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Assessment of Left Ventricular Dysfunction Post Transcatheter Patent Ductus Arteriosus Closure in Pediatric Patients

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ABSTRACT

Objectives: To compare Left Ventricular (LV) function before and after Transcatheter Patent Ductus Arteriosus (PDA) closure and to identify factors associated with LV dysfunction following device closure.

Study Design: Quasi- experimental study.

Place and Duration of Study: Armed Forces Institute of Cardiology/National Institute of Heart Diseases, Rawalpindi, Jan to Jun 2024.

Methodology: A non-probability consecutive sampling technique was used for patient selection. Thirty patients aged upto 18 years who underwent PDA device closure were enrolled. Echocardiographic parameters were measured before, 24 hours after device closure and at 1 and 3 months. Assessment of pre procedure LV function was done upon admission of the patient and the parameters were recorded in predesigned profoma using M mode echocardiography, TDI and Speckled tracking. Trans catheter device closure of PDA was done using standard procedure and following the standard guidelines.

Results: Out of 30 patients, 17(56.7%) were males and 13(43.3%) were females. The median age of patients was 4.00(2.75-6.00) year. After 24 hours of procedure, 5(16.7 %) patients developed LV dysfunction. Patients with LV dysfunction had a median PDA diameter of 8.00(8.00-9.00) mm. LAd/AOd ratio 1.24(1.22-1.32) was significantly higher in patients with LV dysfunction (p<0.05). The LAd, LVEDD, LVESD and LVEDVI showed a significant decline following procedure and throughout the course of 3 months (p<0.001).

Conclusions: Transcatheter PDA closure causes a decrease in LV performance immediately post procedural closure, which recovers completely within 3 months. Pre closure LAd/Aod ratio and PDA diameter were found to be an important markers in anticipating LV dysfunction post device closure.

Keywords: Diameter of aorta, Fractional shortening, Left atrial diameter, Left ventricular dysfunction

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INTRODUCTION

The major pulmonary artery at its bifurcation, or the left pulmonary artery, and the ductus arteriosus, a fetal connection, are located just distal to the beginning of the left subclavian artery. It facilitates the right-to-left shunting of oxygenated blood from the placenta to the fetal systemic circulation, avoiding the fetal pulmonary vasculature. It is a crucial part of fetal circulation. Low arterial oxygen concentration and placental prostaglandin E2 (PGE2) maintain the ductus arteriosus open in the fetus. After a number of birth-related modifications, the ductus arteriosus functionally closes in the first 24 hours after birth and completely anatomically closes in 3–4 weeks. According to the retrospective study conducted from 2013 to 2020 in Africa, the prevalence of PDA is reported to be 1.004 per 1000 live birthsworldwide.[1]

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Echocardiography is the most important method of providing information with regard to size of the PDA and its hemodynamics and it is also the most frequently used tool to assess cardiac chamber size and LV systolic performance.[2] Moreover, echocardiography has also been used as a tool for imaging during PDA device closure of premature neonates.[3]

Transcatheter PDA closure has emerged as the primary method for closing the majority of PDA abnormalities because it has shown to be a safe and successful intervention with outcomes that are on par with surgical closure in the short and long terms.[4,5] Compared to the preclosure state in adults, left ventricular ejection fraction remains low late after PDA closure.[6], and in children, PDA closure might cause an abrupt decline in LV systolic function.[7] A Pakistani study reported, an immediate, although temporary, decline in left ventricular (LV) systolic performance following PDA closure. Ejection fraction

(EF), left ventricular end-diastolic diameter (LVEDd), PDA diameter (PDAd), left atrial diameter (LAd), PDAd/Aortic diameter (AOd) ratio, and LAd/AOd ratio are predictive factors for this decrease in LV systolic function.[8] However, it is still unclear what pre-closure predictors there are for LV systolic dysfunction after PDA device closure. The objective of this study was to investigate the changes of LV systolic function after PDA device closure, and to see how the echocardiography indicators could predict LV dysfunction after PDA closure.

