Remodeling process in right and left ventricle after percutaneous atrial septal defect closure in adult patients

Erişkin hastalarda perkütan yolla atriyal septal defekt kapatılmasının sağ ve sol ventrikülün yeniden şekillenmesi üzerine etkisi

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ABSTRACT

Objective: The aim of this study was to evaluate acute cardiac remodeling after transcatheter closure of atrial septal defect (ASD) in adult patients.

Methods: This study included 19 patients (40.0±13.51 mean age) with secundum ASD who underwent successful transcatheter closure. All patients received routine transthoracic echocardiography, including tissue Doppler left ventricle (LV) and right ventricle (RV) myocardial performance indices (MPI), mitral annular plane systolic excursion (MAPSE) and tricuspid annular plane systolic excursion (TAPSE) before ASD closure, on first day, and at first and third months after closure.

Results: Left ventricle end-diastolic diameter (LVEDD) increased (37±4 mm to 44±5 mm, p<0.001); RVEDD decreased markedly after closure (40±4 mm to 32±5 mm, p<0.001); and differences existed in LV and RV adaptation. While MAPSE (1.87±0.22 cm to 2.01±0.23 cm, p<0.001) and LVMPI improved soon after procedure (0.44±0.04 to 0.36±0.03, p<0.001), RVMPI worsened until the first month post-procedure (0.25±0.02 to 0.31±0.03, p<0.001), but recovered by the third month follow-up visit (0.31±0.03 to 0.27±0.02, p<0.001). TAPSE remained unchanged (2.49±0.46 cm to 2.51±0.32 cm, p=0.078).

Conclusion: Closure of ASD using the Amplatzer Septal Occluder device led to a decrease in RV size and an increase in LV size. In the early period, while LV function improved, RV function deteriorated, probably due to RV functional and anatomical differences.

ÖZET

Amaç: Bu çalışmada, atriyal septal defekti (ASD) olan erişkin hastalarda perkütan yolla ASD kapatma işlemi sonrası sağ ve sol ventrikülün (RV, LV) erken dönem yeniden şekillenme süreci değerlendirildi.

Yöntemler: Bu çalışmaya başarılı transkateter kapatma uygulanan sekundum ASD'li 19 hasta alındı (40.0±13.51 yaş). Triküspit kapak anüler plan sistolik hareketi (TAPSE), mitral kapak anüler plan sistolik hareketi (MAPSE) ve doku Doppler ile yapılan RV ve LV miyokart performans indeksi (MPI) ölçümlerini içeren rutin ekokordiyografik ölçümler yapıldı. Ekokardiyografik ölçümler kateter yoluyla ASD kapatılmasından önce, kapatma sonrası 1. gün, 1. ay ve 3. ayda yapıldı.

Bulgular: LV diyastol sonu çapı arttı (37±4 mm ve 44±5 mm, p<0.001) ve RV diyastol sonu çapı azaldı (40±4 mm ve 32±5 mm, p<0.001). ASD kapatılmasına RV ve LV ventrikül farklı adaptasyon mekanizmalarıyla yanıt verdi. MAPSE arttı (1.87±0.22 mm ve 2.01±0.23 cm, p<0.001) ve doku Doppler LVMPI düzelme gösterirken (0.44±0.04 ve 0.36±0.03, p<0.001), doku Doppler RVMPI 1. aya kadar bozulma gösterdi (0.25±0.02 ve 0.31±0.03, p<0.001) ve bu durum 3. ayda düzeldi (0.31±0.03 ve 0.27±0.02, p<0.001). TAPSE değerlerinde ise anlamlı bir değişim saptanmadı (2.49±0.46 cm ve 2.51±0.32 cm, p=0.078).

Sonuç: Amplatzer septal oklüder cihazı ile yapılan ASD kapatma işlemi RV'nin büzülmesine ve LV'nin genişlemesine neden olmuştur. Erken dönemde, LV fonksiyonları düzelirken RV fonksiyonlarının geçici olarak bozulmasının olası nedeni olarak RV'nin fonksiyonel ve yapısal farklılığı düşünülmüştür.

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Atrial septal defects (ASDs) are the most common form of congenital heart disease diagnosed in adults, accounting for around 10% of all congenital heart defects.[1] Of these, 80% of the defects are of the secundum type, and are usually located in the central part of the atrial septum, causing left-to-right shunt and volume load in the right ventricle (RV).

