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Original Article

An echocardiographic evaluation to determine the immediate and short-term changes in biventricular systolic and diastolic functions after PDA device closure-an observational analytical prospective study (echo- PDA study)

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ABSTRACT

Objectives: —This prospective study with a sizable cohort was undertaken to assess changes in left and right ventricle systolic and diastolic functions after percutaneous patent ductus arteriosus device closure with appropriate follow up evaluation.

Methods: — It is an observational analytical prospective study. Ninety-eight patients were recruited out of which sixty-eight patients underwent percutaneous PDA device closure and were taken for final analysis. The primary objective was to study the left and right ventricular systolic and diastolic functions pre- and post-procedure at 48 h with follow up analysis at six months.

Results: — The mean age of the patients was 7.88 ± 5.05 years with the female to male ratio was 3.85:1. Thirty-three (48.52%) of the patients had immediate post PDA device closure LV systolic dysfunction. It was more common in those having pre-procedure mean low LVEF and those having a significant reduction in mitral A velocity. It became normal at six months follow up. The study reported immediate decrease in mea/n LVEF from 63.55 ± 8.11% to 48.19 ± 7.9%. The changes in LVEDD, LVEF, LVFS and LVEDV were statistically significant ($p < 0.0001$). In diastolic functions, there were significant reductions in peak early and late diastolic velocities. There was no statistically significant difference in right chamber functional assessment.

Conclusion: Asymptomatic LV systolic and diastolic dysfunction in immediate post PDA closure period is a common complication and reported in around 48.5% cases. It was more common in those having pre-procedure mean low LVEF and those having a significant reduction in mitral A velocity.

1. Introduction

Patent Ductus Arteriosus (PDA) is a commonly occurring acyanotic congenital heart disease resulting due to failure of physiological constriction of ductus in newborn. The overall prevalence of PDA as a congenital heart disease is around 6–11%.[1–4] The isolated PDA is present in 1 in 2000 live term births.[5] Most effective and safe method of treating PDA is percutaneous closure with the help of ductal occluder.[6–9] Significant left to right shunt leads to left ventricle (LV) volume overload and LV remodeling resulting into alteration of systolic and diastolic functions of LV and right ventricle (RV). In majority of the cases these changes usually revert after PDA device closure except in a few that develop LV systolic dysfunction. The phenomenon of LV systolic dysfunction has been well observed; however, data are limited. In addition, published literature has scant information regarding predictors of LV systolic dysfunction. Furthermore, LV diastolic dysfunction and effect on right ventricular (RV) functions has not been studied well. Hence,

Abbreviations: LVEF, Left ventricle ejection fraction; LVEDD, Left ventricle end-diastolic dimension; LVEDV, Left ventricle end-diastolic volume; LVFS, Left ventricle fractional shortening.

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this prospective study with sizable cohort was undertaken to assess changes in LV and RV systolic and diastolic functions after percutaneous device closure of PDA with appropriate follow up evaluation.

2. Materials and methods

It is an observational analytical prospective study done at a tertiary care, cardiac teaching center for two years. Based on statistical calculations,[2–4] a total of 98 patients were recruited for the study. Out of which sixteen has complex PDA and was sent for Cardiothoracic surgical intervention. Fourteen patients not included in the study as nine patients who were aged >18 years and had baseline cardiomegaly, while five of the patients didn't give their consent for the study. Thus, sixty-eight patients underwent percutaneous PDA device closure and were taken for follow up and final analysis. All the patients included were in between the age group 6 months to 18 years and had clinical as well as echocardiographic proof of hemodynamically significant isolated PDA i.e., Qp/Qs > 2. Patients with silent PDA, i.e. No murmur, pulmonary vascular disease, i.e., PVRI ≥7 WU. m^2, PDA not suitable for percutaneous device closure and those having associated congenital heart disease that might alter the hemodynamic assessment were excluded. The primary objective was to study the left and right ventricular systolic and diastolic functions pre- and post-procedure at 48 h with follow up analysis at 6 months.

