Predictors of transient left ventricular dysfunction following transcatheter patent ductus arteriosus closure in pediatric age



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Objectives: To evaluate the left ventricular function before and after transcatheter percutaneous patent ductus arteriosus (PDA) closure, and to identify the predictors of myocardial dysfunction post-PDA closure if present.

Interventions: Transcatheter PDA closure; conventional, Doppler, and tissue Doppler imaging; and speckle tracking echocardiography.

Outcome measures: To determine the feasibility and reliability of tissue Doppler and myocardial deformation imaging for evaluating myocardial function in children undergoing transcatheter PDA closure.

Patients and methods: Forty-two children diagnosed with hemodynamically significant PDA underwent percutaneous PDA closure. Conventional, Doppler, and tissue Doppler imaging, and speckle-derived strain rate echocardiography were performed at preclosure and at 48 hours, 1 month, and 6 months postclosure. Tissue Doppler velocities of the lateral and septal mitral valve annuli were obtained. Global and regional longitudinal peak systolic strain values were determined using two-dimensional speckle tracking echocardiography.

Results: The median age of the patients was 2 years and body weight was 15 kg, with the mean PDA diameter of 3.11 \triangleright 0.99 mm. M-mode measurements (left ventricular end diastolic diameter, left atrium diameter to aortic annulus ratio, ejection fraction, and shortening fraction) reduced significantly early after PDA closure (p < 0.001). After 1 month, left ventricular end diastolic diameter and left atrium diameter to aortic annulus ratio continued to decrease, while ejection fraction and fractional shortening improved significantly. All tissue Doppler velocities showed a significant decrease at 48 hours with significant prolongation of global myocardial function (p < 0.001) and then were normalized within 1 month postclosure. Similarly, global longitudinal strain significantly decreased at 48 hours postclosure (p < 0.001), which also recovered at 1 month follow-up. Preclosure global longitudinal strain showed a good correlation with the postclosure prolongation of the myocardial performance index.

Conclusion: Transcatheter PDA closure causes a significant decrease in left ventricular performance early after PDA closure, which recovers completely within 1 month. Preclosure global longitudinal strain can be a predictor of postclosure myocardial dysfunction.

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Introduction

The left to right shunting through hemodynamically significant patent ductus arteriosus (PDA) causes pulmonary overcirculation with resultant left ventricle (LV) volume overload and remodeling. The left ventricle compensates by increasing stroke volume, but in patients with greater shunts, it causes symptoms of congestive heart failure [1,2]. Transcatheter PDA closure is a well-established, safe, and effective procedure. It has become the treatment of choice, with closure rate exceeding 90–95%. Success rate is improving over time owing to device modifications, advancement of novel techniques, and increased operator skill [3]. Theoretically, PDA closure is supposed to alter the LV volume overload and remodeling with improvement of systolic and diastolic heart function gradually. However, some reports demonstrate an immediate deterioration in LV systolic performance, which recovers within a few months [1]. This study was conducted to evaluate the LV systolic and diastolic function before and after PDA closure in children using conventional two-dimensional, Doppler, tissue Doppler, and speckle-derived strain echocardiography, as both speckle tracking echocardiography and tissue Doppler imaging (TDI) have demonstrated the ability to detect early myocardial dysfunction in different diseases [4–6]. Furthermore, we have tried to determine the predictors of LV dysfunction following percutaneous transcatheter PDA closure.

Patients and methods

Patient characteristics

Forty-two children diagnosed with PDA and planned for percutaneous transcatheter ductal closure were enrolled in this study. This was a prospective collaborative study between Cairo University and Ain Shams Specialized Pediatric Hospitals. The interventional work of this study was performed at the Pediatric Cardiology Catheterization Laboratory of Cairo University. Patients were reviewed during the period from February 2012 till June 2014. The indication for PDA closure was a hemodynamically significant shunt causing the LV volume overload. The exclusion criterion was the coexistence of other hemodynamically significant congenital heart diseases or irreversible pulmonary vascular disease. An informed consent was obtained from the patients or their legal guardians after approval of the

