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Predictors of Left Ventricular Functional Outcome after Percutaneous Closure of Atrial Septal Defect in Adults

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Abstract:

Background:In adults, a secundum form atrial septal defect (ASD) is a popular form of congenital heart defects.An elevation in cardiopulmonary exercise potential was discovered six to twelve months after the ASD was closed.BNP (B-type natriuretic peptide) is a prohormone produced by the myocardium and a rise in ventricular wall tension is the trigger for its release. Both markers have been shown to be extremely susceptible for myocardial stress in a variety of cardiovascular disorders in many studies.Aim of work:To identify the predictors of left ventricular outcome after percutaneous closure of atrial septal defect in adults. Subjects and methods:This study is a clinical trial (pre-post single interventional study) carried out in the cardiology department at Zagazig university hospital. The study included 80 cases admitted during one year. All patients were subjected to complete history taking. General examination for all body systems and local cardiological examination were done. 12 lead ECG, transesophageal and transthoracic echocardiography were done. Blood samples were drawn after a fasting period of 12 hours then we measured BNP in a blood sample before, immediately, after and 3-6 months after procedure. Trans catheter-closure of ASD, with full haemodynamic study was also done.Results:there was asignificant decrease in the area of the RA and the RV with increased LVFP with LV wall thickening, impaired LV diastolic function. And atunivariate and multivariate analysis the results showed that age, ASD size, RV area, RA area, Peak TRPG, mitral E/e', TAPSE appeared to be independent predictors of left ventricular dysfunction.Conclusion:percutaneous closure of an ASD leads to immediate and sustained changes in cardiac anatomy and functions involving both sides of the heart. The NT-pro BNP level begins to increase within 24 hours and continue for 30 days after the procedure in relation to an increase in LV dimension and volume. increased LVFP with LV wall thickening, impaired LV diastolic function.LVFP, however, decreased to the normal range by 6 months after closure in most patients. These findings suggest that after transcatheter closure of an ASD with the removal of a left-to-right shunt, the LV may be subjected to hemodynamic stress, depending on volume overload.Keywords:ASD, BNP,NT-proBNP,Cardiology.

Introduction:

In adults, a secundum form atrial septal defect (ASD) is a popular form of congenital heart defects. ASD closure is usually suggested for symptomatic patients or patients with a significant shunt volume. Surgical closure was once the preferred procedure, and it had been used with good success and a minimal risk for over four decades.**[1]**

Currently, interventional ASD closure is becoming a common alternative to surgery due to its promising results and low incidence of complications. Nonetheless, there have been numerous cases of patients having acute heart failure following surgical closure, necessitating partial reopening of the anomaly in certain patients, as well as case reports of patients having acute heart failure following interventional closure.**[2]**

In line with these observations, an elevation in cardiopulmonary exercise potential was discovered six to twelve months after the ASD was closed, rather than the first sixty days.**[3]**

The definite explanation for acute heart failure is unknown, as is the reason for the slowed improvements after ASD closure.While many studies indicate improvement in right ventricular hemodynamics, there is a lack of data on function of left ventricle in patients with ASDs, as well as hemodynamic alterations in the left ventricle after ASD closure, which lead to a rise in ejection fraction and left ventricular end diastolic volume (LVEDV).**[4]**

BNP (B-type natriuretic peptide) is a prohormone produced by the myocardium (preproBNP). BNP and its N terminal fragment 'NT-proBNP' are excreted in equivalent amounts after cleavage in the circulation. A rise in ventricular wall tension, induced by pressure or volume overload of the ventricles, is the trigger for their release. Both markers have been shown to be extremely susceptible for myocardial stress in a variety of cardiovascular disorders in many studies.**[5]**

Aim and objectives: Toidentify the predictors of left ventricular outcome after percutaneous closure of atrial septal defect in adults.

Subjects and methods:

Technical design:A clinical trial (pre-post single interventional study)carried out in the cardiology department at Zagazig university hospital.The study included 80 cases admitted during one year.Inclusion criteria involved adult patients with secundum ASD fit for percutaneous transcatheter closure. On the other hand, exclusion criteria involved patients with previous stroke, coronary artery disease, pulmonary disease, significant valvular lesion (more than mild), ASD with other forms of congenital heart disease and/or life-threatening arrhythmias and finally ASD larger than 35 mm, as measured by transesophageal echocardiography (TEE).