The patients suitable for percutaneous device closure were assessed at baseline, 48 h after the procedure and six months after in follow up with the help of transthoracic echocardiography with 4 and/or 7 MHz probes (Vivid 6, General electronics). The patent ductus arteriosus diameter was measured in the high left parasternal short-axis view. The aortic root diameter, left atrial diameter, right ventricular outflow tract dimension, LV end-diastolic dimension (LVEDD), and LV end-systolic dimension (LVESD) were obtained from the parasternal long-axis view. From these measurements, the following LV parameters were calculated: Fractional shortening (FS) = LVEDD−LVESD/LVESD × 100, and LV ejection fraction (EF) = LVEDD volume−LVESD volume/LVEDD volume × 100, where LVEDD volume = 7 × LVEDD3/2.4+ LVEDD and LVESD volume = 7 × LVESD3/2.4 + LVESD.[10] Final parameters were recorded as an average of three cardiac cycles.[11] LV systolic dysfunction was defined as a fall of absolute left ventricle ejection fraction (LVEF) post-PDA closure 10% or more from baseline or post-PDA closure LVEF of <50%.[1] Apical four-chamber view was used to assess diastolic parameters including the trans mitral flow, the peak early (E) and late atrial (A) diastolic velocities, their ratio, deceleration times, mitral E time velocity integral (E TVI), A time velocity integral (A TVI) and their ratio i.e. E TVI/A TVI with the help of Pulsed-wave (PW) Doppler. Tissue Doppler imaging was used to record mitral annular diastolic velocities in early diastole (E′) and late diastole (A′). E/E' ratio and the Tei index were also calculated. Right ventricular systolic & diastolic assessment was done by analyzing right ventricle (RV) dimensions, right atrial (RA) dimensions, RV tricuspid annular plane systolic excursion (TAPSE) and inferior vena cava (IVC) dimensions. Cardiac catheterization was done for assessment of pulmonary artery pressure and shunt quantification.[12]The PDA was closed by standard technique. Cocoon ductal occluder device (Vascular Innovations Co. Ltd., Bangkok, Thailand) was used in all the cases. The device size was selected 2 mm larger than the smallest size of ductus at pulmonary end. Post device release re-evaluation was done with trans-thoracic echocardiography. After 48 h of procedure subjects who underwent successful percutaneous closure of PDA with no significant residual shunt or complication pre discharge trans-thoracic echocardiography assessment was done. All patients underwent repeat clinical and echocardiography assessment at follow up at 6 months.

2.1. Statistical analysis

The standard SPSS 17 software (https://spss.software.informer.com/17.0) was used to do statistical analysis. Fisher exact test was applied to compare frequencies of categorical data. Where the data are normally distributed, mean with standard deviation and, where the data is skewed, median with quartile range is taken. Continuous variables were compared using two tailed paired Student *t* test with Welch's correction and were presented as mean ± SD. Pre, post and follow up comparison was done by one-way Anova test (two tailed) with post hoc analysis by Tukey test. Subgroup analysis was done with the unpaired *t* test. Where the data is skewed, continuous variables were compared with two tailed paired *t* tests (Wilcoxon matched-pairs signed rank test).

3. Result

Most of the patients belong to age group 1–18 years with the mean age of 7.88 ± 5.05 years (median age being 7 years). Fifty-four (79.4%) were female, while rest fourteen (20.5%) patients were male. The female to male ratio was 3.8:1. The mean size of PDA on lateral aortic arch angiogram was 4.24 ± 1.61 mm, smallest PDA measured 2 mm while the largest one was 10 mm at its narrowest point. There were no peri-procedural complications, only one patient had residual flow which was absent on follow up. Nine (13.8%) patients had evidence of pulmonary arterial hypertension. The procedure related baseline parameters are shown in Table 1. Other baseline parameters and their comparison at 48 h and 6 months of follow up on echocardiographic evaluation are shown in Table 2. The comparison of various other parameters in patients with or without LV systolic dysfunction are summarized in Table 3.

4. Discussion

Percutaneous closure of PDA is an excellent modality of treatment with the excellent outcome.[1] Procedural complications like access site complications, residual shunts, and device related hemolysis, device embolization is well described. However, peri-procedural morbidity associated with surgical closure, are not seen with percutaneous closure of PDA. There are reports of LV systolic dysfunction in patients with PDA secondary to ductal occlusion.[13,14] Regarding LV diastolic properties and RV function assessment post device closure, there is scarcity of data. This prospective study aimed at assessment of pattern of LV and RV systolic and diastolic function in patients with PDA and changes 48 h post procedure and after follow up period of six months. The late age of presentation

Table 1
Baseline procedural characteristics.