Percutaneous closure of ASDs should diminish right heart volumes by eliminating left-to-right shunt, and thus leading to positive remodeling of the RV.[2] However, such adaptation may take a long time, and can be inadequate in adult ASD patients. Ventricular interdependence is a defined phenomenon, and implies that left ventricular impact may well be anticipated post-ASD closure.[3] Furthermore, atrial changes post-ASD closure have been poorly understood.

In this study, changes in cardiac geometry and function in adult Turkish patients treated with percutaneous ASD closure are evaluated by conventional echocardiography, and the tissue Doppler Myocardial Performance Index (MPI).

METHODS

Patient population

From October 2011 to October 2013, 19 consecutive patients with ASD and eligible for percutaneous device closure were enrolled in this study. Indications for closure of the ASD were presence of a hemodynamically significant shunt, findings of right ventricular volume overload, and a Qp/Qs ratio greater than 1.5.

Exclusion criteria were; pulmonary vascular resistance greater than eight Woods despite 100% oxygen inhalation, ASD diameter greater than 38 mm, failure in positioning of transesophageal probe, permanent atrial fibrillation, presence of any contraindication to antiplatelet therapy, and refusal by patient to participate in the study. The study was approved by the local Ethics Committee, and all patients provided written informed consent before enrollment.

Transthoracic echocardiography

Echocardiography was performed on all patients by an experienced cardiologist using the Vivid 3 echocardiography machine (GE Medical Systems, Norway) with a 3.5 MHz probe, and with patients in the left lateral decubitus position. The presence of an ASD was determined using apical four-chamber and parasternal short-axis views. Pulmonary flow velocity and aortic (systemic) flow velocity were determined using "pulsed wave" Doppler positioning just proximal to the pulmonary valve in the parasternal short-axis view, and positioning just proximal to the aortic valve in the apical four-chamber view respectively. The Qp/Qs ratio was calculated using pulmonary flow velocity/systemic flow velocity/0.6 formulae.

Abbreviations:

2D	*Two-dimensional*
3D	*Three-dimensional*
Aa	*Late diastolic*
ASD	*Atrial septal defect*
ASO	*Amplatzer Septal Occluder*
Ea	*Early diastolic*
EDD	*End-diastolic diameter*
EF	*Ejection fraction*
ESD	*End-systolic diameter*
LV	*Left ventricle*
MPI	*Myocardial Performance Index*
MRI	*Magnetic resonance imaging*
RV	*Right ventricle*
TDI	*Tissue Doppler imaging*

Right and left ventricular echocardiographic measurements were performed in accordance with the recommendations of the American Society of Echocardiography,[4,5] while ventricular volumes and ejection fractions were obtained by the modified biplane Simpson's method. Left atrial dimensions, left ventricle (LV) end-diastolic diameter (EDD) and end-systolic diameter (ESD), interventricular septal thickness, and LV end-diastolic posterior wall thickness were measured from the parasternal long-axis view. Mitral inflow E and A velocities were measured from the apical 4-chamber view.

Tissue Doppler imaging (TDI) measurements were made by placing the probe on the lateral wall of the LV, 1 cm apical to the mitral annulus. From the TDI of the left ventricular lateral annulus, systolic (Sa), early diastolic (Ea), and late diastolic (Aa) velocities were recorded. From these TDI recordings, the onset to end time interval of the mitral annular velocity pattern during diastole (a) was measured. The duration of the Sa (b) was measured from onset to end of the Sa. The LV MPI was calculated as (a-b)/b,[6] while MAPSE was measured using two-dimensional (2D) echocardiography-guided M-mode recordings from the apical 4-chamber view, with the cursor placed at the free wall of the mitral annulus.

RV and right atrial (RA) dimensions were measured at end-diastole from a right ventricle-focused apical 4-chamber view. RV basal diameter, subcostal thickness, and outflow tract parasternal short-axis distal and long-axis proximal diameters were used to evaluate RV dimensions. RA major and minor dimen-

sions, and end-systolic area parameters were used to evaluate RA dimensions.

Using 2D imaging for assessment of RV function, the TDI cursor was placed on the RV-free wall, 1 cm apical to the tricuspid annulus from the apical 4-chamber view, and the S', E', and A' were recorded. The pulse wave Doppler velocity range was -20 to 20 cm/sec. From the TDI recordings, the onset to end time interval of the tricuspid annular velocity pattern during diastole (a) was measured. The duration of S' (b) was measured from the onset to the end of the S', while RVMPI was calculated as (a-b)/b.[6] TAPSE was measured using 2D echocardiography-guided M-mode recordings from the apical 4-chamber view, with the cursor placed at the free wall of the tricuspid annulus.

