significant increase in values of **LVEDD, PFS,** and **M mode-measured LVEF** in both one month and three months post ASD closure compared to pre ASD closure. Mitral annular systolic velocity (**M ann-Sa**) showed significant decrease in one month post ASD closure compared to pre closure. There was a significant reduction in value of (**LV-GLS**) % in both one month and three months post ASD closure compared to pre closure.

On the other hand, values of (**4D-LVEF**) showed significant increase in both one month and 3-

months post ASD closure compared to pre closure value and significant increase in 3-months versus one month post ASD closure. There was significant increase in values of (**4D-LVESV**, **4D-LVEDV and 4D-LVSV**) in 3 months post closure compared to pre ASD closure.

As regards the diastolic parameters, there was significant decrease in values of **MV-E vel** and **MV E/Ea** in the 3 months post ASD closure compared to pre ASD closure, and in the 3 months compared to one month post ASD closure.

LV-Tei index value significantly decreased in both one month and three months post ASD closure compared to pre ASD closure, and also in three months post ASD closure compared to one month post ASD closure.

Conventional echo- Doppler and tissue Doppler data	Pre ASD closure	One month post ASD closure	Three months post ASD closure	<i>p-value</i> Pre vs. 1 month post ASD closure	<i>p-value</i> Pre vs. 3 months post ASD closure	<i>p-value</i> One month vs. 3 months post ASD closure
LVEDD (mm)	40±7.4	41.5±7.1	44.4±7.5	<0.001**	<0.001**	< 0.001**
LVESD (mm)	25.5±5	25.9±5.2	26.3±5.3	0.07	0.02*	0.13
PFS (%)	35.8±5	37.4±4.9	40.5±5.2	0.001**	< 0.001**	<0.001**
EF (m-mode)	65.8±4.9	67.5±4.9	71±5	<0.001**	< 0.001**	<0.001**
M ann-Sa (cm/sec)	8.6±2.2	7.9±1.4	8.3±1.9	<0.001**	0.31	0.16
LV-GLS %	19.4±1.5	18.8 ± 1.5	18.7 ± 1.2	<0.001**	< 0.001**	0.28
4D-LVEDV (ml)	75 ± 27	77 ± 19	91 ±21	0.29	< 0.001**	<0.001**
4D-LVESV (ml)	32 ±12.5	32.6±8.5	36.6±9.3	0.78	0.008**	<0.001**
4D-LVSV (ml)	43 ±15	45 ± 11.5	55 ± 13	0.09	< 0.001**	<0.001**
4D-LVEF	57 ±3.7	58 ±3.2	60.1±3.9	0.04*	< 0.001**	<0.001**
MV-E vel (cm/sec)	84±14.3	82.2±15.6	76 ±14.7	0.379	0.001**	0.009*
M ann-Ea	11.7±3	11.7±3.3	12±3.4	0.91	0.45	0.55
MV E/Ea	7.5±1.2	7.3±1.2	6.7±1.1	0.08	< 0.001**	< 0.001**
LV- Tei index	0.45 ±0.09	0.43 ±0.09	0.40 ± 0.08	< 0.001**	< 0.001**	< 0.001**

Table 2: Comparison between pre ASD closure, one month and 3 months post closure in respect to LV conventional echo-Doppler, tissue Doppler data and speckle tracking echocardiography (STE):

EF: (ejection fraction), **LVEDD:** (left ventricular end diastolic dimension), **LVESD:** (left ventricular end systolic dimension), **LV-Tie:** (left ventricular Tei index), **4D-LVEF:** (left ventricular ejection fraction by 4 dimensional echo), **4D-LVEV:** (left ventricular stroke volume by 4 dimensional echo), **4D-LVEDV:** (left ventricular end diastolic volume by 4 dimensional echo), **4D-LVESV:** (left ventricular global longitudinal strain), **MV-E vel:** (transmitral peak early diastolic velocity), **M ann-Ea:** (mitral annular early diastolic velocity), **MV E/Ea:** (transmitral early diastolic velocity), **PFS:** (percent fractional shortening), **STE:** (speckle tracking echocardiography) *: *p value* <0.05, **: *p value* < 0.001.