Parameters	Mean ± S.D.	Median	Quartile range (Q1-Q3)
PDA size on Angio (mm)	4.24 ± 1.61	4.0	3.32–5.1
Qp: Qs	2.64 ± 0.4		
SBP (mm Hg)	111.69 ± 20.03	108	98–123
DBP (mm Hg)	56.07 ± 15.09	56.06	42–68
MAP (mm Hg)	77.76 ± 17.2	78	64–87.5
PASP (mm Hg)	36.48 ± 16.4	30	27.5–40
PADP (mm Hg)	13.21 ± 12.78	10	4–16.5
PAMP (mm Hg)	22.72 ± 13.66	18	14.5–24.0

SBP- systolic blood pressure DBP- diastolic blood pressure, MAP- Mean arterial pressure, PASP- pulmonary artery systolic pressure, PADP- pulmonary artery diastolic pressure, PAMP- pulmonary artery mean pressure.

Table 2
Comparative assessment various echocardiographic parameters at baseline, 48 h post procedure and during follow up after six months.

Parameters	Pre.	Post	Follow up	*p* value	*p* value[a]	*p* value[b]	*p* value[c]
LVEDD (mm)	43.90 ± 8.09	38.89 ± 7.3	37.58 ± 7.12	<0.0001[a]	<0.0001	<0.0001	0.0540
LVESD (mm)	29.02 ± 6.32	28.77 ± 6.07	24.9 ± 5.37	0.7790	0.941	<0.0001	<0.0001
LV EF (%)	63.55 ± 8.11	48.19 ± 7.9	62.10 ± 8.52	<0.0001[a]	<0.0001	0.868	<0.0001
LV FS (%)	34.24 ± 6.65	25.38 ± 6.06	34 ± 7.09	<0.0001[a]	<0.0001	0.9998	<0.0001
LVEDV (ml)	91.55 ± 37.30	68.8 ± 30.7	63.9 ± 27.16	<0.0001[a]	<0.0001	<0.0001	0.125
LVESV (ml)	34.44 ± 17.26	33.83 ± 16.54	24.25 ± 12.9	0.6864	0.9895	0.001	0.001
Aortic Dimension (mm)	18.78 ± 5.13	19.5 ± 4.59	19.59 ± 4.46	0.3969	0.8332	0.7920	0.9969
Left Atrium (mm)	26.71 ± 6.37	23.35 ± 3.91	21.82 ± 3.8	<0.0001[a]	0.0268	0.0007	0.4634
La/Ao Ratio	1.42 ± 0.27	1.21 ± 0.24	1.13 ± 0.21	0.0002[a]	0.0035	<0.0001	0.4158
Mitral E (m/sec)	0.91 ± 0.18	0.77 ± 0.16	0.78 ± 0.16	0.0001[a]	0.0003	0.008	0.9757
Mitral E VTI (cm)	7.81 ± 3.23	5.83 ± 2.12	5.92 ± 1.74	<0.0001[a]	<0.0001	0.0265	0.9885
Mitral A (m/sec)	0.63 ± 0.14	0.46 ± 0.095	0.49 ± 0.09	<0.0001[a]	<0.0001	0.0004	0.839
Mitral A VTI (cm)	3.79 ± 1.86	2.38 ± 0.52	2.69 ± 0.79	0.0014[a]	0.0038	0.0191	0.512
Mitral E/A	1.50 ± 0.31	1.65 ± 0.46	1.58 ± 0.34	0.0686	0.159	0.469	0.797
Mitral DT (msec)	126.9 ± 11.96	127.4 ± 13.28	127.3 ± 11.65	0.2697	0.5064	0.6372	0.9930
Mitral E′ (m/sec)	13.31 ± 2.69	13 ± 2.9	13.09 ± 2.42	0.0951	0.8985	0.9473	0.9911
Mitral E/E′	7.35 ± 1.66	6.25 ± 1.55	6.22 ± 1.48	<0.0001[a]	0.0242	0.0204	0.9976
Tei index	0.53 ± 0.05	0.67 ±0 .010	0.60 ± 0.09	<0.0001[a]	<0.0001	0.0020	0.0415
IVRT	51.55 ± 6.95	68.04 ± 7.63	67.86 ± 11.54	<0.0001[a]	<0.0001	<0.0001	0.9970
RA dimension	23.87 ± 4.00	23.58 ± 3.56	23.54 ± 2.43	0.6402	0.8847	0.8355	0.9969
RV dimension	23.57 ± 4.92	22.99 ± 4.84	22.59 ± 3.14	0.2246	0.4392	0.4183	0.8653
MPA	21.05 ± 4.34	17.57 ± 3.21	16.27 ± 2.49	<0.0001[a]	0.0007	<0.0001	0.3210
RPA	10.21 ± 2.22	9.02 ± 1.24	8.5 ± 1.41	0.0031[a]	0.0223	0.0006	0.4717
LPA	10.66 ± 3.28	9.09 ± 1.23	8.03 ± 1.54	0.0097[a]	0.0224	<0.0001	0.1672
TAPSE	20.76 ± 1.81	18.19 ± 2.66	21.7 ± 3.7	0.0001[a]	0.0025	0.9086	0.0006
TR Gradient	9.27 ± 17.39	3.72 ± 10.43	2.93 ± 8.146	Ns	Ns	ns	Ns