Abbreviations

PDA	patent ductus arteriosus
LVEDD	left ventricular end diastolic diameter
LVEDV	left ventricular end diastolic volume
LA/AO r	atio left atrium to aortic diameter ratio
EF	ejection fraction
FS	fraction shortening
GLS	global longitudinal strain
TDI	tissue Doppler imaging
S	peak systolic annular velocity
Е	peak early diastolic annular velocity
Α	peak late diastolic annular velocity
MPI	myocardial performance index
PW	pulsed wave
ICT	isovolumetric contraction time
IVRT	isovolumetric relaxation time
BSA	body surface area
CI	confidence interval
ROC	receiver operating character
AUC	area under the curve

Ethical Committee of Ain Shams University of Medical Science. The work complies with the principles of the Declaration of Helsinki in 1964.

Transthoracic echocardiographic study

All patients who were scheduled for tranunderwent scatheter PDA closure twodimensional (2D) echocardiography, Doppler imaging, TDI, and speckle tracking imaging. The echocardiographic studies were obtained preclosure (within 1 week prior to ductal closure), early (within 48 hours postclosure), and then at 1 month and 6 months postclosure. Transthoracic echocardiography was performed by an expert pediatric cardiologist, with patients in the supine position, using VIVID 9 General Electronics (GE Ultrasound, Horten, Norway) with a 3-7 MHz phasedarray transducer. The M-mode measurements included interventricular septum, and LV internal and LV posterior wall diameters in systole and diastole. Ejection fraction (EF) and fraction of shortening (FS) were all measured in the parasternal long-axis view. In addition, the aortic and left atrial diameters were measured in parasternal long-axis view at the end of diastole. Twodimensional echocardiographic measurement included the PDA size at the pulmonic end in the high parasternal ductal view in the preclosure echocardiography. Pulsed wave tissue Doppler velocities were obtained at the cardiac base in the apical four-chamber orientation from two locations: the lateral mitral annulus and the interventricular septum. Peak systolic annular velocity (S'), peak early diastolic annular velocity (E'), and peak late diastolic annular velocity (A') were measured. Calculation of the global myocardial performance index (MPI) (Tei index) by pulsed wave TDI: intervals measurements were performed within one cardiac cycle. The Tei index was calculated as follows: Tei = isovolumetric contraction time + isovolumetric relaxation time/ejection time, for LV MPI using pulsed wave TDI at the lateral mitral annulus [7,8]. To reduce the effect of respiration on tissue velocities, and as breath holding is not applicable in young children, three cardiac cycles were recorded and the average velocity and average times were calculated.

Speckle-tracking echocardiography

Two-dimensional grayscale imaging from the apical and parasternal long-axis views was performed. A frame rate of 80-100 frames/s was used, with care to align the walls parallel to the ultrasound beam for apical images. Systolic and diastolic phases of the cardiac cycle were determined from simultaneous electrocardiographic monitoring. Three consecutive cardiac cycles were acquired and digitally stored for an offline analysis. Tracking was automatically performed, and when necessary, it was manually adjusted to cover the entire myocardial wall. The analysis was accepted after visual inspection and when the software indicated adequate tracking. Offline measurements of the two-dimensional strain and strain rate were obtained with Q analysis software using the EchoPACS workstation. The software automatically divided the cross-sectional image into six segments. The global LV longitudinal strain was calculated by averaging all the measured segmental values.

Cardiac catheterization and PDA closure

After obtaining informed consent, cardiac catheterization was done for closing the PDA with a percutaneous device. The procedure was performed under general anesthesia and antimicrobial prophylaxis. Arterial and venous access via femoral artery and vein was obtained during the study. Saturation and pressure measurements were performed from the aortic arch and the main pulmonary artery before PDA occlusion. PDA was closed by detachable coils Nit-Occluders or Amplatzer duct occluders, depending on the size of the duct and interventionist's decision. Post-PDA closure, aortic angiogram was performed to assure complete occlusion of the duct after 5 minutes of closure.