Methods:All patients were subjected to complete history takingwith stress on age, sex, congenital heart diseases and family history of heart disease.General examination for all body systems and local cardiological examination were done.12 lead ECG, transesophageal and transthoracicechocardiography were done. Blood samples were drawn after a fasting period of 12 hours then we measured BNP in a blood sample before, immediately, after and 3 -6 months after procedure.Trans catheter-closure of ASD, with full haemodynamic study was also done.

Administrative considerations: Written informed consent was obtained from all participantsafter clear explanation of the study and the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University (Institutional Research Board IRB). The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical Analysis:

The results were collected, analyzed, tabulated, and summarized statistically using SPSS v20.0 software for data processing and statistics. Quantitative data were expressed as mean ± standard deviation (SD). Qualitative data were expressed as frequency and percentage.To compare means of two groups, independent sample t test was used when appropriate.The level statistical significance was set at $p \leq 0.001$.

Results:

Table 1 depicts the baseline data of all participates. Collectively; the age ranged from 13 years old to 60 years old with mean of 45.5 ± 16.3years old. We had 49 females (61%). Only 7 cases had chronic AF (9%). As regards, NYHA class 41% were in NYHA class I, 47 % in class II and 11 % were class III.

Peak tricuspid regurgitation (peak TRPG) Right ventricular pressure was (36.6±10.8 mmHg), and mean pulmonary artery systolic pressure (28.66±5.14 mmHg). Qp/Qs ranged from 1.8 to 2.9 with mean of 1.97±0.24. The NT-pro-BNP level was 85.5 ±21.07

The size of ASDs ranged from 10mm to 38mm with mean of 20.6 ± 6.79mm. Moreover, the mean ASD device size (mm) was 23.58 ± 7.3

Table 2 shows that there was statistically significant difference between both groups regardingage ($p< 0.001$). , the younger age associated with significant improvement. Also, there was statistically significant difference regarding NYHA status with improvement of the NYHA functional class after ASD closure ($p<0.001$). Furthermore, there were a statistically significant difference ($p< 0.001$) with respect to BNP, with increase level within 48 hours after closure with some improvement over the 3-6 months follow up. In addition, there was statistically significant difference ($p<0.001$) regarding PR interval with improvement after ASD closure, but there was no improvement of the QRS duration of the RBBB post closure with only improvement of the axis but not the duration .

Table (3)shows echocardiographic findings comparing the effect of ASD closure before, after and 3-6 months follow up. It was found thatthe comparison of baseline measures with those of the follow-up examinations at 48 hours and 3 months after the procedure revealed a significant decrease in the area of the RA and the RV.

Global systolic and diastolic function indices of RV, such as TAPSE decreased immediately after closure and continued to decrease during the 3-6 months follow up.

The degree of TR and the Systolic PAP were significantly after the procedure and At 3 – 6 months.

The area of the LA was not significantly different at 48 hours or 3months when compared with the baseline values.

The LVEDV was greater immediately after the procedure and 3 months after the procedure.

LVEDD and LVESD was greater immediately after the procedure but only the LVEDD was statistically significant, and both increased during the 6 months follow up and both statistically significant .

Also there was significant decrease in MDT in 48 hrs after ASD closure compared to its levels before closure, and significant increase in its level after 3-6 months compared its levels immediately after the operation and 48 hrs after ASD closure On the contrary, there was a significant increase in MITRAL E/e' in 48 hrs after ASD closure (), then continuedecrease over the next 3-6 months after closure.

Among the 80 patients 15 patients showed symptoms and signs of LV dysfunction supported by lab results and NYHA functional class and were prescribed antifailure medications, which showed no or deterioration over the 6 months follow up.

table(4) showing the demographic data affecting the LV function post ASD device closure .

There was no effect of sex of the patients in the outcome of LV function with P value >0.05.

LV dysfunction was observed more in older patients (49.2±17) with a Pvalue <0.001. also was observed in patients with higher Rt ventricular pressure (39 ± 11) with a p value <0.001 , and in patients with large ASD (31.0±4.7) with significant left to Rt shunt , Qp/Qs of 39 ± 11 with a p value of 0.07

On comparison of echocardiography parameters, table (5) shows that

Pre-procedural lateral E/e' was significantly higher (11.58 ± 4.80) in patients, who developed LV dysfunction compared with patients, who had preserved or improved left ventricular function. Also, MAPSE (25.9± 3.1), TAPSE (26.9± 2.8) were significantly increased, whilst RA area (16.1±3.3), RV area (16.1±3.3), peak TRPG (32.7±9.3) were significantly lower in patients with good left ventricular dysfunction compared with those, who developed LV dysfunction. The severity of tricuspid regurgitation changed significantly between both groups at 6 months of follow-up.