Right ventricular (RV) Echo-Doppler data:

There was significant reduction in values of RV measures (basal RVD, mid RVD, long RVD, RVOT_{prox}, RVOT_{dist}, TAPSE and RVSP) in both one month and 3 months post ASD closure compared to pre ASD closure. However, value of TAPSE was significantly higher in the three months compared to one month post ASD closure.

RV-FAC showed significant reduction in one month post ASD closure compared to pre ASD closure, while in the 3 months post closure it showed a significant increase in value in comparison to 1 month post closure. **T ann-Sa** and **RV-GLS** showed significant reduction in both one month and three months post ASD closure compared to pre ASD closure, however these two values were significantly higher in the three months compared to one month post ASD closure. There was significant reduction in values of the **4D RV** volumes in both one month and 3 months post ASD closure compared to pre ASD closure.

The value of **4D-RVEF** showed significant reduction in one month post ASD closure compared to value of pre ASD closure; with no significant change after three months either compared to pre ASD closure or one month post closure. There was significant decrease in the values of **TV-E vel**, **TV E/Ea** and **RV-Tei** index in both one month and three months post ASD closure compared to pre ASD closure value (**Table 3**).

Table 3: Comparison between pre ASD closure, one month and three months post ASD closure in respect to different echo-Doppler data:

RV echo-Doppler data	Pre ASD closure	One month post ASD closure	Three months post ASD closure	<i>p-value</i> Pre vs. one month post ASD closure	<i>p-value</i> Pre vs. 3 months post ASD closure	<i>p-value</i> One month vs. 3 months post ASD closure
Basal RVD (mm)	46.6 ± 5.8	43.7 ± 5.8	39.5±5.5	<0.001**	<0.001**	<0.001**
Mid RVD (mm)	42.5±5.4	40.3±5.3	35.6±5	<0.001**	<0.001**	<0.001**
Long RVD (mm)	75.1±8.2	73.5±8.2	67.5±8.1	<0.001**	<0.001**	<0.001**
RVOT _{prox} (mm)	36±5.9	33.6±5.7	29.2±5.5	<0.001**	<0.001**	< 0.001**
RVOT dist (mm)	26.5±4.2	25.4±3.6	21.6±3.7	<0.001**	<0.001**	<0.001**
TAPSE (mm)	26.3±4.1	20.2±2.7	21.8±2.7	<0.001**	< 0.001**	< 0.001**
RV-FAC	41.3±6.1	38.7±5	42±4.7	<0.001**	0.468	<0.001**
RVSP (mm/Hg)	46.2±11.9	41.2±10.2	34.1±9.5	<0.001**	<0.001**	<0.001**
T ann-Sa (cm/sec)	13 ±1.5	10.9±1.3	12.3±1.8	<0.001**	0.02*	<0.001**
RV-GLS %	22.4±4.2	19.4±2.8	21.4±2.3	<0.001**	0.04*	<0.001**
4D-RVEDV (ml)	141.4±49	123.3±46	93±32	<0.001**	<0.001**	<0.001**
4D-RVESV (ml)	74.6±30	67±28	49.6±20	<0.001**	<0.001**	< 0.001**
4D-RVSV (ml)	67 ± 22	56 ± 20	43 ± 13	<0.001**	<0.001**	<0.001**
4D-RVEF	47.9 ± 5.7	46 ± 5.1	47.3 ± 5.6	0.01*	0.42	0.09
TV-E vel (cm/sec)	87.7±18.4	72.3±13.9	62.4±12.2	<0.001**	<0.001**	< 0.001**
T ann-Ea	14.5±3.1	13±2.8	13.1±2.9	0.001**	0.007**	0.951
TV E/Ea	5.8±0.96	5.6±1	4.9±1.1	0.02*	<0.001**	< 0.001**
RV-Tei	0.39±0.07	0.38±0.07	0.35±0.07	0.02*	<0.001**	< 0.001**

