Zagazig University Medical Journal
www.zumj.journals.ekb.eg

https://doi.org/10.21608/zumj.2024.327869.3637 Volume 31, Issue 1.1, JAN. 2025, Supplement Issue

Manuscript ID ZUMJ-2410-3637
DOI 10.21608/zumj.2024.327869.3637

ORIGINAL ARTICLE

Biventricular Diastolic Functions Before and After Interventional Occlusion of Interatrial Communication

Mohamed Osman Hafez [1], Heba AbouZeid [1], Yomna Refaat Abdel-Hafeez [2]*, Salah Elsayed Ibrahim [3]

[1]Pediatric Cardiology Unit, Pediatrics Department, Faculty of Medicine ,Zagazig University, Zagazig, Egypt

[2] Pediatrics Department, Faculty of Medicine ,Zagazig University, Zagazig, Egypt

[3] Clinical Pathology Department, Faculty of Medicine ,Zagazig University, Zagazig, Egypt

***Corresponding author:**
Yomna Refaat Abdel-Hafeez

Email:
yomnarefat91@gmail.com

Submit Date 13-10-2024
Revise Date 07-11-2024
Accept Date 10-11-2024

ABSTRACT

Background: Interventional closure of an interatrial communication (IAC) can result in early and/or persistent differences in cardiac physiology and structure with changes in right heart size and function secondary to altered cardiac hemodynamics. We aimed to assess initial consequences of catheter-based closure of IACs on diastolic function of both ventricles.

Methods: This prospective study included 68 children. Study participants were divided into patients with IAC and a control group with normal intracardiac anatomy (n = 34 in each group). Transthoracic Echocardiographic evaluation was performed before and one week after IAC occlusion.

Results: The peak tricuspid inflow E and A wave velocities, mitral E and A wave maximal inflow velocities, and estimated pulmonary artery systolic blood pressure were 78.12 ± 18.17 , 60.31 ± 19.06, 84.50 ± 14.52, 67.96 ± 21.29 cm/sec and 28.67 ± 5.91 mmHg in the cases compared with 80.14 ± 27.77, 65.38 ± 41.9, 94.73 ± 34.96, 59.47 ± 19.42 cm/sec and 27.94 ± 3.44 mmHg in the control group (all p values > 0.05). left atrial and ventricular areas were smaller in cases compared with control group at baseline. One week after transcatheter occlusion of IAC, peak tricuspid E and A wave velocities as well as pulmonary artery systolic pressure were significantly reduced (78.12 ± 18.17, 60.31 ± 19.06 cm/sec and 28.67 ± 5.91 mmHg vs. 67.75 ± 13.43, 49.27 ± 14.27 cm/sec and 25.74 ± 4.58 mmHg, p values = 0.009, 0.009 and 0.026).

Conclusions: LA and LV areas were smaller in cases compared with healthy children pre-intervention which points to sizable IACs which allow shunting of blood flow away from LA and LV in our patients. A significant reduction in early and late peak tricuspid inflow velocity was observed in our patients after percutaneous IAC occlusion. This reduction in RV inflow velocity may be due to the reduction of RV volume overload after the intervention.

Keywords: Biventricular Diastolic Function ;Interatrial Communication ;Interventional Occlusion

INTRODUCTION

Interatrial communication (IAC) is a common congenital heart defect .Percutaneous IAC occlusion has now become a preferrable technique for management of most cases of secundum IAC [1][2]. A variety of devices have been designed to percutaneously close IAC .Large IACs may present with symptoms due to excess volume loading of the right atrium (RA) and right ventricle (RV) [3][4].

After IAC closure, blood volume changes rapidly. Previous studies have shown that IAC occlusion can cause right heart volume reduction secondary to reduced shunting from left atrium (LA) to RA [5]. After interventional IAC occlusion, there are inconsistencies regarding the maintenance of RV and RA functional activity [6]. Transthoracic Echocardiography (TTE) can usually confirm the presence of IAC, which is best seen in the subcostal view. However, "Echo loss" in the interatrial septum is often seen where contrast bubble studies improve the diagnostic accuracy of Echocardiography. TTE can reveal secondary hemodynamic consequences of IAC, such as RA dilatation, RV dilatation/insufficiency, tricuspid annular dilatation, tricuspid regurgitation, and pulmonary hypertension [7]. In previous studies, the effect of interventional IAC closure on left ventricular function was not clearly defined [6].Our objective was to appraise early outcomes of interventional secundum IAC occlusion on right and left ventricular function during diastole.