Significant *p* value.
[a] Pre-closure versus 48 h post-closure.
[b] Pre-closure versus six-month post-closure.
[c] 48 h post-closure versus six-month post-closure.

Table 3
Comparison of baseline parameters in patients with or without LV systolic dysfunction.

Parameters	LV dysfunction ($n = 33$)	Without lv dysfunction ($n = 35$)	*p*-value
Age	7.433 ± 4.609	8.357 ± 5.611	0.6336
LVIDD (mm)	43.38 ± 7.14	44.48 ± 9.12	0.79
LVIDS (mm)	29.19 ± 4.66	29.12 ± 7.72	0.97
LV EF (%)	59.99 ± 8.46	66.57 ± 7.538	0.036*
LV FS (%)	32.3 ± 6.8	35.49 ± 6.59	0.21
EDV (ml)	87.27 ± 32.38	95.47 ± 42.13	0.56
ESV (ml)	33.05 ± 12.1	35.73 ± 20.96	0.67
Aortic Dimension (mm)	17.35 ± 3.914	20.12 ± 5.87	0.15
Left Atrium (mm)	25.59 ± 3.55	27.75 ± 8.19	0.36
Ao/La Ratio	1.457 ± 0.255	1.387 ± 0.29	0.49
Mitral E (m/sec)	0.89 ± 0.20	0.92 ± 0.18	0.68
Mitral E VTI (cm)	7.321 ± 2.871	8.267 ± 3.56	0.44
Mitral A (m/sec)	0.5864 ± 0.157	0.6667 ± 0.123	0.14
Mitral A VTI (cm)	3.386 ± 2.028	4.167 ± 1.66	0.27
Mitral E/A	1.552 ± 0.3842	1.375 ± 0.126	0.12
Mitral DT (msec)	87.07 ± 20.71	85.60 ± 11.37	0.82
Mitral E′ (m/sec)	13.00 ± 2.512	13.60 ± 2.898	0.56
Mitral e/E′	7.539 ± 1.809	7.183 ± 1.545	0.58
Tei index	0.521 ± 0.057	0.5387 ± 0.045	0.37
IVRT	50.1 ± 8.52	52.9 ± 5.04	0.31
Right atrial dimension	24.3 ± 2.78	23.5 ± 4.95	0.61
Right ventricular dimension	23.4 ± 4.75	23.7 ± 5.23	0.88
MPA	21.5 ± 4.7	20.6 ± 4.09	0.59
RPA	10.3 ± 2.22	10.1 ± 2.31	0.85
LPA	11.1 ± 3.56	10.3 ± 3.07	0.50
TAPSE	20.3 ± 2.33	21.2 ± 1.01	0.19
TR Gradient	6.97 ± 11.5	11.4 ± 21.7	0.49
PASP	36.5 ± 19.6	36.5 ± 13.5	0.996
PADP	13.4 ± 16.1	13.1 ± 9.32	0.95
MPAP	23.1 ± 16.5	22.4 ± 10.9	0.90
SBP	109 ± 24.3	114 ± 15.5	0.53
DBP	54.8 ± 17.0	57.3 ± 13.5	0.67
MBP	78.6 ± 20.0	76.9 ± 14.8	0.80
PDA size	4.39 ± 2.11	4.13 ± 0.94	0.72
PDA systolic gradient	73.6 ± 25.1	81.0 ± 27.3	0.46
PDA diastolic gradient	26.7 ± 15.0	24.4 ± 15.6	0.71

* Significant *p* value.

