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Original Article

Evaluation of Early Changes in Atrial and Ventricular Speckle-Tracking After the Device Closure of Atrial Septal Defects

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ABSTRACT

Background: The transcatheter device closure of secundum type atrial septal defects (ASDs) has now become the first-line strategy in patients with suitable anatomies. Device closure has remarkable effects on the hemodynamics and function of the heart. Precise diagnosis of these changes is significant in determining patients' outcomes and prognoses. In the current study, we evaluated volumetric, functional, strain, and strain rate changes in the cardiac chambers following successful device closure.

Methods: The present prospective cohort study was conducted on 45 patients eligible for ASD device closure. The patients were evaluated for volumetric, functional, Doppler, strain, and strain rate data regarding the left and right atria and ventricles preprocedurally and 48 hours postprocedurally.

Results: Significant changes were found in the left ventricular (LV) end-diastolic volume index (P=0.03), the right ventricular (RV) diameter ($P\leq$0.001), the left atrial (LA) volume index (P=0.05), the right atrial (RA) volume index (P=0.001), and the right and left-sided E/e' ratio (P=0.001 and P=0.004, respectively). Our findings also showed significant reductions in the strain values of all 3 phases of the RA and the LA and the RV free wall after ASD device closure. The LV global longitudinal strain decreased after the procedure but did not reach statistical significance.

Conclusions: The LA, RA and RV strain values showed significant reductions after device closure. The decline in LA function following closure was greater in those with larger ASDs. In adult patients undergoing the procedure, abnormal LA function is a clinically relevant issue demanding pre and postprocedural precautions and treatments. ***(Iranian Heart Journal 2022; 23(4): 80-87)***

KEYWORDS: Atrial septal defect, Device closure, Speckle-tracking, Strain and strain rate

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Secundum type atrial septal defects (ASDs) constitute one of the most common congenital cardiac defects, resulting in left-to-right shunts, morphologic and functional changes in cardiac chambers and subsequent volume overload, arrhythmias, heart failure, and pulmonary hypertension.[1] The device closure of secundum type ASDs has become the standard of care in those with suitable rims. Following closure, the device disk forms part of the interatrial septum. The presence of a new septal prosthetic wall has functional implications.[1,2] The elimination of the communicating defect changes cardiac chamber preloads.[3] The left-sided preload increases, whereas the preload of the right side chambers is reduced.[3] As preload is a determinant of cardiac function, related alterations in functional and hemodynamic parameters are expected.

In patients with ASDs, routine echocardiographic changes are generally detected after remarkable structural and functional changes. Such changes signify the need for echocardiographic parameters that could predict changes before their occurrence. Speckle-tracking and deformation indices are instances of such measures with the capacity to detect subclinical changes, acceptable reproducibility, and angle independency.[4] Previous studies have generally focused on atrial hemodynamics following device closure. In the present study, we aimed to assess global functional and hemodynamic changes in patients undergoing transcatheter ASD closure.

METHODS

The present prospective cohort study evaluated 76 patients scheduled for ASD device closure based on suitable anatomy in transesophageal echocardiography, significant left-to-right shunts, and no evidence of irreversible pulmonary hypertension.[2] From this cohort, patients under 16 or over 50 years of age, those with cardiovascular risk factors (eg, diabetes mellitus, hypertension, hyperlipidemia, and smoking), a history of previous cardiac surgeries or concomitant congenital heart diseases, a history of cardiac device implantation or systemic diseases (eg, liver, kidney, lung, and endocrine disorders), valvular stenosis, regurgitations exceeding mild-to-moderate levels, pulmonary systolic pressures exceeding 35 mm Hg, cardiomyopathies, and cardiac arrhythmias, especially atrial fibrillations were excluded. All the selected patients were in the sinus rhythm. Subsequently, 5 patients were excluded due to increased left ventricular (LV) end-diastolic pressures in the catheterization laboratory after sizing balloon inflation and implantation of a fenestrated device and 3 patients were excluded due to device-related complications (erosion and residual shunts). Eventually, 45 patients fulfilled the eligibility criteria for the study. The institutional ethics committee approved the study protocol.

Echocardiography was done during single-lead electrocardiography and adjusted to record 3 consecutive cardiac cycles.