A lack of clarity exists regarding pre-closure echocardiographic predictors of left ventricular (LV) systolic dysfunction after PDA device closure in pediatric patients. The aim of this study was also to highlight the factors which can lead to LV dysfunction as mentioned in the results. In addition, to highlight the importance of timely intervention which leads to LV remodeling and return of LV parameters to normal levels. These findings will help pediatric patients receiving this intervention to achieve better outcomes and make better clinical decisions.

METHODOLOGY

This Quasi experimental study was conducted at Paediatric Cardiology Department, Armed Forces Institute of Cardiology & National Institute of Heart Diseases Rawalpindi from Jan-Jun 2024 after the approval from Institutional Ethical Review Board (IERB) (Ltr.no 9/2/R&D /2022/227-23rd Dec- 2022).

The sample size of n=7 patients was determined using WHO sample size calculation software, considering prevalence 0.4% of PDA patients[8], a confidence level of 95%, and 5% margin of error. However, data was collected from n= 30 patients using non probability consecutive sampling

Inclusion Criteria: Pediatric patients up to 18 years of age, both gender, and diagnosed with PDA who needed trans catheter PDA closure were included in this study

Exclusion criteria: Patients with other congenital heart diseases with duct dependent pulmonary circulation and severe pulmonary vascular disease were excluded

Informed consent was obtained from the guardian / parent of the child and those who met inclusion criteria included in the study. The patients were booked in the outpatient department and were given dates for the procedure. All the cases were admitted a day before the procedure with detailed echo assessment done on the same day. Echocardiography was carried out using GE Vivid echo machine with a 4 MHz transducer. All the parameters were recorded on a predesigned profoma namely the patient's age, weight, height, BSA, PDA diameter, LA diameter, LVEDD, LVESD, LVEDVI, LVESVI, EF, FS, GLS scoring. Assessment of pre procedure LV function was done upon admission of the patient and the parameters were recorded in predesigned profoma using M mode echocardiography, TDI and Speckled tracking. Trans catheter device closure of PDA was done using standard procedure and following the standard guidelines. Post occlusion echo assessment was done using the same parameters at 24 hours post PDA device closure and the patients were observed in the ward for any untoward effects or complications up till the time of discharge. Patients were then followed after 1st month and 3rd month in the outpatient department, with follow up dates being mentioned. Assessment of the left ventricular function by 2D echocardiography, measuring same parameters was done. The entire process of patient selection, intervention done, follow up and outcome assessment as shown in figure below.

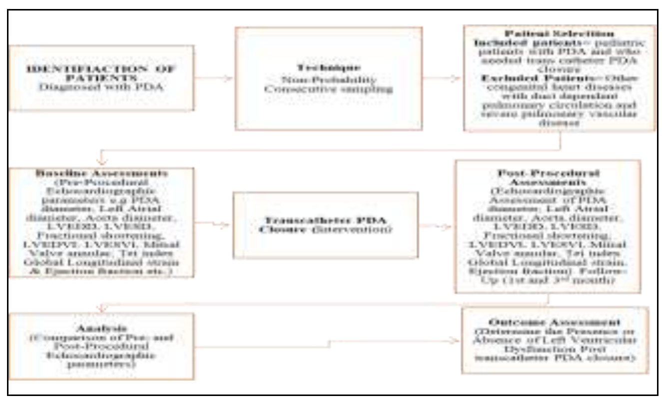

Figure: Flow Chart Depicting Quasi-Experimental Study Design with Single Group Approach

Data were entered and analyzed by using Statistical Package of Social Science SPSS version 23:00. Normality of data was explored by Shapiro wilk test. All the continuous variables like age, PDA diameter and echocardiographic parameters (PDA diameter, LAd, AOd, LVEDD, LVESD, FS, LVEF, LVEDVI, LVESVI, Tei and Global longitudinal strain) were found to be normally distributed except BSA therefore, median and interquartile range were computed. For qualitative variables (gender, Krichenko classification and LV dysfunction, percentage and frequencies were reported Chi-square test was applied to find the association between gender, Krichenko classification and LV dysfunction. The median difference of PDA diameter, LAd, AOd, LVEDD, LVESD, FS, LVEF, LVEDVI, LVESVI, Tei and Global longitudinal strain between patients who developed post procedure LV dysfunction and those who did not was determined using an Mann Whitney U test. The echocardiographic parameters before and after the procedure, in the first and third months, were compared using a Wilicoxon Signed rank. test A *p* value of ≤0.05 was considered as statistically significant