METHODS

This prospective study comprised 68 children divided into 2 groups; patients with secundum IAC and controls without structural heart disease (n = 34 in each group). Participants were recruited from May 2021 to May 2024. Study participants were diagnosed and managed at the Unit of Pediatric Cardiology at Zagazig University Hospital, Egypt. In our study, we followed the guidelines of World Medical Association's Code of Ethics (Declaration of Helsinki) for research in humans. Parents of all participants gave written consents for their involvement. Our institutional review board permitted this study (IRB#7019/31-8-2021).

Inclusion criteria inculde Children with secundum IAC requiring occlusion; significant left-to-right blood flow (flow ratio more than 1.5-1), presence of complaints, or evidence of significant shunt by Echocardiography.

Exclusion criteria inculde Primum IAC, partially abnormal pulmonary venous drainage, pulmonary vascular resistance exceeding 4.6 Wood units, or more than two-thirds of systemic vascular resistance after reversibility testing, right-to-left shunt with an arterial oxygen saturation below 95% in room air, a concomitant cardiac defect necessitating surgery, diseases of coronary arteries, moderate to severe disease of cardiac valves, and compromised function of LV with an ejection fraction of 55% or lower on TTE.

In our study, we included thirty-four patients with secundum IAC. The cases were identified through Echocardiogram at the Pediatric Cardiology Unit of the Pediatric Department at Zagazig University Hospital in Egypt. Thirty-four subjects of similar sex and age to those of patients were selected as control group. Upon examination, the control children showed no unusual findings in their general and local physical examination. Their intracardiac anatomy was determined to be normal based on TTE.

The entire study population was subjected to comprehensive history collection encompassing personal history, presenting complaints, current and previous medical histories, family medical backgrounds, reason for admission, disease onset and duration, medical treatments, and complications. We evaluated the participants' body measurements and looked for any general physical abnormalities. All individuals underwent assessment of vital signs, physical examinations including cardiac evaluation, and Echocardiography.

Echocardiography:

All participants, including both patients and controls, underwent an Echocardiographic evaluation. Echocardiography was carried out using an Epiq CVx Release 6 Philips machine with simultaneous ECG monitoring. We utilized either S 8-3 or X5-1 megahertz probes. Measurements such as LV systolic and diastolic dimensions, Aortic and LA dimensions and fractional shortening of LV were obtained using M-mode Echocardiography from parasternal window in short-axis views of the heart in agreement with the commendations of American Society of Echocardiography [8].

Diastolic mitral and tricuspid valves inflow: We positioned Pulsed Wave Doppler sample volume of 3 mm between each valve leaflet tip in apical four-chamber view. Early (E) and late (A) diastolic inflow velocity peaks were estimated twice for each of the two atrioventricular valves. For statistical analysis, the average of the two recordings was used for each peak velocity [9].

Systolic pulmonary artery pressure (sPAP): Was measured using modified Bernoulli equation. The peak velocity of the tricuspid valve regurgitation (TR) was determined by CW-Doppler inspection. The RV systolic pressure (RVSP) is the same as the sPAP in absence of right ventricular outflow tract obstruction. To calculate RVSP, right atrial pressure (RAP) was added as follows:

$RVSP = 4 \times [TR\, peak\ \ velocity\, (m/\text{sec})]^2 + RAP$ [10, 11].

Patients' Echocardiographic parameters were assessed prior to and one week after IAC interventional occlusion. Echocardiography was done only at baseline in the control group.