Table (6) depicts the univariate and multivariate predictors of left ventricular dysfunction after percutaneous device closure of ASD. At univariate analysis the results showed that, age, ASD size, RV area, RA area, Peak TRPG, mitral E/e', TAPSE appeared to be independent predictors of left ventricular dysfunction. Whilst, at multivariate analysis age, ASD size, RV area, Peak TRPG, mitral E/e', and TAPSE remained independent predictors of left ventricular dysfunction 6 months after percutaneous closure of ASD.

Table (1): Demographics and Clinical Data of all patients

Female N (%) Males N (%)		49(61.25%) 31(38.75%)	
Age at time of device closure of ASD (yrs)		45.5 ± 16.3	
Weight (kg)		68.8±10.7	
Height (cm)		164±9.6	
Body surface area (m^2)		1.63 ± 0.51	
NYHA functional class			
I		33(41.25%)	
II		38 (47.5%)	
III		9 (11.25%)	
Atrial fibrillation		7 (9%)	
Peak TRPG (mmHg)		36.6±10.8	
Pulmonary hypertension (RVSP ≥50 mmHg by TTE)		19 (%)	
Qs/Qp		1.97±0.24	

NT-pro-BNP		85.5 ±21.07	
ASD diameter			
Echo (mm)		20.6 ± 6.79	
Stretch balloon size (mm)		21.1 ± 6.4	
ASD device size (mm)		23.58 ± 7.3	

Table (2): Clinical and electrocardiographic measurements before percutaneous atrial septal defect and 6 months after ASD closure.

	Before Device Closure	48 hours After Device Closure	6 months After Device Closure	p-value
NYHA				
Class I	42 (52.8)	44(55.6)	71(88.9)	<0.001
Class II	37(47.2)	15(19.4)	9(11.1)	
Class II		20(25)		
AF	7	6	3	<0.001
NT-proBNP, pmol/L	52.81±21.07	130.05±96.52	92.05±60.63	<0.001
Electrocardiography				
PR-interval ms	175 ± 23	170 ± 25	157 ± 19	<0.001
QRS duration ms	98 ± 12	98 ± 12	97 ± 10	0.5

Table (3): echocardiographic findings comparing the effect of ASD closure before , after and 3-6 months follow up .

	before	within 48hrs	(3-6 months)	P value 48 versus before	P value (3-6months) versus before	P value (3-6 months) versus 48 hrs after
LVEF	62.15±3.26	62.5±3.28	62.15±3.11	0.009	0.9	0.009
LVED	40.74±2	42.1±2.06	43.29±2.25	<0.001	<0.001	<0.001
LVESD	26.53±1.22	26.76±1.41	28.76±1.66	<0.001	<0.001	<0.001
LVEDV	73.33±8.7	79.24±9.2	84.6±10.5	<0.001	<0.001	<0.001
LA diameter	31.96±2.75	31.99±2.77	31.96±2.76	0.2	0.9	0.18
MAPSE	23.16±2.15	21.99±2.75	25.26±2.96	<0.01	>0.05	<0.001
RV area	21.13±4.3	16.9±4.19	15.26±3.91	<0.001	<0.001	<0.001
RA area	17.61±4.21	16.51±3.6	12.51±3.05	>0.05	<0.01	0.01
MDT	231.03±46.77	222±51.2	233.92±48.32	<0.001	0.001	<0.001
MITRAL E/e’	7.27±1.41	7.81±1.63	7.58±1.53	<0.001	<0.001	<0.001

TAPSE	21.97±2.4	19.66±2.18	18.52±2.97	<0.001	<0.001	<0.001
mPA	19.5±3	18.9±2.27	19.4±1.67	>0.05	<0.001	<0.001
Systolic PAP	39.65±5.18	33.22±2.93	23.14±2.20	>0.05	<0.001	<0.001
TR						
None or trace	11 ()		26 ()			<0.001
Mild	28 ()		43 ()			<0.001
Moderate	29 ()		13 ()			<0.001
Severe	12 ()		3 ()			<0.001

Table (4): Demographic data of patients with improved versus those with without improvement after percutaneous closure of ASD