RESULTS

Thirty patients were included in the study. The median age of the patients was 4.00(2.75-6.00) year and among them 17(56.7%) were males and 13(43.3%) were females. The most common type of PDA seen was Type A 20(66.7%) as per the Krichenko Classification. The median PDA diameter measured was 4.00(3.00-6.63) mm. (Table I)

Table-I: Demographic Characteristics and Krichenko Classification of Pda of Study Participants (N=30)

Variables		Frequency (%)
Demographics		
Gender	Male	17(56.7)
	Female	13(43.3)
Age (year)	Median(IQR)	4.00(2.75-6.00)
BSA (kg/m2)	Mean± SD	0.69±0.12
		Frequency (%)
Krichenko Classification	A	20(66.7)
	B	6(20.0)
	C	4(13.3)
PDA diameter (mm)	Median(IQR	4.00(3.00-6.63)

PDA= Patent Ductus Arteriosus

In this study, 5(16.7%) of our patients developed LV dysfunction which was mild 24 hours after the procedure. Amongst them, one (3.3%) of the patients had mild LV dysfunction before undergoing device closure and he subsequently went on to develop moderate LV dysfunction 24 hours after the procedure. On follow up studies of this patient the left ventricular function improved with follow up at one month which showed mild LV dysfunction and subsequently there was no LV dysfunction at 3 months post procedure as shown in table-II

Patients with left ventricular dysfunction had a median PDA diameter of 8.00(8.00-9.00) mm, which differed significantly (*p*<0.001) from patients without post-procedural LV dysfunction. The median LVESVI of patients with LV dysfunction was found to be considerably greater 38.00(37.00-39.00) ml/m2 compared to patients without LV dysfunction 33.00(31.00-37.00) ml/m2 (*p*=0.03).

Table-II: Frequency of Left Ventricular Dysfunction Before and After PDA Stenting (n=30)

Variable	Frequency (%)
Before Procedure	1(3.30)
At 24 hours	5(16.7)
At 1st month	1(3.3)
At 3rd month	0

Furthermore, LAd/AOd ratio 1.24(1.22-1.34) was significantly higher in patients with LV dysfunction (*p*=0.02) meaning the greater the left sided heart dilatation and hence the more the hemodynamically significant PDA, the more the chances of post procedure left ventricular dysfunction (Table III).

A statistically significant decline in the left atrial diameter [25.00(22.75-26.00) mm vs. 22.00(21.00-24.00) mm; *p*<0.001], LVEDD [44.00(39.75-45.25) mm vs. 42.00(37.00-43.25) mm; *p*<0.001], LVESD [30.00(28.00-32.00) mm vs. 29.00(27.00-30.00) mm; *p*<0.001], and LVEDVI [89.50(80.25-95.00) l/m2 vs. 86.50(78.50-92.25) l/m2; *p*<0.001] throughout the course of three months. The LVEF [60.00(58.00-61.25) % vs. 59.50(58.00-60.00) %; *p*=0.88], fractional shortening [36.00(32.00-40.00) % vs. 36.00(32.00-39.25) %; *p*=0.81], and left ventricular function as determined by GLS [-24.00(27.00-22.75) vs. -24.50(27.00-23.00); *p*=0.37] all demonstrated a decline following procedure and throughout the course of 3 months. In addition, the LVESVI [33.00(31.75-38.00) ml/m2 vs. 34.00(32.00-38.00) ml/m2; *p*= 0.07] showed a mild increase on follow up studies (Table-IV)

DISCUSSION

This study showed a fall in the left ventricular systolic function within 24 hours after successful PDA device closure which was transient and did not require any medical treatment. The left ventricular ejection fraction, fractional shortening as well LV function measured through GLS showed a decline in the first 24 hours following the procedure and returning to normal at one and three months post device closure. Further adding to this, the LV dysfunction reverted to baseline levels on follow up in the current study, consistent with the previous studies.[9,10]