Statistical analysis

Variables were expressed as mean ± standard deviation. Differences between the followed

groups were estimated by *t* test for either paired or unpaired data. Linear regression analyses and partial correlation test by Pearson's method were performed to assess univariate relations. TDI comparisons with controls and the change in TDI parameters with time were analyzed by ANOVA with *post hoc* Bonferroni comparison; differences were significant at p < 0.05. The Bland–Altman plot analysis was used to determine the percentages (%) of mean difference and the percentages of limits of agreement between measurements [9]. Statistical Package for the Social Science (SPSS Inc., Chicago, IL, USA) release 15 for Microsoft Windows (2006) and MS Excel version 10 for MS Windows (Microsoft Corp., Redmond, WA, USA).

Results

Patient characteristics

The median age of the 42 PDA patients who underwent percutaneous closure was 2 years, with a median weight of 15 kg (Table 1). The mean PDA pulmonary end diameter was 3.11 ± 0.99 mm. All the patients underwent a safe procedure without complications and were re-evaluated by echocardiography within 48 hours, 1 month, and 6 months.

M-mode and 2D echocardiography

Initially by M-mode, PDA patients had significantly higher left ventricular end diastolic diameter (LVEDD) and left atrium diameter to aortic annulus (LA/AO) ratio than controls $(3.50 \pm 0.6$ cm vs. 3.13 ± 0.5 cm, p = 0.019, and 1.68 ± 0.4 vs. 1.42 ± 0.3 , p = 0.005, respectively; Table 2). By Bland–Altman analysis, PDA pulmonary end

Table 1. Demographic data of the studied population.

	Cases (<i>n</i> = 42)	Controls $(n = 20)$
Age (y) Mean ± SD	2.88 ± 2.76 (1-12)	3.66 ± 2.41 (1-9)
(range) Median	2	3
Weight (kg) Mean SD (range)	12.89 8.44 (5–41)	14.10 4.99 (6–28)
Median	10	13.25
BSA (m ²) Mean ± SD (range) Median	12.89 ± 8.44 (5-41) 10	14.10 ± 4.99 (6–28) 13.25
<i>Sex</i> Male Female	14 (27.5%) 37 (72.5%)	12(60%) 8 (40%)

BSA = body surface area; SD = standard deviation.

	,				
	Controls $(n = 20)$	Cases			
		Preclosure ($n = 42$)	Postclosure		
			48 h (<i>n</i> = 42)	1 mo $(n = 42)$	6 mo (<i>n</i> = 42)
LVEDD (cm)	3.13 ± 0.5^{a}	3.50 ± 0.6^{b}	$3.29 \pm 0.6^{\circ}$	3.07 ± 0.6^{d}	2.91 ± 0.6
LA/AO	1.42 ± 0.3^{e}	1.68 ± 0.4^{b}	$1.322 \pm 0.3^{\circ}$	1.16 ± 0.3^{d}	1.09 ± 0.3^{f}
EF (%)	68.6 ± 6.2	70.1 ± 6.5^{b}	$63.1 \pm 5.8^{\circ}$	67.5 ± 5.5^{d}	69.2 ± 5.6
FS (%)	38.4 ± 5.03	38.7 ± 5.3^{b}	$32.9 \pm 4.5^{\circ}$	36.5 ± 5.1^{d}	37.6 ± 4.6

Table 2. Comparison of left ventricular end diastolic diameter, left atrium/aorta ratio, ejection fraction, and fractional shortening between cases at baseline and post-PDA closure, and controls.

EF = ejection fraction; FS = fractional shortening; LA/AO = left atrium diameter to aortic annulus ratio; LVEDD = left ventricular end diastolic diameter; PDA = patent ductus arteriosus.

^a p = 0.019, comparison between controls and preclosure cases.

^b p < 0.001, comparison between preclosure and 48 hours.

^c p < 0.001, comparison between cases at 48 hours and 1 month.

^d p < 0.001, comparison between cases at 1 month and 6 months.