Basal RVD: (basal right ventricular diameter), **Long RVD:** (longitudinal right ventricular diameter), **Mid RVD:** (mid right ventricular diameter), **RV-FAC:** (right ventricular fractional area change), **RVOT** _{distal}: (distal right ventricular outflow tract), **RVOT** _{proximal}: (proximal right ventricular outflow tract), **RVSP:** (right ventricular systolic pressure), **TV-E vel:** (transtricuspid early diastolic velocity), **RV-Tie:** (right ventricular Tei index), **TAPSE:** (tricuspid annular plane systolic excursion), **TDI:** (tissue Doppler velocity), **T ann-Sa:** (tricuspid annular systolic velocity), **RV-GLS:** (right ventricular global longitudinal strain), **4D-RVED volume:** (right ventricular end diastolic volume by 4 dimensional echo), **4D-RVESV:** (right ventricular end systolic volume by 4 dimensional echo), *: *p value* <0.05, **: *p value* < 0.001.

DISCUSSION

The preferred method for treating ASD in most patients with suitable anatomy is the transcatheter device closure^[17]. In contrast to studies addressed to the effect of ASD closure on RV function, it was noticed that relatively fewer studies have been evaluated the effect of ASD correction on LV function with reported variable effects^[18]. RV volume overload decreases dramatically and LV volume increases after closure of ASD^[19].

In respect to the effect of transcatheter ASD closure on LV systolic function, the current study revealed significant increase in LV dimensions and most parameters of the left ventricle (LVEDD, PFS and M-mode-measured LVEF) one month and 3 months post closure. Also, this study demonstrated an increase in 4D-measured LVEDV, LVESV, LVSV and LVEF in 3 months post ASD closure. These findings are in agreement with multiple previous studies^[19]. Improvement of LV systolic function after closing the ASD is explained on the basis of removing the RV volume overload, and its effect on LV filling, and this would be supported by the phenomenon of ventricular interdependence^[20].

On the other hand, as a contrary to the effect of ASD closure on improvement LV ejection fraction, the current study showed significant decrease in the mean value of **LV-GLS** one month and 3 months post closure in comparison to pre closure value, and a reduction in the **M ann-Sa** in one month after ASD closure. Reduction in the **LV-GLS** in the current study coincides with those reported in the study of **Alkhateeb** *et al.*^[21], in their assessment of ASD patients at baseline, at 24 hours and 1 month after ASD device closure. Likewise another study^[18], reported a decrease in LV-GLS after 3 months post ASD closure in comparison to pre closure.

Also a study done by **Miki** *et al.* in 2021^[19], revealed a decrease in LV-GLS post ASD closure, but with improvement of circumferential direction; they explained that by the leftward shifting or flattening of the interventricular septum preclosure due to RV volume overload occuring during diastole, leading to a D-shaped deformation of the LV short-axis morphology, which returns to the normal circular LV shape post closure at the expense of septal myocardial shortening in the circumferential direction during systole. In patients who have undergone ASD closure, the leftward shifting of the interventricular septum disappears, resulting in recovery of LV circumferential shortening^[19].

In respect to LV diastolic function, the results of current study revealed improvement as demonstrated by significant reduction in **LV-Tie** after one month and 3 months of ASD closure. This finding is in agreement with previous studies^[18, 22], where a significant decrease in LV-Tie after 6 months of ASD correction was demonstrated, and this was explained by the

improvement in LV function due to improved LV filling after the ASD closure.

Regarding the effect of ASD closure on RV function the current study demonstrated a significant decrease in all RV dimensions and parameters of RV systolic function one month and 3 months post closure. Our results in this respect are in accordance with most of the previous studies^[23,24].