Transesophageal echocardiography

Transesophageal echocardiography (TEE) was routinely performed on all patients screened for transcatheter closure in order to assess ASD morphology, and exclude additional lesions such as an anomalous pulmonary venous connection. The anterior-superior rim, posterior rim, vena cava superior (VCS) rim, vena cava inferior (VCI) rim, and anterior-inferior rim were all calculated to determine ASD eligibility for percutaneous closure.

Catheter intervention

All procedures were carried out under local anesthesia and guided by fluoroscopy. The Amplatzer Septal Occluder (ASO) (AGA Medical, Golden Valley, Minnesota) was used on all subjects. After calculating the Qp/Qs ratio using left and right heart saturations, all patients underwent balloon sizing of the defect. The preferred ASO was 2-4 mm larger than the stretched diameter.

Aspirin therapy (100 mg/day) was initiated at least 2 days prior to, and maintained for at least 6 months after, the intervention. Intravenous heparin was administered intraprocedurally. After routine hemodynamic evaluation, the ASD closure was performed.

Follow-up

Follow-up echocardiographic examinations were performed after the procedure at 1 day, 1 month, and 3 months.

Statistical analysis

Statistical analyses were conducted using the SPSS for Windows 11.5 program. The distribution of continuous variables was assessed by the Shapiro-Wilk test for normal distribution. Continuous variables are presented as mean±standard deviation or median (interquartile range), where applicable.

Repeated measurements of ANOVA were used to determine any statistically significant difference among the follow-up times. If there was a statistically significant difference in the variance analyses, the Bonferroni adjusted multiple comparison test was used to determine follow-up times that caused this. Results were accepted as significant if $p<0.05$.

The Friedman test was used to determine any significant difference in the median values among follow-up times. In the case of a statistically significant difference in the Friedman test, the Bonferroni adjusted Wilcoxon Signed Rank test was used to determine the follow-up times that caused this. According to the Bonferroni Correction, results were accepted as significant if $p<0.0083$.

Bland-Altman plots were used to assess both inter- and intra-observer agreement regarding RVMPI. The

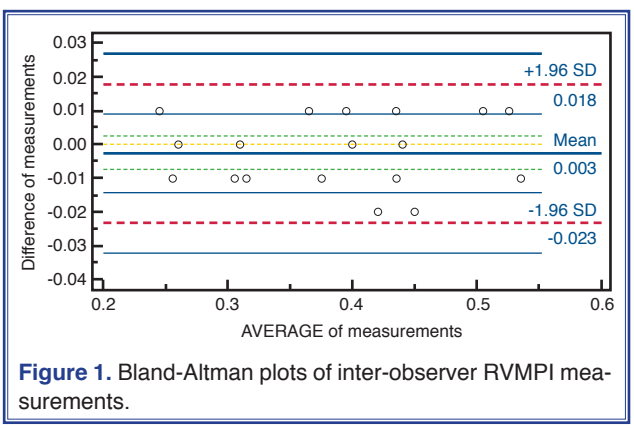

Figure 1. Bland-Altman plots of inter-observer RVMPI measurements.

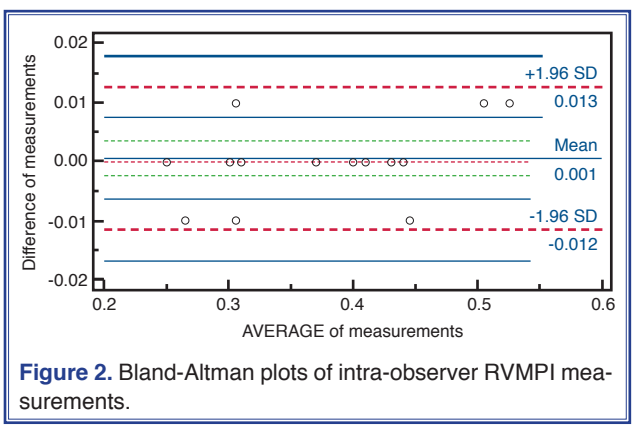

Figure 2. Bland-Altman plots of intra-observer RVMPI measurements.