Standard 2, 3, and 4-chamber views for the LV global longitudinal strain and strain rate measurements, the right ventricular (RV) standard view for the RV free wall strain and strain rate, and left atrial (LA) and right atrial (RA) guideline-based focus views were obtained for offline analysis with a frame rate of 40 to 80 per second.[5,6] Chamber diameters, volumes, ejection fractions, and Doppler data (mitral inflow E and A waves, the deceleration time, the E/A ratio, the averaged e', the E/e' ratio, tricuspid inflow E and A waves, the E/A ratio, the S wave, and e' with tissue Doppler and tricuspid systolic plane excursion [TAPSE]) were acquired pre and 48 hours postprocedurally.

Statistical Analysis

Data were acquired using a Philips EPIQ 7 machine and analyzed with the Q-lab software, version 13. The statistical analyses

were performed using the SPSS software, version 22.0 (SPSS, Chicago, IL, USA). The results were considered statistically significant if 2-sided *P* values were less than 0.05. Continuous data were reported as the mean ± the standard deviation (SD), and numerical values with normal distributions for comparison before and after device closure were assessed using the paired *t* test and the Wilcoxon method.

Ethical Considerations

Informed consent was obtained from all the patients after they had received complete explanations. The study protocol was approved by the Research Ethics Committee of Iran University of Medical Sciences (ethics code: IR.IUMS.REC1399.102).

RESULTS

The study population consisted of 33 female and 12 male patients. The mean age of the patients was 37.8±10.9 years. The mean ASD size and the mean device diameter were 20.6±5.3 mm and 23.3±6.1 mm, respectively. The largest device used was 36 mm in size. The volumetric, functional, and Doppler values before and after the procedure are summarized in Table 1. Cardiac chamber volumes showed significant changes following device closure. The LA, RA, and RV volumes decreased early after the procedure, whereas the LV volume increased (42.26±6.96 mm^3 vs 43.74±6.93 mm^3 postprocedurally, *P*=0.03). Regarding Doppler findings, the left-sided E/e' showed a statistically significant increase (*P*=0.004). The E wave, E/e' and TAPSE decreased.

Table 1: Standard volumetric and Doppler echocardiography findings of the patients with ASDs pre and postprocedurally

Variables	Mean ± SD	*P* value
LVEDVI preprocedural (mL/m^2) LVEDVI postprocedural (mL/m^2)	42.26±6.96 43.747±6.93	0.03
LVESVI preprocedural (mL/m^2) LVESVI preprocedural (mL/m^2)	23.36±6.49 23.71±5.17	0.5
LVEF preprocedural(%) LVEF postprocedural(%)	50.66±3.47 50.11±3.91	0.2
RV diameter preprocedural (mm) RV diameter postprocedural (mm)	4.04±0.43 3.81±0.37	<0.001
LAVI preprocedural (mL/m^2) LAVI postprocedural (mL/m^2)	24.34±5.7 23.22±5.03	0.005
RAVI preprocedural (mL/m^2) RAVI postprocedural(mL/m^2)	34.90±8.09 31.73±6.70	<0.001
MVE wave preprocedural (cm/s) MVE wave preprocedural (cm/s)	74.75±15.74 78.15±16.95	0.1
MV E/A preprocedural MV E/A postprocedural	1.29±0.39 1.27±0.32	0.7
MV e' wave preprocedural (cm/s) MV e' wave postprocedural (cm/s)	10.20±2.29 10.80±2.81	0.6
MV E/e' preprocedural MV E/e' preprocedural	6.75±1.73 7.83±1.99	0.004
MV DT preprocedural l(ms) MV DT postprocedural (ms)	165.7±33.53 171.88±35.86	0.2
MV S wave preprocedural(cm/s) MV S wave postprocedural (cm/s)	11.60±1.99 9.80±1.39	0.3
TV E wave preprocedural (cm/s) TV E wave postprocedural (cm/s)	70.82±15.84 59.84±13.66	<0.001
TV A wave preprocedural l(cm/s) TV A wave postprocedural (cm/s)	56.11±15.34 53.11±10.91	0.2
TV DT preprocedural (ms) TV DT postprocedural (ms)	176.96±58.08 171.96±40.78	0.2
TV e' wave preprocedural(cm/s)	12.68±3.56	0.015