(the mean age 7.88 ± 5.05 years) in the present study could be due to late medical seeking and asymptomatic natural history of patent ductus arteriosus.[15] The higher number of female subjects in our study is in concordance with other studies suggestive of higher prevalence of PDA among female sex. Majority of the participating subjects had baseline normal LVEF which in line to other studies done on same topic except few isolated case reports.[16,17] In present study, on 48-h post procedure assessment, 48.52% patients had LVEF <50% which by definition LV systolic dysfunction. In previous studies various definitions of LV systolic dysfunction has been utilized. In study by Jeong et al[18] in adult patients cut off of LVEF <50% was used to define LV systolic dysfunction whereas Saurabh et al defined LV systolic dysfunction as a post-PDA closure absolute LVEF of <50% and/or reduction in LVEF of ≥10% from the baseline.[1] In absence of consensus on definition of LV systolic dysfunction in patients with PDA at baseline and after PDA closure, both were considered in the present study. In prior studies, the incidence of LV systolic dysfunction post PDA device closure ranges from 19% to 25% which was much lesser then present study.[13–19] This could be due to higher age group in present study which is in concordance to earlier study, which concluded that LV dysfunction in adolescents and adults is more common in patients with PDA operated >2 year of life.[20] On post procedure echocardiographic evaluation, there was statistically significant fall in LVEDD, LVEF, LVFS, LVEDV and left atrium size without statistically significant fall in LVESD and LVESV. Thus, patients with LV systolic dysfunction had significant reduction in LV diastolic volumes, consequent to reduction in preload. In contrast LV systolic volumes remained static. These finding are in agreement with the previous studies. On follow up of patients of LV systolic dysfunction, there was complete recovery of LVEF and LVFS; this was mainly due to statistically significant fall in LVEDD and slight fall in LVESD. This was in concordance to previous studies which found complete recovery of LV function on follow-up.[16–19] Galal et al, also found trend towards regression in LVEDD, though not significant while LVESD remained static. These patients had significant reduction in LVEF and LVFS.[16] All the parameters in patients with LV systolic dysfunction subgroup were statistically similar to that in patients with normal LV systolic function ($p > 0.05$), except the pre-PDA closure mean LVEF which was significantly lower in patients who developed LV systolic dysfunction. This was in a similar line with earlier work, by Saurabh et al1 who reported that patients who develop post closure LV systolic dysfunction had a lower mean LVEF pre-PDA closure, as compared to patients who do not develop LV systolic dysfunction.[18] In observations made by Yeong Hyang Kim et al[13] he concluded that in children undergoing percutaneous PDA closure the LVEDV showed a statistically significant fall in patients with LV systolic dysfunction (p 0.026) as compared to those without LV systolic dysfunction (p 0.152). The discrepant reduction in LVEDV in comparison to LVESV is hypothesized as the cause of LV systolic dysfunction. It can be argued that rapid reduction in preload may itself lead to underestimation of LV systolic function. However, LVEF <50% is unlikely to be a consequence of preload reduction alone. The predictors of post PDA ductal closure, LV systolic dysfunction are not yet well identified. Post closure simultaneous reduction in LV preload and increase in afterload due to abolition of low resistance pulmonary circulation led to preload-afterload mismatch resulting in left ventricular systolic dysfunction. In majority of the cases, these changes get reverse within 48 h due to reverse remodeling of LV in the form of significant decline in LVEDD except few cases where because of chronic volume overload and irreversible myocardial injury, LV dysfunction persists. Other possible hypothesis suggested by McNamara et al[21] who stated that there is decreased coronary perfusion in PDA patients. Heart tries to compensate for this decreased perfusion of myocardium and reach a satisfactory compensatory state. However, post closure, due to increased afterload and decreased preload, left ventricle is unable to compensate. Another theory suggested by El Khuffash et al,[22] who found increased troponin level in patients of PDA, which suggest subclinical injury inflicted by PDA. Assessment of hemodynamic parameters revealed statistically significant rise in aortic diastolic blood pressure, mean aortic pressure and fall in pulmonary pressures and right ventricular pressures with exception to aortic systolic pressure. These findings were in accordance to study by Harada et al[23] and Eorela et al,[17] who have shown that the systolic and diastolic blood pressures increase after coil occlusion of PDA. In view of wide variability and expected effect of preload on LV diastolic filling patterns as well as myocardial velocities diastolic properties were assessed and analyzed, as such without an attempt, to define LV diastolic function. In absence of consensus on LV diastolic dysfunction in PDA, baseline parameters were compared with that of immediate post procedure and follow up, to assess changes consequent to percutaneous PDA closure. There were very limited data on diastolic function assessment in available literature. In our study, assessment of LV diastolic functions revealed statistically significant reduction in mitral E and A velocity which was consequent to reduction in preload after PDA closure. However, the reduction in mitral E/A ratio was statistically not significant. There was no significant change in mitral E deceleration time (EDT). This finding was in concordance to earlier studies by Elseikh et al[10] and Schmitz et al,[24] which found significant reduction in mitral E and A velocities following PDA closure. They suggested decreased flow across mitral valve, decrease in sympathetic activity and normal LV compliance as possible explanation for this fall. However, our findings are discordant to Eorela et al, who didn't find statistically fall in mitral A velocity and statistically significant fall in mitral EDT.[17] In present study the mitral septal annular velocity (E′) did not change significantly. However mitral E/E′ ratio showed significant reduction in immediate post closure state. This reduction in mitral E/E′ is mainly due to reduced preload and consequent mitral E velocity and not due to change in mitral E'. Similar to our findings Kim et al also did not find significant change in either mitral E′, E/E′ ratio compared to baseline either in post immediate PDA closure state or on follow up.[13] Studies by Saurabh et al and Elseikh et al, also found similar findings and suggested change in mitral E/E′ was mainly due to reduced mitral E velocity with insignificant change in mitral annular velocity.[1,10] Tei index, which is combined parameter of LV systolic and diastolic dysfunction showed a statistically significant rise following device closure which is in agreement with study by Elseikh et al[10] and Cheung et al[25] who showed that the Tei index is affected significantly by acute changes in loading conditions. Also, Lutz et al[19] stated that an increase in preload decreases the Tei index indicating its sensitivity to acute increases in left ventricular preload. IVRT also showed statistically significant prolongation. This was in concordance to study by Schmitz et al,[24] who showed that preload augmentation in fact causes a decrease in isovolumic relaxation time. We suggested that changes in LV diastolic parameters were mainly due to sudden change in preloading conditions resulting in fall in mitral velocity, prolongation of IVRT and Tei index immediately and these parameters didn't change significantly even at follow up after six months. As compared to patients with normal post closure LVEF, diastolic parameters in patients who developed post closure LV systolic dysfunction didn't show any statistically significant difference. It might be because of slow improvement in LV diastolic properties. Short term follow up might have limited our study in analyzing these changes. Though, it could be argued that PDA affects left side of heart, we aimed to identify any subtle change in echocardiographic parameters and how they evolve during follow-up. We found statistically