All patients had normal sPAP measured by Echocardiography so, invasive measurements of RA, LA, RV, LV pressures and PVR were not indicated at the time of IAC closure. IAC closure was done only if RA and RV were dilated. As dilation of RA and RV highlights the hemodynamic significance of IAC, pulmonary to systemic flow (Qp:Qs) ratio was not estimated. We indirectly estimated shunt magnitude by calculation of cardiac chamber areas at baseline. LA area calculations were done in apical views at end-systole. RA area estimations were attained in an apical four-chamber view just before the tricuspid valve opens. Right ventricle end-diastolic (RVED) area was assessed by outlining of inner endocardial border of RV at tricuspid valve closure. Left ventricle end-diastolic (LVED) area was measured by demarcation of internal endocardial border of LV at mitral valve closure. Right ventricle end systolic (RVESD) area was assessed by tracing of inner endocardial border of RV at the frame that precedes tricuspid valve opening. Left ventricle end-systolic (LVESD) area was calculated by outlining of internal endocardial border of LV at the frame just preceding mitral valve opening.

Diastolic mitral valve tissue Doppler velocities: We used Pulsed Wave tissue Doppler to quantity È (early diastolic) wave peak velocities of mitral valves. Tissue Doppler sample volume of 3 mm was located at the medial and lateral annular aspects of mitral valve. The average of two values for È peak velocity in each patient was obtained and used in statistical analysis.

Statistical analysis

Data were transferred to the Statistical Package for the Social Sciences, version 23 (IBM SPSS Statistics for Windows, version 23.0. Armonk, NY: IBM Corp.). Means±standard deviations, medians, Chi square, unpaired student's t, Mann Whitney U, two way analysis of variance tests were used when suitable. Level of significance was considered below 0.05.

RESULTS

Age, gender, height, weight, and body mass index were homogenously distributed among individuals **(Table 1**). The left atrial, main pulmonary artery and its branches diameters, IV septal diameter, and LV posterior wall thickness did not varyand controls. On the contrary, notable reduction was found in cases with respect to LVIDd, (LVIDs). On the other hand, fractional shortening (FS) of LV was higher in cases (Table 2).

There were no statistically meaningful changes in the analyzed groups for Tricuspid E, Tricuspid A, Mitral E, Mitral A peak wave velocities, and sPAP **(Table 3)**.

Regarding Comparison of cardiac areas, there were no statistically important changes regarding RA and RV areas. LA and LV areas were smaller in cases compared with control group **(Table 4).**

When comparing the case group's mitral E and mitral A wave peak inflow velocities before and one week after the IAC interventional occlusion, no statistically significant alterations were seen. Substantial differences were seen in the estimated pulmonary artery systolic pressure and Tricuspid E and A waves between the two groups. We reported no statistically important difference between the case group before and one week following IAC transcatheter occlusion regarding Mitral È **(Table 5).**

Table (1): Demographic and anthropometric factors in studied groups.

	Case group (N=34)	Control group (N=34)	P-value
Age(years)	5 (3-14)	6 (3.5-12)	0.6329
Gender			
Male	16	20	0.3311
Female	18	14	
Weight(kg)	19.25 (13-40)	22 (14-42)	0.389
Height(cm)	116.04 ± 15.71	118.47 ± 14.30	0.5071
Body mass index(kg/m²)	15.74 ± 1.65	16.15 ± 1.15	0.2388

Table (2): Cardiac dimensions and LV systolic function in studied groups.

	Case group (N=34)	Control group (N=34)	P-value
Aortic diameter(mm)	23.07±3.15	21.23±3.82	0.0339*
Left atrial diameter(mm)	22.54±2.25	23.54±4.29	0.2330
Main pulmonary(mm)	18.56±2.61	17.46±3.69	0.1606
Left pulmonary(mm)	8.97± 2.3	8.65± 1.73	0.5190
Right pulmonary(mm)	9.28± 2.39	9.17± 2.07	0.8399
LV Posterior wall(mm)	6.25±1.01	6.23±0.64	0.922
IV septum(mm)	6.57±0.76	6.25±0.63	0.0631
LVIDd(mm)	34.48±5.42	42.40±5.63	0.0001*
LVIDs(mm)	20.20± 3.05	27.67± 4.10	0.0001*
FS %	40.31±8.81	36.20±5.55	0.0245*

FS; Fractional shortening, LV; left ventricle, LVIDd; Left ventricle internal diameter at end diastole, LVIDs; Left ventricle internal diameter at end systole, IV septum; inter-ventricular septal thickness.