TV e' wave postprocedural (cm/s)	11.44±3.15	
TV S wave preprocedural(cm/s) TV S wave postprocedural (cm/s)	13.15±2.72 12.73±2.83	0.3
TV E/A wave preprocedural TV E/A wave postprocedural	1.27±0.38 1.10±0.32	0.01
TV E/e' wave preprocedural TV E/e' wave postprocedural	5.69±2.14 5.45±1.84	0.01
TAPSE preprocedural (cm) TAPSE postprocedural (cm)	23.93±4.14 22.37±3.65	0.003
RV FAC preprocedural(%) RV FAC postprocedural (%)	34.12±5.94 34.76±5.67	0.4

ASD, Atrial septal defect; LVEDVI, Left ventricular end-diastolic volume index; LVESVI, Left ventricular end-systolic volume index; LVEF, Left ventricular ejection fraction; RV, Right ventricle; LAVI, Left atrial volume index; RAVI, Right atrial volume index; MVE wave, Mitral valve E wave; MV E/A, Mitral valve E wave/A wave; MV DT, Mitral valve deceleration time; MV S wave, Mitral valve S wave; TV A wave, Tricuspid valve A wave; TV E wave, Tricuspid valve E wave; TV DT, Tricuspid valve deceleration time; TAPSE, Tricuspid annular plane systolic excursion; RV FAC, Right ventricular fractional area change

Table 2: Simultaneous changes in the strain and strain rate of the right and left chambers pre and post procedurally and their correlation with ASD size

Values	Mean±SD	Difference of the Strain and Strain Rate Before and After the Procedure (*P* value)	Correlation With ASD Size (*P* value)	Correlation Between the Difference of the Strain and Strain Rate and ASD Size (*P* value)
LV GLS preprocedural(%) LV GLS postprocedural (%)	18.38±6.48 17.90±5.87	0.15	0.2 0.3	0.9
LV strain rate preprocedural(%) LV strain rate postprocedural (%)	1.91±0.36 1.84±0.35	0.04	0.6 0.6	0.2
RVFWSL preprocedural(%) RVFWSL postprocedural (%)	20.6±3.004 19.48±2.228	0.01	0.4 0.4	0.5
RV strain rate preprocedural(%) RV strain rate postprocedural (%)	1.84±0.28 1.81±0.27	0.33	0.6 0.8	0.7
LA Sr preprocedural(%) LA Sr postprocedural (%)	27.34±8.35 21.85±7.19	<0.0001	0.9 0.03	0.02
pLASRr preprocedural(%) pLASRr postprocedural (%)	2.31±0.52 2.26±0.55	0.48	0.4 0.02	0.06
LA Scd preprocedural(%) LA Scd postprocedural (%)	16.12±7.22 12.93±5.76	<0.0001	0.8 0.03	0.006
pLASRcd preprocedural(%) pLASRcd preprocedural(%)	2.47±0.69 2.40±0.79	0.37	0.2 0.1	0.4
LA Sct preprocedural(%) LA Sct postprocedural (%)	10.84±2.95 8.88±2.98	<0.0001	0.8 0.5	0.4
pLASRct preprocedural(%) pLASRct preprocedural(%)	2.43±0.63 2.26±0.52	0.016	0.4 0.2	0.01
RASr preprocedural(%) RASr postprocedural (%)	33.85±7.10 29.37±7.77	<0.0001	0.5 0.7	0.3
pRASRr preprocedural(%) pRASRr preprocedural(%)	2.52±0.61 2.44±0.59	0.31	0.4 0.2	0.1
RAScd preprocedural(%) RAScd postprocedural (%)	19.04±6.02 17.44±5.39	0.03	0.7 0.3	0.1
pRASRcd preprocedural(%) pRASRcd preprocedural(%)	2.55±0.70 2.47±0.73	0.32	0.1 0.01	0.1
RASct preprocedural(%) RASct postprocedural (%)	14.74±4.41 12.27±4.12	0.001	0.6 0.1	0.4
pRASRct preprocedural(%) pRASRct preprocedural(%)	2.67±0.69 2.58±0.68	0.20	0.5 0.2	0.4