Table 1. Changes in echocardiographic parameters before and after ASD closure

Variables	Before	1st day	1st month	3rd month	*p*
LVEDD (mm)	37.02 (4.01)*	43.01 (4.98)†	43.98 (5.01)†	44.01 (4.99)†	**<0.001**
LVESD (mm)	25.01 (1.03)*	24.98 (2.02)*	25.01 (1.02)*	25.02 (1.01)*	0.304
LVEF (%)	62.01 (2.97)*	67.99 (4.02)†	69.02 (3.01)†	75.03 (3.98)‡	**<0.001**
RVEDD (mm)	40.02 (3.98)*	35.02 (6.01)†	33.03 (4.98)†,‡	32.02 (4.97)‡	**<0.001**
LVMPI	0.44 (0.04)*	0.37 (0.03)†	0.38 (0.03)†	0.36 (0.03)†	**<0.001**
RVMPI	0.25 (0.02)*	0.31 (0.03)†	0.38 (0.02)‡	0.31 (0.02)†	**<0.001**
TASV (cm/sec)	17.47 (2.11)*	15.10 (2.20)†	13.73 (1.96)‡	14.68 (2.21)†	**<0.001**
LA diameter (cm)	3.41 (1.00)*	3.52 (0.85)*	3.51 (0.67)*	3.32 (0.67)*	0.270
RA diameter (cm)	4.82 (0.77)*	3.91 (0.85)†	3.62 (0.65)†,‡	3.41 (0.55)‡	**<0.001**
RAEDV (ml)	67.02 (11.91)*,†	60.51 (15.42)*,‡	51.12 (11.13)‡,§	50.13 (14.51)§	**<0.001**
RAESV (ml)	39.03 (14.24)*	33.51 (11.22)*	30.53 (12.52)*	29.21 (8.63)*	0.053
TAPSE (cm)	2.17±0.40‖	2.49±0.46‖	2.49±0.38‖	2.51±0.32‖	0.078
MAPSE (cm)	1.87±0.22‖	2.03±0.22¶,**	2.08±0.21¶	2.01±0.23 ‖,**	**<0.001**
RVEDD/LVEDD	1.04±0.11‖	0.77±0.11¶	0.74±0.09¶	0.73±0.09¶	**<0.001**
RA EF	45.02±7.53‖	56.23±7.24¶	61.21±8.27**	66.02±6.86**	**<0.001**

LVEDD: Left ventricle end diastolic diameter; LVESD: Left ventricle end systolic diameter; LVEF: Left ventricle ejection fraction; RVEDD: Right ventricle end diastolic diameter; LVMPI: Left Ventricle Myocardial performance index; RVMPI: Right Ventricle Myocardial performance index; LA: Left atrium; RA: Right atrium; RAEDV: Right atrium end diastolic volume; RAESV: Right atrium end systolic volume; TASV: Tricuspid systolic annular velocity; TAPSE: Tricuspid annular plane systolic excursion; MAPSE: Mitral annular plane systolic excursion; RVEDD: Right ventricle end diastolic diameter; LVEDD: Left ventricle end diastolic diameter; RAEF: Right atrium ejection fraction. The same indexes: *, †, ‡, § in each row indicates that there is no statistically significant difference between follow-up times (p>0.0083, Bonferroni Adjusted Wilcoxon Signed Rank test). The same indexes: ‖, ¶, ** in each row indicates that there is no statistically significant difference between follow-up times (p>0.05, Bonferroni Adjusted multiple comparison test).

means of agreement differences (i.e. bias) and upper and lower limits set at 0.95 confidence interval were also calculated. Inter- and intra-observer reproducibility showed perfect agreement for RVMPI measurements before ASD closure (Bland-Altman mean difference: -0.003, 0.95, CI: -0.023-0.018) (Figure 1) and (Bland-Altman mean difference: 0.005, 0.95 CI: -0.012- 0.013) (Figure 2) respectively. Bland-Altman plots were performed using MedCalc, version 11.1.1.0 (MedCalc software, Broekstraat 52, B-9030 Mariakerke, Belgium).

RESULTS

Patient mean age was 40±13.51. Four were male, and 15 female. Mean height was 162.42±5.81 cm, mean weight 68.23±6.21 kg, and mean body mass index 25.32±2.13 kg/m2. Mean defect diameter was 17.13±5.52 mm, mean Qp/Qs 2.54±1.11, and mean pulmonary artery systolic pressure 44.31±12.23 mmHg. Mean fluoroscopy time during percutaneous closure was 17.52±5.83 minutes.