Patients with large PDA may develop left-sided volume overload and pulmonary hypertension. The

Table-III: Association of Demographics and Pre-echocardiography Parameters with LV Dysfunction Post Transcatheter PDA Closure (n=30)

Variables		LV Dysfunction No (N=25)	LV Dysfunction Yes (N=05)	*p*-value
Demographics		**Frequency (%)**		
Gender	Male	14(56.0)	3(60.0)	1.00
	Female	11(44.0)	2(40.0)	
		Mean± SD		
BSA(kg/m2)		0.68±0.12	0.73±0.13	0.47
		Median(IQR)		
Age(year)		4.00(3.00-5.00)	5.00(4.00-6.00)	0.47
Echocardiographic Parameters				
PDA diameter (mm)		4.00(3.00-5.00)	8.00(8.00-9.00)	<0.001
Left Atrial diameter (mm)		25.00(22.00-26.00)	25.00(25.00-26.00)	0.28
Aorta diameter (mm)		20.00(18.00-21.00)	21.00(19.00-21.00)	0.88
LVEDD (mm)		44.00(39.00-45.00)	46.00(46.00-47.00)	0..73
LVESD (mm)		30.00(28.00-31.00)	32.00(31.00-33.00)	0.02
Fractional shortening (%)		36.00(32.00-40.00)	34.00(32.00-40.00)	0.08
LVEF (%)		60.00(58.00-61.00)	61.00(60.00-62.00)	0.88
LVEDVI (ml/m2)		88.00(82.00-94.00)	95.00(94.00-98.00)	0.48
LVESVl (ml/m2)		33.00(31.00-37.00)	38.00(37.00-39.00)	0.03
Mitral Valve annular Tei index		0.32(0.32-0.33)	0.33(0.30-0.34)	0.06
Global Longitudinal strain		-24.00(27.00-23.00)	-26.00(26.00-24.00)	0.91
PDAd/AOd		0.19(0.16-0.23)	0.42(0.41-0.43)	<0.001
LAd/AOd		1.20(1.16-1.29)	1.24(1.22-1.32)	0.03

LV= Left Ventricular; LVEDD=Left Ventricular End-diastolic diameter; LVESD= Left Ventricular End-Systolic diameter; LVEF= Left Ventricular Ejection Fraction; LVEDVI= Left Ventricular End-Diastolic Volume Index; LVESVI= Left Ventricular End Systolic Volume Index; PDAd/AOd=ratio of Patent Ductus Arteriosus to Aortic root diameter; LAd/AOd= ratio of Left Atrial diameter to Aortic Root diameter; PDA= Patent Ductus Arteriosus

Table-IV Echocardiography Parameters Before and after PDA Closure (n=30)

Variables	Pre-procedure	Post procedure within 24 hour	*p*-value	1st month	**p*-value	3rd month	***p*-value
	Median(IQR)						
LAd (mm)	25.00(22.75-26.00)	23.00(21.00-25.00)	<0.001	22.50(21.00-24.00)	<0.001	22.00(21.00-24.00)	<0.001
AOd (mm)	20.00(18.00-21.00)	20.00(18.00-21.00)	0.15	20.00(18.00-21.00)	0.05	20.00(18.00-21.00)	0.09
LVEDD (mm)	44.00(39.75-45.25)	42.00(37.00-44.00)	<0.001	42.00(37.00-44.00)	<0.001	42.00(37.00-43.25)	<0.001
LVESD (mm)	30.00(28.0-32.00)	29.00(27.00-30.00)	<0.001	29.00(27.00-29.25)	<0.001	29.00(27.00-30.00)	<0.001
FS (%)	36.00(32.00-40.00)	34.00(29.75-38.00)	0.03	35.50(32.00-39.25)	0.59	36.00(32.00-39.25)	0.81
EF (%)	60.00(58.00-61.25)	58.00(54.75-60.00)	0.001	59.50(58.00-61.00)	0.07	59.50(58.00-60.00)	0.88
LVEDVI (ml/m^2)	89.50(80.25-95.00)	87.50(79.25-93.00)	<0.001	87.00(78.50-92.25)	<0.001	86.50(78.50-92.25)	<0.001
LVESVl (ml/m^2)	33.00(31.75-38.00)	33.00(32.00-38.00)	1.00	33.50(32.00-38.00)	0.02	34.00(32.00-38.00)	0.07
MVA Tei Index	0.32(0.30-0.34)	0.33(0.31-0.37)	0.004	0.32(0.31-0.33)	0.49	0.32(0.31-0.33)	0.75
GLS	24.00(27.00-22.75)	23.50(26.25-22.00)	0.02	24.50(27.00-23.00)	0.96	24.50(27.00-23.00)	0.37