Table (3): Echocardiographic parameters in studied groups.

	Case group (N=34)	Control group (N=34)	P-value
Tricuspid E (cm/sec)	76.6 (49.6-128)	67.9 (51.4-152)	0.7238
Tricuspid A (cm/sec)	53.5 (31.7-99.1)	45.6 (27.4-152)	0.5229
Mitral E(cm/sec)	85.65 (34.5-125)	106(10.6-132)	0.1199
Mitral A(cm/sec)	65.9 (25.9-101.2)	64.3(10.6-124)	0.0905
Pulmonary arterial systolic pressure(mmHg)	28.67 ±5.91	27.94 ± 3.44	0.5358

Mitral A; Mitral late inflow diastolic peak velocity, Mitral E; Mitral early inflow diastolic peak velocity, Tricuspid A; Tricuspid late inflow diastolic peak velocity, Tricuspid E; tricuspid early inflow diastolic peak velocity.

Table (4): Comparison of cardiac areas in studied groups at baseline.

	Case group (N=34)	Control group (N=34)	P-value
RA area(cm^2)	9.75±2.10	9.82±2.07	0.8903
LA area(cm^2)	7.54± 1.71	9.10± 2.13	0.0014*
RVED area(cm^2)	13.89±3.63	14.67±4.38	0.4269
RVES area(cm^2)	10.15±3.32	11.02±2.61	0.2340
LVED area (cm^2)	13.7 (10.3-27.5)	18.465 (10.2-29.4)	0.0043*
LVES area(cm^2)	9.87 (4.84-20.6)	13.7 (7.52-27.1)	0.0003*

LA; left atrium, LVED: Left ventricle end-diastolic, LVES: left ventricle end-systolic, RA; right atrium, RVED; Right ventricle end-diastolic, RVES; Right ventricle end-systolic.

Table (5): Echocardiographic parameters in case group before compared with one-week post-IAC interventional occlusion.

	Case group before (N=34)	Case group after (N=34)	P-value
Tricuspid E (cm/sec)	76.6 (49.6-128)	71.85 (39.7-89.2)	0.0094*
Tricuspid A (cm/sec)	53.5 (31.7-99.1)	45.55 (33.3-78.3)	0.0087*
Mitral E (cm/sec)	85.65 (34.5-125)	89.35 (13.1-132)	0.3353
Mitral A (cm/sec)	65.9 (25.9-101.2)	62.1 (30.5-132)	0.9905
Mitral È(cm/sec)	13.80 ± 2.87	13.23± 2.49	0.3849
Pulmonary arterial systolic pressure(mmHg)	28.67 ±5.91	25.74± 4.58	0.0255*

Mitral A; Mitral late inflow diastolic peak velocity, Mitral E; Mitral early inflow diastolic peak velocity, Tricuspid A; Tricuspid late inflow diastolic peak velocity, Tricuspid E; tricuspid early inflow diastolic peak velocity.

DISCUSSION

Interatrial communication occurs in 1.64 per 1,000 live births. Long-term left-to-right shunt through the interatrial communication can cause adverse changes in cardiac structure and function. Cardiac remodeling includes right heart dilatation, right sided heart failure, atrial arrhythmias, and increased pulmonary vascular resistance [12].

Noninvasive Echocardiography assesses left ventricular diastolic function of LV. Accurate assessment of left ventricular diastolic function is significant in cases of preserved and reduced ejection fraction of LV. Tissue Doppler is suitable for demonstration of altered LV relaxation patterns. Tissue Doppler examination is not always necessary in patients with reduced LVEF, as those patients usually have impaired LV relaxation and therefore a significant increase in circulating volume will result in increased LV filling pressures secondary to changes in compliance of LV. Therefore, in cases of reduced EF, trans-mitral flow velocities and deceleration time, left atrial volume, pulmonary venous Doppler, and estimated sPAP are sufficient to accurately assess LV filling pressure [13, 14]. Our study revealed no significant variances between the two study groups in dimensions of LA, MPA, LPA, RPA, IV septum, and LV posterior wall. In contrast, we detected significant reduction in LVIDd, LVIDs, and increase of fractional shortening (FS) in our cases, as shown in Table 2. Mean normal value for FS was reported to be 36% with 95% prediction limits of 28% to 44% [19]. Although FS was higher in our cases compared to control group but both groups had their FS within the normal range. Kılıçaslan et al. [16] did not find significant changes between the IAC and control groups in ejection fraction of LV but they reported that left ventricle end diastolic volume, LV end systolic volume, and LV stroke volume increased at follow-up 24 hours and 30 days after the closure of IACs. Dehn et al. [17] reported no significant variances between the IAC and control groups in MPA diameter, LVIDd, LVIDs. They also reported that LV function did not differ between cases with IAC and controls.