Table 1 shows the changes in echocardiographic parameters before, and 1 day, 1 month, and 3 months after transcatheter ASD closure. There was a significant difference between pre-procedure LVEDD, and post-procedure LVEDD on the first day ($p<0.001$), at first month ($p<0.001$), and at third month ($p<0.001$). However, there was no significant difference in post-procedure LVEDD values between the first day and first month ($p=0.658$), first day and third month ($p=0.944$) and first and third months ($p=0.135$). LV ejection fraction (EF) noticeably improved after the first day ($p<0.001$). There was no meaningful difference between the pre- and post-procedural LV ESD values. There was a marked decline in RV diameters on the first day, at first month, and at third month post-procedure compared to pre-procedure values ($p<0.001$). RV tissue Doppler MPI values on the first day, at first month, and at third month post-procedure increased statistically compared to pre-procedure values ($p<0.001$). RV tissue Doppler MPI values greatly increased after the first post-procedure month compared to the first day ($p<0.001$).

However, there was a relevant decline after the third month compared to the first month (p<0.001) (Figure 3). Tricuspid annular plane systolic velocity (TASV) values were decreased on the first day, at first month, and at third month post-procedure compared to pre-procedure values (p<0.001), while there was an increase at the third month compared to the first month (p<0.001).

LV tissue Doppler MPI values were considerably reduced on the first day, at first month, and at third month post-procedure compared to pre-procedure values (p<0.001). However, there was no meaningful difference in LV tissue Doppler MPI values between the first day and first month (p=0.804) and first day and third month (p=0.084) post-procedure (Figure 4).

There was no noteworthy difference in LV tissue Doppler MPI values between the first and third month post-procedure (p=0.108). According to these results, LV tissue Doppler MPI values decreased immediately following the procedure, while RV tissue Doppler MPI values increased until the first month post-procedure, then decreased, but did not reach pre-procedure values. There was no correlation between ASD diameters, Qp/Qs, ventricle diameters, RV tissue Doppler values, or LV tissue Doppler values.

The pre-procedure RVEDD/LVEDD ratio significantly decreased on the first day (p<0.001), at first month (p<0.001), and at third month (p<0.001) post-procedure (Figure 5). However, there was no significant difference in the RVEDD/LVEDD ratio between the first day and first month (p=0.107), the first day and third month (p=0.081), or between the first and third months post-procedure (p=0.293). No correlation existed between changes in RVEDD/LVEDD ratio and ASD diameter and Qp/Qs ratio.

TAPSE values improvement did not reach statistical significance after the first day post-procedure (p=0.078). There was no significant change in TAPSE values between the first day and first month (p=0.844), the first day and third month (p=0.825), or the first and third months (p=0.586) post-procedure. Evaluation of MAPSE values showed a statistically important increase in MAPSE on the first day, at first month, and at third month (p<0.001) post-procedure. When compared to pre-procedure values, RAEF values were markedly increased on the first day, at first month, and at third month (p<0.001) post-procedure (Table 1).

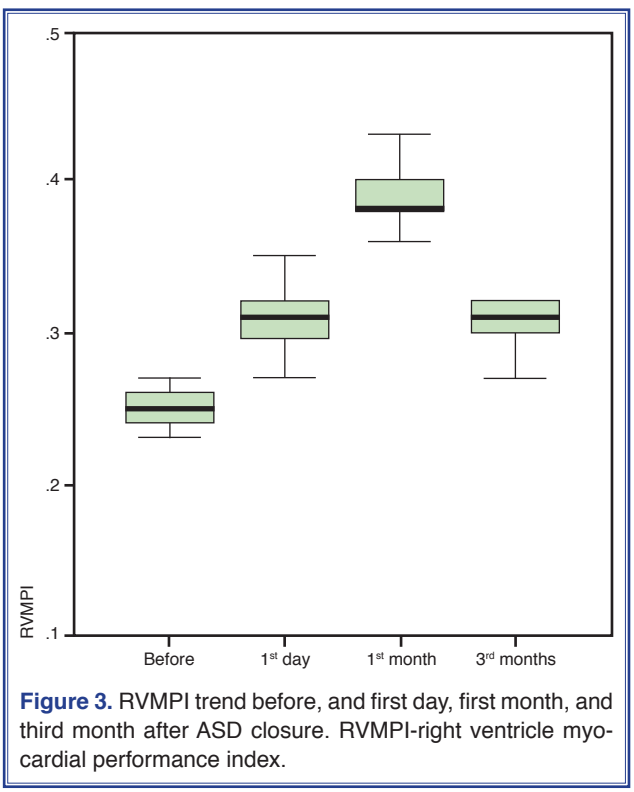

Figure 3. RVMPI trend before, and first day, first month, and third month after ASD closure. RVMPI-right ventricle myocardial performance index.