LV= Left Ventricular; LVEDD=Left Ventricular End-diastolic diameter; LVESD= Left Ventricular End-Systolic diameter; LVEF= Left Ventricular Ejection Fraction; LVEDVI= Left Ventricular End-Diastolic Volume Index; LVESVI= Left Ventricular End Systolic Volume Index; MVA= Mitral Valve annular; GLS= Global Longitudinal Strain. p value= Pre-Procedure vs. 1st month;**p value= Pre-Procedure vs. 3rd month*

physiological effect and clinical relevance of PDA are mostly dependent on its size, the magnitude of the shunt, and the patient's underlying cardiovascular state. Closing a hemodynamically significant PDA results in an increase in afterload by eliminating the low resistance pulmonary circulation from the LV outflow circulation and a decrease in preload to the LV due to the elimination of the left to right shunt. Previous studies have reported a change in LV performance following closure followed by return to baseline levels within a few months after device closure of PDAs in children while another study showed the incidence and predictors of post-PDA closure changes in LV functions.[11]

The left heart dimensions, namely the left atrial diameter, LVEDD, LVESD and LVEDVI showed a significant decline within 24 hours following PDA closure and continued to decrease significantly at one month and 3 months follow up (p<0.001). According to Gupta et al. findings, which showed a significant drop in LVESD (p<0.001) both immediately before closure and throughout later follow-ups, this decline continued at one-month and three-month follow-ups.[11]

Larger PDAs 88.00(8.00-9.00) mm had a statistically significant impact on the development of post procedure LV dysfunction (p<0.001) .In addition, this study showed that the larger the LAd/AOd ratio 1.24(1.22-1.34) mm, meaning the greater the left sided heart dilatation and hence the more the hemodynamically significant PDA, the more the chances of post procedure left ventricular dysfunction (p<0.02). Previous study showed the incidence and predictors of post-PDA closure changes in LV functions with larger PDA sizes leading to LV dysfunction (p=0.013).[12]

The left ventricular remodeling is caused by a significant left-to-right shunt through PDA, hence, it is expected that the left ventricular reverse remodeling will occur after the duct closes. In this study, the following parameters namely the 25.00(22.75-26.00) mm vs. 22.00(21.00-24.00) mm; p<0.001), LVEDD (44.00(39.75-45.25) mm vs. 42.00(37.00-43.25) mm; p<0.001), LVESD (29.67±2.95 mm vs. 27.90±2.65 mm; p<0.001), and LVEDVI (89.50(80.25-95.00) ml/m^2 vs. 86.50(78.50-92.25) ml/m^2; p<0.001), FS (36.00(32.00-40.00) % vs. 36.00(32.00-39.25)%; p=0.81), and LVEF (60.00(58.00-61.25) % vs. 59.50(58.00-60.00) %; p=0.88), decreased in the first 24 hours following PDA closure. Our results are comparable to the study performed by Talat et al. which showed a notable decline in LVEDD (34.59 ± 10.99 mm at baseline versus 27.49±9.36 mm at day 1 following procedure), LVEF (66.51±9.00 vs. 51.01±12.56%) and LVFS (36.23±7.72 vs. 26.04±7.64%) immediately post PDA device closure as well as the fact that the larger the PDA, the more the left sided heart dilatation, the more the chances of LV dysfunction post PDA device closure.[8]