In the current study there was a significant reduction of T ann-Sa and TAPSE one month and three months after closure compared to pre closure. This finding is in accordance to what was reported by Akula et al. ^[25], who explained on the basis of increased RV end diastolic volume in ASD and hence apparent increase in RV systolic function following Starling's law of the heart, and after ASD closure the reduction in the preload return the RV systolic function to normal level. The load dependent parameters such as TAPSE and T ann-Sa had significantly decreased post-ASD device closure creating a doubt of RV systolic dysfunction, but are normal according to the ASE guidelines. Likewise, our findings of reduced TAPSE post ASD closure compared to pre closure are in accordance with other previous investigators who reported similar results after ASD closure^[26-28].

The right ventricular fractional area change (**RV-FAC**) in the present study showed a significant reduction of the mean value in one month after closure compared to pre closure. This finding is in agreement with **Mohammed** *et al.*^{[29],} and can be explained by the same explanation for reduction in TAPSE after ASD closure.

The present study showed a significant reduction of **RV-GLS** in one month and three months post closure. This finding coincides with those of **Jategaonkar** *et al.*^[27], and other previous studies^[18,21] that demonstrated a significant reduction of the global RV strain after 3 months of closure, and explained that by the increased myocardial strain values of ASD patients when compared with age-matched healthy adults due to chronic RV volume overload in patients with ASD, that returns to normal after ASD closure.

The 4D measured **RVEDV** and **RVESV** in the current showed significant reduction after one month and three months post ASD closure. This is in accordance with what was reported by **Miki** *et al.*^[19] and **Agha** *et al.*^[22].

In the present study, the **4D-RVEF** showed a significant reduction in one month after closure compared to pre closure, which coincides with **Miki** *et al.* ^[19] demonstrating the same result. However, after 3 months of follow-up in the present study, there was some increase in 4D-RVEF in comparison to one month of closure, this finding is in agreement with another study conducted by **Agha** *et al.*^[22], who demonstrated nonsignificant increase in 4D-RVEF six months after ASD closure.

In the current study although TAPSE, RV-FAC, TV-Sa and RV-GLS showed reduction in one and three months post ASD closure, the value in these 4 parameters were significantly higher in the three month post closure compared with one month post closure. These findings would suggests that the RV systolic function might be improved on the longer term after ASD closure. This suggestion is supported by the findings of Akula et al.^[25], who reported in their study that the transient decrease in basal RV systolic function early post closure had improved by 6 months after ASD device closure. This speculation would also be validated with the use of other gold standard imaging modality like cardiac magnetic resonance (CMR). In accordance with this speculation, Schoen et al.^[30], found improvement in RVEF by magnetic resonance imaging in association with a reduction in RV systolic pressure. In another study conducted by Teo et al.^[31], they reported that a significant reduction in RV volumes at 6 months after ASD closure while the RVEF was significantly increased in cardiovascular magnetic resonance (CMR), and they explained that by the fact that CMR is an accurate and reproducible imaging modality for the assessment of cardiac function and volumes.

As regards the RV diastolic function, we found that there were a significant decrease in **TV E/Ea** and **RV-Tie** after one and three months of closure. **Akula** *et al.*^[25], and **Mangiafico** *et al.*^[32], reported that RV-Tie is a reproducible index, which could be easily calculated by standard Doppler evaluation, as it is independent from load dependency and from RV geometry, and has been extensively evaluated in patients with ASD to assess the global systolic and diastolic RV function. So they recommended this index as a useful measure for assessment of RV function.

Study Limitatations: This study is limited by the relatively small number of cases, the relatively short follow-up period after ASD closure, and the absence of other gold standard imaging modality for evaluation of ventricular fuction.

CONCLUSION

Transcatheter closure of secundum ASD leads to structural and functional changes in both ventricles, with improvement of LV systolic and diastolic function, improvement of RV diastolic function and an apparent deterioration in RV systolic function that would be related to the preload status with over-estimation in function before ASD closure and normalization post closure.

Further studies with larger cohort size and longer follow-up periods post ASD closure, with inclusion of other imaging modality are recommended to validate the results of the current study.

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