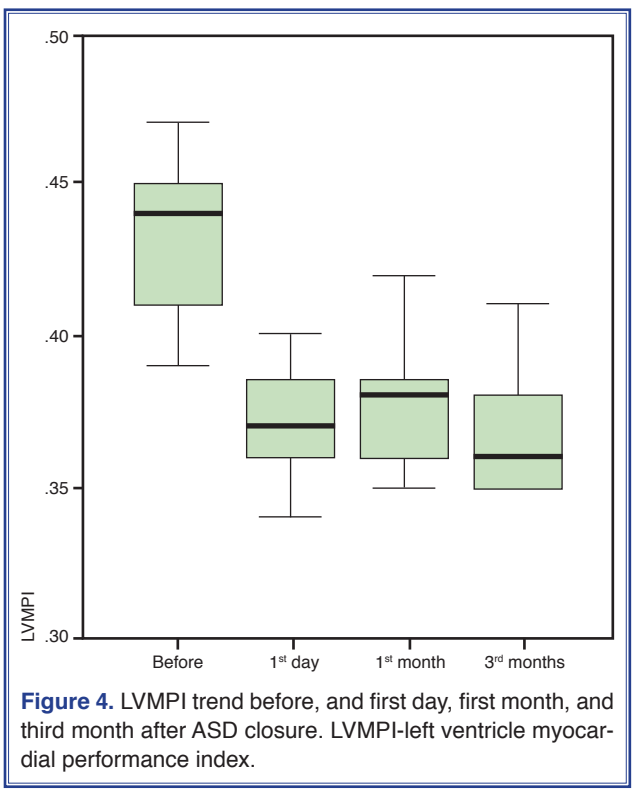

Figure 4. LVMPI trend before, and first day, first month, and third month after ASD closure. LVMPI-left ventricle myocardial performance index.

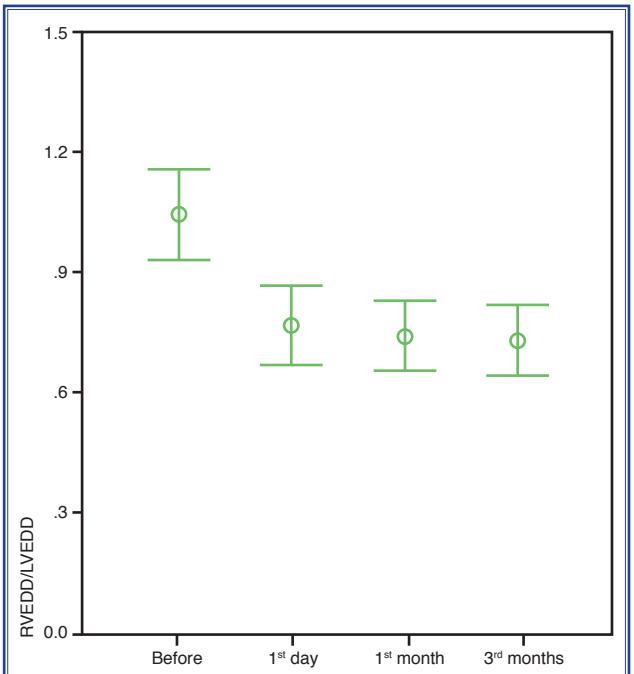

Figure 5. Serial changes in RVEDD/LVEDD ratio before, and first day, first month, and third month after ASD closure. RVEDD-right ventricle end-diastolic diameter; LVEDD-left ventricle end-diastolic diameter.