PDA causes to left ventricular volume overload, which is needed to increase the left ventricular output by Frank-Starling response, and it can therefore overcome the left-to-right shunt and maintain systemic circulation.[13] These results asserted that early PDA closure in childhood will benefit left ventricular remodeling. Trans catheter PDA closure is now the recommended approach with reported minimal complications and outcomes similar to those of surgically closed PDA.[14] LVEF, LVEDD, and mPAP were identified as significant predictors of late LV systolic function. 15The devices were designed in a way so that the defects can be closed with transcatheter approach in preterm newborns.[16,17] up till adulthood. Most patients were asymptomatic. Some present with heart failure and pulmonary hypertension leading to Eisenmenger syndrome.[18] Failure to close PDA timely leads to development of pulmonary hypertension with one study stating that patients with moderate to large PDA tend to develop moderate to severe pulmonary hypertension with females being 1.8 times more at risk of developing pulmonary arterial disease.[19]

Eerola et al. showed that changes in left ventricular systolic function were caused by ductal closure in children.9 This study also reported that LVFS declined from 38.0% on day 1 to 36.8% after six months.[9] However, our study demonstrated a comparable decrease in LVFS from 36.00(32.00-40.00)% to 36.00(32.00-39.25) %.Similarly, Galal et al. was off the view that device closure of large PDA led to a significant immediate lowering of left ventricular systolic function in the pediatric age group with fractional shortening decreasing from 34% to 24% post closure.10 Similarly, the current study demonstrated that left ventricular ejection fraction (60.00(58.00-61.25)% vs. 58.00(54.75-60.00)%; p<0.001) reduced within 24 hours after transcatheter PDA closure, pointing towards impaired LV systolic function. Large and moderate PDA usually causes heart failure and failure-to-thrive in children.[20]

Our study found that the frequency of LV systolic dysfunction post device closure was 16.7%. This was similar to the studies performed by Jeong *et al.,*[6], which showed frequency of LV systolic function post device closure as 11%, Kim et al. 12 18.6% and Eerola *et al.,* 9 15.2%.

With regards to left heart dimensions, the left atrial diameter, LVEDD, LVESD and LVEDVI showed a significant decline (p<0.001) within 24 hours following PDA closure and showed a further decline which was significant, at one month and 3 months follow up. These findings were compatible to a certain extent to the previous study which showed a significant decline in the LVESD (*p*<0.001) at day-1 and at the short term follow up after PDA closure.[21] In addition, the study performed by Hou M et al. also showed a significant decline in LVEDD and LVESD (*p*<0.05) within 24 hours post PDA device closure.[22]

Keeping in view the above mentioned results of the study and comparison of the results with the previous studies, it is clear that patients undergoing PDA device closure should be evaluated post procedure for development of LV dysfunction which if severe would require timely management. However, severe LV dysfunction was not noted in our study population.

The aim of this study was also to highlight the factors which can lead to LV dysfunction as mentioned in the results. In addition, to highlight the importance of timely intervention which leads to LV remodeling and return of LV parameters to normal levels. These findings will help pediatric patients receiving this intervention to achieve better outcomes and make better clinical decisions

This study showed an early fall in LV systolic function within 24 hours after successful PDA closure which was transient and did not require any medical treatment. One month after device closure, this deterioration in LV systolic function began to improve as shown by improvement of LVEF, fractional shortening and GLS score.

LIMITATIONS OF THE STUDY

This study was carried out in a resource limited setting hence left ventricular function as well as dimensions were assessed with 2D echocardiography. Cardiac MRI is generally considered the gold standard for assessing LV function, volumes and dimensions.

CONCLUSION

Transcatheter PDA closure causes a decrease in LV performance immediately post procedural closure, which recovers completely within 3 months. Pre closure LAd/Aod ratio and PDA diameter were found to be an important markers in anticipating LV dysfunction post device closure.

Conflict of Interest: None.

Funding Source: None

Authors' Contribution

Following authors have made substantial contributions to the manuscript:

MWB: Concept, study design,data inerpretation & analysis, drafting the manuscript, approval of the final version to be published

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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