ASD, Atrial septal defect; LV GLS, Left ventricular global longitudinal strain; RVFWSL, Right ventricular free-wall longitudinal strain; LASr, Left atrial strain during the reservoir phase; LAScd, Left atrial strain during the conduit phase; LASct, Left atrial strain during the contraction phase; RA, Right atrial; pLASRr, Peak strain rate during the reservoir phase; pLASRcd, Peak strain rate during the conduit phase; pLASRct, Peak strain rate during the contraction phase; RA Sr, Right atrial during the reservoir phase; pRASRr, Peak strain rate during the reservoir phase; RAScd, Strain during the conduit phase; pRASRcd, Peak strain rate during the conduit phase; RASct, Strain during the contraction phase; pRASRct, Peak strain rate during the contraction phase

Strain values for the RV, the LA, and the RA showed a significant decline after device closure. There was a decline in the LV global longitudinal strain, but the change was not statistically significant.

Concerning the strain rates, only the LV strain rate (1.9±0.36 vs 1.8±0.035 postprocedurally, *P*=0.04) and the strain rate during the contraction phase of the LA (2.43±0.63 vs 2.26±0.52 postprocedurally, *P*=0.016) exhibited significant changes (Table 2). Despite significant changes detected in the strain study, the LV and RV ejection fractions remained constant.

The relationships between ASD size and its impact on the strain and strain rate values are shown in Table 2. There was a significant inverse relationship between ASD size and the LA reservoir and conduit functions and the peak strain rate during the reservoir phase. Moreover, ASD size showed no statistically significant relationship with the right-sided (the RA and the RV) strain and strain rates; however, the reductions in the strain and strain rates were greater after the closure of larger-sized ASDs (Table 2).

DISCUSSION

In the current study, we evaluated the early and simultaneous effects of ASD device closure on cardiac chamber volumes and hemodynamics. A limited number of previous studies have evaluated changes in echocardiographic parameters, including functional, Doppler, volumetric, and strain values, following ASD device closure. These studies have generally focused on the assessment of a single heart chamber. The results are variable and controversial, mostly due to the heterogeneity of patient selection and a combination of surgical and device closure procedures. Our study included a greater number of patients with device closure than previous studies.

The heart of an adult patient with an ASD has endured many years of volume overload, with the resultant global structural abnormalities.[1] The main chronic changes include increased diameters and volumes of the right-sided heart chambers as opposed to decreased volumes of the left-sided cardiac chambers, resulting from reduced filling and leftward bulging of the ventricular septum.[1,3,7] Conventionally measured echocardiographic values for the assessment of function only show changes when there are advanced structural and geometric deformations.[3,4] Device closure has hemodynamic consequences that are not readily detected by these methods. In the current study, we utilized the strain and strain rate measurements early following device closure to detect the subclinical and minor abnormalities of function at this stage.

In our study, the diameters and volumes of the RA, the RV, and the LA decreased after device closure, and the LV showed increased volumes. Right-sided diameters showed a reduction early on, due to the elimination of volume overload, and the LV diameters increased due to the relief of the RV septum deviation and increase in preload.[7] The LA volume decreased despite an increase in filling volumes, which we believe was caused by the bulk of the disk of the occluder device interfering with normal

filling in a chronically under filled and remodeled LA.[3] The diminished volume increase in the LA is more evident in large ASDs, where the device disk forms a long metallic wall in the LA. The filling pressures of the heart were estimated by the measurement of E/e', which represents ventricular end-diastolic pressures in the LV and the RV. The results revealed significant changes, including an increase in the LV and a decrease in the RV, after device closure, which could be explained by the shifts in preload.[3,8] We observed a significant decline in TAPSE and s' in the tissue Doppler imaging of the RV. There was also a slightly statistically insignificant increase in the fractional area change of the RV.

Strain imaging is used to measure local myocardial function based on fiber thickening, shortening, and lengthening.[5] The LA and RA functions consist of 3 phases in correlation with the ventricular function. The reservoir phase is reported with a positive strain value and is defined as a peak systolic atrial strain at the end of LA filling during LV systole and before mitral opening. The conduit phase has a negative strain value and occurs at the time of ventricular diastole, during which the blood is conducted to the LV. The last phase is the atrial contraction, which also has a negative value and occurs in the last phase of ventricular diastole and during the A wave of the mitral inflow. The RA function has the same phases.