DISCUSSION

This study evaluated echocardiographic changes in the left and right heart during a three-month follow-up period after percutaneous closure of an ASD. A decrement was shown in multiple echocardiographic parameters such as the RVEDD, right atrial diameter, RV/LV EDD ratio (regarded as an indicator of cardiac geometry), right atrial diameter, and LVMPI as early as the first day post-procedure. In addition, LVEDD, MAPSE, and LVEF were increased. TAPSE and left atrial diameter remained unchanged in the three-month post-procedure period, while RVMPI increased in the first month, but was found to be decreased at the third month post-procedure. Previous studies have also demonstrated similar findings of decreased right atrial and ventricular dimensions after percutaneous ASD closure. Veldtman et al.[7] found that right ventricle anatomy sustained a brisk improvement within one month of defect closure, with associated mechanoelectrical gain. Kort et al.[8] found a reduction in RV volume, but RA volume remained increased when compared to control subjects at 24 months after closure. Ağaç et al.[9] noted reductions at the first month in echocardiographic parameters such as RVEDD, right atrial diameter, and RVEDD/LVEDD ratio after percutaneous ASD closure. Thilén et al.[10] demonstrated that cardiac remodeling primarily took place within the first week following closure, and was frequently finished by four months. They notably revealed that both RA and RV decreased in size, while the LV increased, and the LA remained unchanged. Additionally, the ventricles seemed to have a greater capacity for remodeling than the atria in this study. Santoro et al.[11] observed 24 patients with echocardiographic follow-up at 24 hours and 4 weeks after percutaneous closure of large ASDs. They noted a relevant decrease in RV diastolic diameter and an important expansion in LV diastolic diameter at 24-hours post-closure. These changes continued at one month, and while a marked reduction in RA diameter was apparent at this stage, LA size remained unchanged.

A study conducted by Salehian et al.[12] showed left atrial diameter decreased at the first day post-procedure, and decreased significantly at the sixth month when compared to pre-procedure values. The belief was that this decrease was due to improvement in left ventricular pressures and decreased left atrial preload after ASD closure. In a cohort of patients of mean age 42.6±16.3, Giardini et al.[13] showed significant dilatation of the left atrium immediately following ASD closure, due to hemodynamic changes. The authors speculated that this dilatation was the result of rapid increases in left atrial and left ventricular diastolic pressures.

In our study, It was thought that left atrial remodeling would be echocardiographically evident at 3 months following percutaneous ASD closure. However, left atrial diameter did not significantly change in the first 3 months after ASD closure. One reason could be rigidity in the closure device, which may negatively affect left atrial contraction. RVEDD/LVEDD ratios, which are another parameter of cardiac geometry, significantly decreased after ASD closure in our study, results which are similar to those of Du et al.,[14] Santoro et al.,[11] Ağaç et al.,[9] and Kaya et al.[15]

Discussion centers on the possible correlation between decrease in RVEDD/LVEDD ratio and pre-procedure right and left ventricular diameter. Monfredi et al.[16] showed that pre-procedure absence of increased ventricle diameter is an important marker affecting

remodeling positively. Du et al.[14] treated ASD patients using the Amplatzer device, and demonstrated a rapid decrease in right ventricle volume load, and an increase in right ventricular function. Also shown was a decrease in right ventricular diameter, which was inversely correlated with age, and pre-procedure diameter of the right ventricle. Because of this correlation, they recommend early treatment of ASD, as more beneficial to patients.

Santoro et al.[11] showed that positive remodeling starts immediately following percutaneous ASD closure, and that there was no correlation with the duration and severity of volume load. The present study also found that there was no correlation between biventricular remodeling and pre-procedure diameter of the right ventricle and the duration of volume load. It can be anticipated that ASD closure would lead to decreased right heart volumes due to removal of left-to-right shunt. Whether this enhances RV performance or merely inhibits further decline remains debatable.

The MPI is a proven index, used to assess the global evaluation of right ventricular systolic and diastolic functions. Ağaç et al.[9] demonstrated that patients having abnormal baseline RVMPI values had no significant changes at the first month following closure, while the long-axis RV function parameter TAPSE decreased after closure. In line with that study, Salehian et al.[12] reported a high basal RVMPI value before closure, with the value normalizing following closure. LVMPI values also decreased. We have shown a deterioration in previously normal RVMPI values at the first month following closure, with these values normalized at the third month despite decreased LVMPI values soon after the procedure. Wu et al.[17] also demonstrated similar findings with regard to the RVMPI and LVMPI trend at three-month follow-up, and thought that right ventricle temporary and subclinical dysfunction might be due to acute right ventricular mass reduction after the decrease in right ventricular volume load.

Contrarily, we demonstrated that TAPSE remained unchanged, while long-axis LV function parameter MAPSE increased. However, an increasing trend in TAPSE values after the first month of closure was observed. This may be attributed to the short follow-up echocardiographic examination and small sample size of patients in our cohort. Furthermore, the anatomical complexity of the right ventricle, difference in orientation of myocardial fibers, and reduced wall thickness may play a role in this result.