^e p = 0.005, comparison between controls and preclosure cases.

f p = 0.001, comparison between controls and cases at 6 months.



Fig. 1. Correlation between the measurements of pulmonary end of PDA by 2D echocardiography and angiography by Bland–Altman analysis. Angio = angiography; PDA = patent ductus arteriosus; Pul = pulmonary; SEE = standard error of the estimate; 2D = two dimensional.

diameter measured by 2D echocardiography had a significant correlation with that measured by angiography (Fig. 1). Following the transcatheter ductal closure, a significant reduction of LVEDD, EF, FS, and LA/AO was observed compared with preclosure values (p < 0.001). The LVEDD and LA/AO ratio continued to decrease significantly at 1 month (p < 0.001) and 6 months (p < 0.001) to reach values insignificantly different from those of the controls. There was a significant immediate decline of EF and FS at 48 hours, which improved at 1 month (p < 0.001) and continued to improve at 6 months (p < 0.001) (Table 2).

TDI echocardiography

The LV tissue Doppler velocities and MPI showed no significant difference compared with controls at baseline. There was a considerable reduction in E', A', S', and E'/A' ratio immediately

after PDA closure. All these measurements significantly increased at 1-month follow-up and continued to improve at 6 months (p < 0.001). Regarding the MPI, there was a significant prolongation at 48 hours postclosure (p < 0.001), which reverted to the preclosure values at 1 month and showed no change at 6 months (Table 3 and Fig. 2A). The patients with a significantly prolonged MPI (>20%) post-PDA closure were of significantly younger age (p = 0.041), had higher preclosure global longitudinal strain (GLS) (p = 0.043) as well had significantly decreased GLS at 48 hours (Table 4). By multivariable binary logistic regression analysis for the predictors of significant (>20%) prolongation of the MPI from the preprocedure value, only preclosure GLS showed a good correlation with postclosure prolongation of the MPI, denoting its ability in predicting postclosure myocardial dysfunction (Fig. 2B). A plot of all the previously suggested

ORIGINAL ARTICLE

	Controls $(n = 20)$	Cases			
		Preclosure $(n = 42)$	Postclosure	Postclosure	
			48 h (<i>n</i> = 42)	$1 \mod (n = 42)$	6 mo (<i>n</i> = 42)
<i>E</i> ′ s (cm/s)	0.12 ± 0.02	0.12 ± 0.02^{a}	0.10 ± 0.02^{b}	$0.11 \pm 0.02^{\circ}$	0.11 ± 0.02
A' s (cm/s)	0.06 ± 0.02	0.068 ± 0.02^{a}	0.061 ± 0.02^{d}	$0.069 \pm 0.02^{\circ}$	0.07 ± 0.01
S' s (cm/s)	0.07 ± 0.01	0.071 ± 0.01^{e}	0.066 ± 0.01^{b}	$0.067 \pm 0.01^{\circ}$	0.083 ± 0.2
E'/A' s	2.06 ± 0.74	1.84 ± 0.66^{a}	1.778 ± 0.77^{b}	$1.702 \pm 0.60^{\circ}$	1.78 ± 0.66
E' 1 (cm/s)	0.12 ± 0.03	0.112 ± 0.02^{a}	0.108 ± 0.08^{b}	$0.103 \pm 0.03^{\circ}$	0.107 ± 0.03
A' l (cm/s)	0.05 ± 0.01	0.06 ± 0.02^{f}	0.049 ± 0.01^{g}	$0.055 \pm 0.02^{\circ}$	0.06 ± 0.02
S' 1 (cm/s)	0.06 ± 0.02	0.064 ± 0.01^{h}	0.060 ± 0.01^{g}	$0.058 \pm 0.01^{\circ}$	0.06 ± 0.01
E'/A' 1	2.42 ± 1.03	2.090 ± 0.96^{i}	1.997 ± 0.72^{d}	$1.999 \pm 0.8^{\circ}$	2.094 ± 0.8
MPI	0.39 ± 0.05	0.38 ± 0.05^{a}	0.44 ± 0.05^{b}	0.38 ± 0.06	0.38 ± 0.06

Table 3. Comparison of myocardial function by tissue Doppler velocities and myocardial performance index between cases at baseline and post-PDA closure, and controls.