Park [19] reported that IAC is not accompanied by LA or LV enlargement because the increased pulmonary blood flow to LA does not stay in left sided chambers as it is shunted immediately to RA through the IAC. In this study, LA and LV areas were smaller in cases compared with healthy children pre-intervention which indicates a high magnitude of left to right shunting diverting the blood flow away from LA and LV (table 4).

Our study showed no significant changes between the studied groups in peak E and A waves of tricuspid valve, mitral E wave, mitral A wave, and sPAP (Table 3). Our results were in harmony with those of Karunanithi et al. [20], who reported no significant differences between the IAC and control groups in estimated sPAP. Our study showed no significant variances between the case groups before and one week after interventional IAC occlusion regarding peak mitral E or A wave inflow velocities. In contrast, we found statistically significant differences in peak tricuspid E and A wave velocities as well as estimated sPAP; A significant reduction in the peak entry velocity of the tricuspid E and A waves was observed in our cases after intervention but tissue Doppler-derived mitral È velocity peak did not vary significantly between both groups (Table 5). Our results were consistent with those of Kılıçaslan et al. [16] who reported that there were no significant variabilities between the case groups before 24 hours, and 30 days after intervention in E and A wave mitral velocities. Similarly, our results were consistent with those of Song et al. [18] who revealed no significant alteration between the case groups before and after IAC occlusion in mitral valve E or A wave velocities.

Study Limitations: Sample size of 68 participants may be fairly small, which may affect the accuracy of results. A larger study population would have yielded stronger inferences and allowed subgroup analyses to explore the early effects of IAC occlusion on atrial and ventricular dimensions and diastolic function. Our study was performed at a single center, that may limit the hypothesis of reaching similar conclusions in other populations or health care settings.

CONCLUSIONS

LA and LV areas were smaller in cases compared with healthy children pre-intervention which points to sizable IACs which divert blood flow away from LA and LV in our patients. Significant reductions in peak tricuspid E and A inflow velocities were observed in our cases after percutaneous interatrial communication occlusion. This reduction may be due to reduction of RV volume overload secondary to interventional IAC closure.