In contrast to the findings in the above-mentioned studies, Monfredi et al. failed to show any significant change in either RV or LV MPI values. One reason for the former results in evaluating RV performance may be the limitation of two-dimensional echocardiography in evaluating the RV due to its unsuitable geometry.[16] O'Brien et al.[18] showed a decrease in RVEDD 24 hours after ASD closure. However, this was not correlated with a decrease in right ventricular volume. They contributed these findings to the geometric complexity of the right ventricle. It has been shown in several studies that due to this complexity and the failure of simple geometric formulas in calculation, evaluating right ventricular volume and functions is more difficult than evaluating those of its left counterpart.[19]

Wu et al.[17] demonstrated similar findings with regard to RVMPI and LVMPI trends at three-month follow-up, and concluded that right ventricle temporary and subclinical dysfunction might be due to acute right ventricular mass reduction after the decrease in right ventricular volume load. Additionally, in the present study, we have also demonstrated that TAPSE remains unchanged while long-axis LV function parameter MAPSE is increased.

The functional complexity of the right ventricle, or the difference in orientation of Speckle-tracking, 2D strain analysis, and three-dimensional (3D) echocardiography, has recently been employed as a novel approach in assessing RV function before and after percutaneous ASD closure. Ding et al.[20] monitored 46 patients with 3D echocardiography following percutaneous closure of ASDs with scans at 24 hours, 3 days, and 1 month after closure. They also showed a significant decrease in RV end-diastolic volume and RV end-systolic volume at 24 hours post-closure. Eerola et al.[21] demonstrated decreases in diastolic RV dimensions, along with significant increases in LV diastolic and systolic dimensions by 3D echocardiography. These results are consistent with the results of the present study in terms of morphological changes in the right ventricle in the early stage of percutaneous ASD closure.

In this study, a significant decrease in RVEDD after percutaneous ASD closure has been shown.

Vitarelli et al.[22] evaluated 39 patients before and 6 months after closure, and demonstrated that 3D-RVEF and global and regional RV longitudinal strain were considerably higher than in the control group, and decreased meaningfully after closure. Jategaonkar et al.[23] studied 33 patients before and 3 months after closure. They found that while global longitudinal strain and regional peak systolic strain of the RV were significantly decreased by ASD closure, global longitudinal strain rate was not. In accordance with the latter study, we have demonstrated that RVMPI values were normalized at the third month after closure, but deterioration in the tissue Doppler RVMPI value was observed during the first month after closure.

A number of authors have utilized cardiac magnetic resonance imaging (MRI) to evaluate the effects of ASD closure on the heart.[24-26] MRI provides high temporal and spatial resolution of cardiac function and morphology, and has been demonstrated to be beneficial for the follow-up of patients with ASDs.[27,28] However, its routine use in clinical practice is neither economical nor feasible.

There are some limitations to our study. Several studies have cited significant hemodynamic changes occurring in the first 3 months after ASD closure.[7,14] The present study was designed based on this evidence. However, patient follow-up of more than 3 months is needed for a complete evaluation of hemodynamic changes. Our study was conducted on a limited number of patients, which makes it difficult to arrive at a definitive statement. Although many complex echocardiographic techniques were employed, further techniques, such as 3D echo, speckle tracking, and strain assessment, would provide a more thorough evaluation of the effects of percutaneous ASD closure on the heart. Another limitation of our study is the lack of a control group to compare the baseline values of various echocardiographic parameters.

In conclusion, our study shows that acute shrinkage of the right ventricle occurs after ASD closure, accompanied by expansion of the left ventricle. These changes in the ventricles are not correlated with preprocedure ventricle diameters. Each ventricle shows a different adaptive response to the rapid hemodynamic changes and the negative effects of the rigid closure device. Both MAPSE, an indicator of left ventricular longitudinal movement, and left ventricular tissue Doppler MPI, an indicator of left ventricular global function, improve rapidly after ASD closure. However, TAPSE, an indicator of right ventricular longitudinal movement, does not improve with time. Right ventricular tissue Doppler MPI, an indicator of right ventricular global function, worsens after ASD closure, but improves after 3 months. The structural and anatomical differences of each ventricle, and delayed response of the right ventricle to acute volume changes in our patients' age group may be the reasons for these results.

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Key words: Atrial septal defect; echocardiography; left ventricle; right ventricle; ventricular remodeling.

Anahtar sözcükler: Atrial septal defekt; ekokardiyografi; sol ventrikül; sağ ventrikül; ventriküler yeniden şekillenme.