Characteristics	Good function group (n=65)	LV dysfunction group (n=15)	P value
Sex			>0.05
Male	26	5	
Female	39	10	
Age (years)	31.7±9.5	49.2±17	<0.001
Device s ize (mm)	16.5±3.2	31.0±4.7	<0.001
Qp/Qs	1.9±0.3	2.3± 0.3	0.07
RVP (mmHg)	23±9	39 ± 11	<0.001
NT-proBNP (pg/mL)			
Pre	61±18.15	89±21.56	<0.005
6 months Post	51.05±16.63	203±36.33	<0.001
p value	>0.05	<0.001	

Table (5): Basic echocardiographic characteristics of patients with good versus those with left ventricular dysfunction after percutaneous closure of ASD

Parameter	Good function group (n=65)	LV dysfunction group (n=15)	P value
LVEDD (mm)	40.06 ± 3.28	43.39 ± 3.52	0.001
LVESD (mm)	26.86 ± 2.98	27.79 ± 3.11	0.11
LVEF (%)	63.41 ± 5.56	61.69 ± 5.08	0.48
LA volume (mL)	35.83 ± 15.67	37.48 ± 14.57	0.09
E/e’	8.79 ± 3.19	11.58 ± 4.80	0.005
MAPSE	25.9± 3.1	22.6± 2.2	<0.01
TAPSE	26.9± 2.8	22.1± 2.5	<0.001
RV area	25.8±2.9	30.5±3.12	<0.001
RA area	16.1±3.3	20.6±3.3	<0.001
Peak TRPG	32.7±9.3	39.25 ±12.5	<0.001
mPAP (mmHg)	31.8 ± 11.6	36.5 ± 12.20	<0.01

*p≤0.001 is significant.

Table (6): univariate and multivariate analysis for demographic , neurohormonal and echocardiographic variables to indentify predictors of LV dysfunction post ASD device closure

	Univariate		Multivariate	
	OR (95% CI)	P value	OR (95% CI)	P value
age	0.75 (0.36-0.92)	<0.01	0.85 (0.79-96)	<0.03
ASD size	1.89 (1.12-2.57)	<0.01	1.55 (1.17-3.35)	<0.03
NT- pro-BNP	0.51(0.82-1.41)	>0.05	--	--
LVEF	0.88(0.12-1.17)	0.737	--	--
LVEDD	2.04(0.9-5.01)	0.16	--	--
LVESD	1.24(0.48-1.67)	0.243	--	--
LVEDV	0.41(0.23-1.06)	0.192	--	--
LA	0.11(0.05-1.27)	0.184	--	
MAPSE	5.6 (3.25-7.31)	0.08	0.97(0.35-1.16)	0.45
RV area	1.33 (1.11-2.49)	<0.001	1.12 (1.05-2.07)	<0.01
RA area	2.19(1.166-3.15)	<0.001	1.22(0.25-2.19)	>0.05
Peak TRPG	1.83 (1.03–1.90)	<0.003	1.59 (1.01–1.55)	<0.05
Mitral E/e’	1.59 (1.15–2.17)	0.002	1.53 (1.12–2.29)	<0.01
TAPSE	1.95(1.37-3.11)	<0.01	1.73(1.43-3.85)	<0.02
Systolic PAP	0.65(0.21-0.85)	<0.05	0.18(0.03-1.67)	>0.05

Discussion:

ASD is a form of congenital heart defects that allows contact between the heart's left and right sides. Numerous irregularities in the cardiac veins terminations as well as the interatrial septum contribute to interatrial communications.**[6]**

According to Opotowsky and colleagues, pulmonary vascular resistance is elevated at birth and right ventricular performance is poor. Left to right shunt is the most common haemodynamic feature in secundum atrial septal defect, occurring often throughout late ventricular systole and early diastole and rising throughout atrial contraction and expiration.**[7]**

B-type natriuretic peptide (BNP), also known as ventricular natriuretic peptide, is a 32-amino-acid polypeptide naturally produced by the heart's ventricles in consequence of the increase stretching of cardiac muscles.**[8]**

Our study was aiming to identify the predictors of left ventricular outcome after percutaneous closure of atrial septal defect in adults via assessing the effect of ASD closure on LV function and estimation of the level of NT-proBNP before and after ASD closure.