significant fall in in pulmonary artery dimensions at post closure and follow up which could be explained by cutoff of the excess blood which was shunted from aorta to pulmonary artery.

4.1. Study limitations

The present study lacks age and sex matched controls which could have provided benchmark for assessment of LV diastolic function. Indexing for body surface area would have an improved assessment of different parameters in our population. In the present study since the majority of the parameters studied are load dependent which can influence the LV dimensions and functions. We have not studied the effect of other periprocedural complications like device related coarctation, worsening of aortic regurgitation (AR) and transient anesthetic effect while assessing transient LV dysfunction. Though the occurrence of the overall effect of these confounding factors is very rare with selection of appropriate size devices and use of short-lasting anesthetic agent. In present study short term follow up was done. Long term follow-up assessment might have resulted in insight into further changes in diastolic properties of LV. This study is limited to children with PDA, cannot be applied to other age group patients.

5. Conclusion

Asymptomatic LV systolic and diastolic dysfunction in immediate post PDA closure period is a common complication and reported in around 48.5% cases. It was more common in those having preprocedure mean low LVEF and those having a significant reduction in mitral A velocity. PDA device closure is also associated with altered diastolic properties of LV which shows improvement during follow up. The improvement in diastolic function of LV lags behind that of improvement in LV systolic function.

Ethical clearance

Ethical clearance was obtained from Institutional Ethics Committee of GSVM Medical College and Hospital, Kanpur on date 23/10/2019 via Reference No. EC/215./Sep//2019.

Declaration of competing interest

The authors declare no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ihj.2021.06.017.

What was known?

PDA physiology results in alteration of systolic functions of LV. In majority of the cases, these changes usually revert after PDA device closure except in a few that develops LV systolic dysfunction. The phenomenon of LV systolic dysfunction has been well observed; however, data are limited. Published literature has scant information regarding predictors of LV systolic dysfunction. Furthermore, LV diastolic dysfunction and effect on right ventricular (RV) functions has not been studied well.

What is new?

This prospective study with a sizable cohort was undertaken to assess changes in both LV and RV systolic and diastolic functions after percutaneous device closure of PDA with appropriate follow up evaluation and further insight into predictors of LV systolic dysfunction.

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