The strain rate is defined as changes in strain with respect to time. Similar to strain, there are 3 peaks in the strain rate curve. The first peak occurs during ventricular systole or the peak strain rate during the reservoir phase. The second peak happens during early diastole and is the peak strain rate during the conduit phase. The third peak occurs in late diastole simultaneous with A wave and is the peak strain rate during the contractile function.[5]

In children and adults with secundum type ASDs, the strain values of the cardiac chambers are in the lower limits of the normal range and are overall lower than those of normal healthy controls.[9-12] Chronic geometrical changes and volume overload in hearts with ASDs lead to the remodeling of the sarcomeres.[6] Sarcomeres are contractile units of the heart whose changes are the mainstay of the alterations seen in strain values. It is difficult to predict the timing of the initiation of these changes; they might be present at birth or progress as an acquired disorder.

In the current study, we detected statistically significant declines in the LA and RA reservoir, conduit, and contractility phase strains and the RV free-wall strain. The LV global longitudinal strain also showed a decrement, which was not statistically significant. The LA has chronically adapted to a low preload before closure. After closure, an increase might be expected in preload, leading to increased strain values; nonetheless, multiple factors affect the LA strain values in this setting.[13] The reservoir function of the LA demonstrates the capacity for expansion during atrial filling or, in other words, LA compliance. As was noted previously, there is baseline atrial remodeling in patients with ASDs, and the disk of the device occupies a part of the LA chamber, affecting the LA reservoir phase and leading to a reduction in the strain value.[3, 6,14] A reduced RA reservoir strain value as an indicator of the RV diastolic function is also diminished due to reductions in preload and chronic structural deformities.[3,6]

Suzuki et al[1] found reductions in LA reservoir, conduit, and contractility phases after device closure and concluded that there was a resistance in the LA conduction of the blood. They also stated that there was an interaction between the device and the atrial bundle integrity, causing LA dysfunction

after the procedure. In accordance with our study, the degree of reduction in the reservoir and conduit phases had an inverse relationship with the ASD size in the investigation by Suzuki and colleagues. Preprocedural decreases in atrial strain values did not have a correlation with ASD size in our study.

Hajizeinali et al[6] compared the LA and RA functions in post-surgical and device closure patients and found a reduction in the reservoir phase of the LA function in the device group, whereas there were reductions in the RA strain values in post-surgical patients, probably due to surgical incisions.

Cakal et al[13] evaluated left-sided strain and strain rates in adult patients with secundum ASDs and found no significant differences between patients with ASDs and normal subjects. Additionally, the LV and LA strain values increased after device closure.

Di Salvo et al[15] evaluated pediatric patients and showed the preservation of the LA and RA functions in the device closure group. However, these studies were performed using older speckle-tracking techniques, which could explain the differences with more recent studies.

In a study by Ozturk et al,[17] RA deformation was assessed. In contrast to our study, the peak reservoir phase had an increase after the procedure. Ozturk and colleagues also reported that the RA reservoir function had an inverse relationship with systolic pulmonary artery pressure and was dependent on preload and afterload.

A recent study published in 2020 reported a reduced RV global longitudinal strain value and an early decline in the LV global longitudinal strain value, demonstrating subclinical LV dysfunction, which increased during follow-up due to improved preload.[16]

Our study was designed to evaluate early, mid, and long-term changes following ASD device closure. Unfortunately, our investigation was interrupted by the COVID-19 pandemic. In addition, follow-up echocardiography for device position was performed by local cardiologists due to travel restrictions and limitations imposed by the COVID-19 pandemic.

CONCLUSIONS

Strain parameters exhibited a decline in our adult patients with ASDs. These parameters were further reduced early following device closure, particularly in ASDs with larger diameters. Such changes may have clinical implications in older patients with large ASDs. The genetic or acquired basis of cardiac functional impairment in ASDs and how medical therapy would alter the abnormalities could be the subject of future research.

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