Considering our results, we analyzed the level of NT pro BNP among the studied ASD patients, and our results showed that there is a significant increase in NT-proBNP in the first 48 to 72 hrs after ASD closure, then significant decrease in its level within 3-6 months. We also showed that despite NT-proBNP it does not reach pathological value especially in moderate size ASD but increases early after interventional closure with a return to normal values during further follow up. The

increase of NTproBNP was associated with an increase in left ventricular dimensions and an improvement in function as shown by echocardiography.

Our findings were consistent with those of Weber and colleagues, indicating that eliminating the left-to-right shunt after ASD closure improves left ventricular filling, resulting in increased left ventricular measurements and improved left ventricular efficiency.A rise in NT-proBNP concentrations shortly following ASD closure, followed by a return to normal concentrations late after ASD closure, accompanied these haemodynamic modifications.**[9]**

During our study, we found significant increase in LVEDD and LVEDV in the first 48 to 72 hours after ASD closure compared to its levels immediately after closureand continue to increase over the next 3 to 6 months but were within the normal range according to age. LVEDV unadjusted and adjusted for body surface area (BSA) at baseline measured by MRI were observed to be lower than known normal values, according to Lorenz and colleagues. From baseline to early follow up, we observed an improvement in LVEDV and LVEDD, as well as their respective BSA-adjusted values. Nevertheless, from early to late follow-up, the values stayed constant.**[10]**

Our study demonstrated that there was a significant decrease in MDT in 48 hrs after ASD closure compared to its levels immediately after closure, and significant increase in its level after 3-6 months compared its levels immediately after the operation and 48 hrs after ASD closure. On the contrary, there was a significant increase in MITRAL E/e' in 48 hrs after ASD closure, then continue decrease over the next 3-6 months after closure. E/e' and color M-mode velocity of propagation, indices of relaxation, showed no major changes, according to Gomez and colleagues.**[11]**

Another research, on the other hand, found a reduction in e' only after or 24-48 hours after ASD closure. Six months or more after the operation, the decreased E/e' returned to its pre-closure stage.**[12]**

Numerous variables, including pre-closure relaxation status, age, and post-closure heart rate, blood pressure, or geometric changes in both the LV and the RV, may explain the contradictory findings. The impact of volume load, which is commonly associated with systolic pressure increase, could theoretically delay the relaxation process. **[13]**

We also found during our study a significant decrease TAPSE within 48 hrs after ASD closure, and significant decrease in its level within 3-6 months. The measures of RV (i.e., TAPSE and basal systolic tissue Doppler velocity) have been observed to decrease dramatically throughout 24 hours of closure and to continue to drop over the following six to eight weeks, according to Monfredi and colleagues.**[14]**

In our study there was also a significant decrease in RV area in 48 hrs after ASD closure compared to its levels immediately after closure, and significant decrease in its level after 3-6 months compared its levels immediately after the operation and 48 hrs after ASD closure. The diameters of the RV and RA were both reduced after the early phase of transcatheter closure of ASD, according to Agac and colleagues. **[15]**

In our study there was 30 patients above the age of 40 years old , they had higher LV indices before closure compared to younger patients , 13 patients were older than 55 years old they even had higher indices , in the form of increased LVED, MITRAL E/e' and LVEDV before ASD closure . Our results demonstrated higher LVED, MITRAL E/e' and LVEDV in patients >40 years compared to those <40 years immediately after ASD closure and 48hrs and 3-6 months but were within the high normal range (p value <0.001).

Makino and colleagues studied 40 patients who underwent transcatheter ASD closure. The average age was 33.1±12.0 in ASD younger group, 66.4±9.0 in ASD older group, 36.8±8.0 in healthy younger group and 68.1±9.8 in healthy older group. There was no significant difference between ASD closure group and healthy control group in all parameter of left ventricular diastolic function These results suggested that the left ventricular diastolic function in patient after ASD closure is same as healthy subjects ***(Makino et al., 2018).***

Echo findings showed LV dysfunction in the form of increase of LVEDD , LVESD and LVEDV , also diastolic dysfunction showed by impaired mitral; E/A and tissue Doppler mitral E/e' , all this were associated with increase of NT pro BNP immediately after the procedure which continued to increase over the next 3 -6 months follow up .

Also ***Masutani et al 2009*** reported that 2 of 39 consecutive patients who underwent transcatheter ASD closure developed clinical heart failure. Another study ***Takaya Y el al 2017*** divided 206 patients >60 years old into 3 groups depending on LV diastolic function and assessed outcomes after transcatheter ASD closure; more than half of the patients with severe diastolic dysfunction were prescribed diuretics prior to the procedure, and plasma BNP level did not increase after the procedure in these patients. Pre-procedural prescription of diuretics might prevent the development of clinical heart failure.