Conflict of Interest :None

Financial disclosure: None

REFERENCES

1. Mezawa H, Tomotaki A, Yamamoto-Hanada K, Ishitsuka K, Ayabe T, Konishi M, et al. Prevalence of Congenital Anomalies in the Japan Environment and Children's Study. J Epidemiol. 2019;29(7):247-56.
2. Bacha E. Transcatheter Versus Surgical Closure of Atrial Septal Defects. JACC CardiovascInterv 2016;(9):87−8.
3. Oremus M, Don-Wauchope A, McKelvie R, Santaguida PL, Hill S, Balion C, et al. BNP and NT-proBNP as prognostic markers in persons with chronic stable heart failure. Heart Fail Rev 2014;(19):471−505.
4. Fang F, Luo XX, Lin QS, Kwong JS, Zhang YC, Jiang X, et al. Characterization of mid-term atrial geometrical and electrical remodeling following device closure of atrial septal defects in adults. Int J Cardiol 2013;(168):467−71.
5. Badano LP, Kolias TJ, Muraru D, Abraham TP, Aurigemma G, Edvardsen T, et al. Standardization of left atrial, right ventricular, and right atrial deformation imaging using two-dimensional speckle tracking Echocardiography: a consensus document of the EACVI/ASE/Industry Task Force to standardize deformation imaging Eur Heart J Cardiovasc Imaging. 2018; 19(7):830-33
6. Voigt JU, Pedrizzetti G, Lysyansky P, Marwick TH, Houle H, Baumann R, et al. Definitions for a common standard for 2D speckle tracking echocardiography: consensus document of the EACVI/ASE/Industry Task Force to standardize deformation imaging. J Am Soc Echocardiogr 2015; (28):183−93.
7. Alkashkari W, Albugami S, Hijazi ZM. Current practice in atrial septal defect occlusion in children and adults. Expert Rev Cardiovasc Ther ; 2020;18(6):315–29.
8. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by Echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015; 28(1):1-39
9. Mao S, Wang Y, Jiang G and Zhao Z. Effects of levothyroxine therapy on left and right ventricular function in neonates with congenital hypothyroidism: a tissue Doppler Echocardiography study. Eur J Pediatr. 2007; 166(12):1261-5.
10. Lopez L, Colan SD, Frommelt PC, Ensing GJ, Kendall K, Younoszai AK, et al. Recommendations for quantification methods during the performance of a pediatric Echocardiogram: a report from the Pediatric Measurements Writing Group of the American Society of Echocardiography Pediatric and Congenital Heart Disease Council. J Am Soc Echocardiogr. 2010; 23(5):465-77.
11. Muroke V, Jalanko M, Haukka J and Sinisalo J. Cause-Specific Mortality of Patients with Atrial Septal Defect and Up to 50 Years of Follow-Up. J Am Heart Assoc. 2023; 12(2): e027635.
12. Havlenova T, Skaroupkova P, Miklovic M, Behounek M, Chmel M, Jarkovska D, et al. Right versus left ventricular remodeling in heart failure due to chronic volume overload. Sci Rep. 2021;11(1):17136.
13. Dokainish H. Left ventricular diastolic function and dysfunction: Central role of Echocardiography. Glob Cardiol Sci Pract. 2015; 3:1-12
14. Nagueh SF. Left ventricular diastolic function: understanding pathophysiology, diagnosis, and

prognosis with Echocardiography. JACC Cardiovasc Imaging 2020; 13: 228–44.
15. Geenen LW, Uchoa de Assis L, Baggen VJM, Eindhoven JA, Cuypers JAAE, Boersma E et al. Evolution of blood biomarker levels following percutaneous atrial septal defect closure in adults. Int J Cardiol Heart Vasc. 2020; 30:100582.
16. Kılıçaslan B, Ekinci S, Kurşun M, Özel E, Solmaz H, Çabuk AK, et al. Association of cardiac adaptations with NT-proBNP levels after percutaneous closure of atrial septal defect. Turk Kardiyol Dern Ars. 2019; 47(4):258-64.
17. Dehn AM, Dannesbo S, Sellmer A, Høffner L, Blixenkrone-Møller E, Sillesen AS, et al. Atrial Septal Defect: Larger Right Ventricular Dimensions and Atrial Volumes as Early as in the First Month After Birth-a Case-Control Study Including 716 Neonates. Pediatr Cardiol. 2023; 44(7): 1578-86.
18. Song J, Lee SY, Baek JS, Shim WS ,Choi EY. Outcome of transcatheter closure of oval shaped atrial septal defect with amplatzer septal occluder. Yonsei Med J. 2013; 54(5):1104-9.
19. Park MK: Park's Pediatric Cardiology for Practitioners, 6th. Ed., ELSEVIER, Philadelphia (2018): 87 and 124
20. Karunanithi Z, Andersen MJ, Mellemkjær S, Alstrup M, Waziri F, Skibsted Clemmensen T, et al. Elevated Left and Right Atrial Pressures Long-Term After Atrial Septal Defect Correction: An Invasive Exercise Hemodynamic Study. J Am Heart Assoc. 2021; 10 (14): 1-12

Citation

Hafez, M., AbouZeid, H., Abdel-Hafeez, Y., Ibrahim, S. Biventricular Diastolic Functions before and After Interventional Occlusion of Interatrial Communication. ***Zagazig University Medical Journal*****, 2025; (49-55): -.**
doi: 10.21608/zumj.2024.327869.3637