Also ***Schubert et al 2005*** reported that hemodynamic measurement during complete balloon occlusion of ASD could be helpful for predicting LVFP elevation following closure. In their study, 15 of 59 patients (25%) had LV restriction defined as an increase of LA pressure >10mmHg. Of these 15 patients, 13 received anti-heart failure pre-medication; subsequently, catheter ASD closure was successfully performed. The remaining 2 patients who underwent ASD closure without pre-medication had congestive heart failure requiring additional therapy. Baseline LA pressure and the prevalence of pulmonary hypertension and atrial fibrillation were higher in patients with LV restriction than in those without.

On the other hand these patients also had higher NT pro BNP post closure which was more related to the diastolic function (mitral E/e', and MDT).

And these result were consistent with the result ***Redfield et al. (2005)*** which stated that long-standing underloading superimposed on the aged stiff ventricle may cause a marked rise in intrinsic LV diastolic stiffness in some elderly ASD patients, which could be unmasked by the acute increase in LV preload following closure of the defect.

Also ***Masutani et al. (2009)*** we demonstrated previously that e' before ASD closure and age are independent predictors of brain natriuretic peptide (BNP) levels after ASD closure, and that two patients with the lowest values of e' presented with heart failure-related symptoms.

In addition, RV reverse remodeling with the defect closure may not be uniform and could affect LV systolic and diastolic function through RV/LV interaction. Furthermore, aging is associated with ventricular systolic and vascular stiffening [41, 42], in addition to ventricular diastolic stiffness [41]. These changes can interact to cause relaxation delay and EDP rise [43] after ASD closure in elderly patients.

In the present study, patients with LV dysfunction had significantly larger ASD than those without. This is consistent with other variables, that is, greater maximum ASD size, device size used, and the ratio of pulmonary to systemic blood flow.

This was consistent with the result of ***MichiyoYamanoet,al*** which also found same result .

However, maximum ASD size was not selected as an independent predictor , which is consistent also with the result found by ***Schubert et al***

On our multivariate analysis for predicting post-procedural LV diastolic dysfunction these factors were identified as pre-procedural ASD size, RV area, Peak TRPG, mitral E/e', and TAPSE remained independent predictors of left ventricular dysfunction 6 months after percutaneous closure of ASD.

These results were similar to the results by ***MichiyoYamanoet,al 2020*** that on multivariate analysis for predicting post-procedural LVFP, these factors were identified as pre-procedural LV RWT, lateral E/e', and peak TRPG. Peak TRPG, used practically as an indicator of systolic pulmonary artery pressure.

Conclusion:

Percutaneous ASD closure causes immediate and long-lasting modifications in cardiac anatomy and function on both sides of the heart. NT-proBNP, a neurohormonal marker representing myocardial discomfort, is not elevated in patients with a moderate-sized ASD. However, after interventional ASD termination, NT-proBNP concentrations rise significantly within days, followed by an improvement in left ventricular measurements and a fall to basal concentrations during 4 months.

Percutaneous closure of an ASD leads to immediate and sustained changes in cardiac anatomy and functions involving both sides of the heart. The NT-pro BNP level begins to increase within 24 hours and continue for 30 days after the procedure in relation to an increase in LV dimension and volume. increased LVFP with LV wall thickening, impaired LV diastolic function.LVFP, however, decreased to the normal range by 6 months after closure in most patients. These findings suggest that after transcatheter closure of an ASD with the removal of a left-to-right shunt, the LV may be subjected to hemodynamic stress, depending on volume overload.

Age, ASD size, RV area, RA area, Peak TRPG, mitral E/e', TAPSEcan be considered as independent predictors of left ventricular dysfunction.

Recommendations:

We recommend considering natriuretic peptide as a valuable tool that can be used in the pre-and postoperative follow up of patients with ASD who underwent interventional closure via continuous assessment and monitoring of its level. We also recommend proper assessment of LV systolic and diastolic function in elderly patients undergoing ASD closure.

the mechanisms responsible for the development of heart failure after ASD closure are more likely to be multifactorial rather than involving diastolic dysfunction alone, and should be clarified in future studies to secure the safety of this procedure.

To use MRI and more advanced echo techniques like speckle tracking to properly assess the ventricular function , systolic and diastolic , to predict worsening of ventricular function post ASD closure .

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