A' l = mitral peak late diastolic lateral annular velocity; A' s = interventricular septum peak late diastolic velocity; E' l = mitral peak early diastolic lateral annular velocity; E' s = interventricular septum peak early diastolic velocity; l = lateral; MPI = myocardial performance index; PDA = patent ductus arteriosus; s = septal; S' l = lateral annulus peak systolic velocity; S' s = interventricular septan peak systolic velocity; S' s = interventricular septan peak systolic velocity.

^a p < 0.001, comparison between preclosure and 48-hour measurement.

^b p < 0.001, comparison between Preciosite and 40-hour measurements.

^c p < 0.001, comparison between 1-month and 6-month measurements.

^d p = 0.002, comparison between 48-hour and 1-month measurements.

^e p = 0.017, comparison between preclosure and 48-hour measurements.

^f p = 0.024, comparison between preclosure and 48-hour measurements.

^g p = 0.006, comparison between 48-hour and 1-month measurements.

^h p = 0.003, comparison between preclosure and 48-hour measurements.

 i p = 0.05, comparison between pre closure and 48-hour measurements.



Fig. 2. (A) Changes in MPI before and after PDA closure. (B) ROC curve derived from the multivariable binary logistic regression model for prediction of the occurrence of significantly (> 20%) prolonged MPI from preprocedure value. CI = confidence interval; MPI = myocardial performance index; PDA = patent ductus arteriosus; ROC = receiver-operating characteristic.

Table 4. Comparison of patients with significantly (> 20%) or insignificantly (< 20%) prolonged MPI post-PDA closure.^a

Variables	Insignificantly (< 20%) prolonged MPI (<i>n</i> = 29)	Significantly (> 20%) prolonged MPI $(n = 13)$	р
Age (y)	2.0 (1.2–4.3)	1.0 (0.8–2.5)	0.058
$BSA(m^2)$	0.59 (0.26)	0.49 (0.22)	0.243
PDA diameter (mm)	3.1 (0.9)	3.4 (0.8)	0.484
ESPAP (mmHg)	30.9 (14)	35.8 (13.2)	0.296
LVEDD (z-score)	2.9 (1.4)	3.0 (1.7)	0.879
Preclosure MPI	0.36 (0.05)	0.39 (0.03)	0.041
Preclosure GLS	-20.1 (8.0)	-23.8 (3.6)	0.043
GLS at 48 h	-19.3 (3.0)	-18.8 (2.4)	0.633
Percent change of GLS	-10.0 (7.2)	-20.2 (9.8)	0.001
Significant decrease of GLS (> 20%)	4 (13.8%)	10 (76.9%)	0.0001

BSA = body surface area; ESPAp = estimated systolic pulmonary artery pressure; GLS = global longitudinal strain; LVEDD = left ventricular end diastolic diameter; MPI = myocardial performance index; PDA = patent ductus arteriosus; SD = standard deviation.

^a Data are presented as mean (SD) or number (%).

Variable	Regression coefficient	SE	р	Odds ratio	95% CI for odds ratio
Age (y)	-0.32	0.22	0.133	0.72	0.47-1.10
PDA diameter (mm)	0.33	0.45	0.462	1.39	0.58-3.38
LVEDD (z-score)	-0.13	0.28	0.639	0.88	0.51-1.52
Preprocedure GLS (%)	-0.25	0.13	0.049	0.78	0.61-0.999
Model diagnostics					
–2 log likelihood test		p = 0.04	8		
Area under ROC curve		0.78 (95	% CI, 0.63–0.8	89)	
Best cutoff criterion		Predicted probability > 0.238			
Sensitivity (%)		84.6 (95	% CI, 54.6–98	5.1)	
Specificity (%)		58.6 (95	% CI, 38.9–76	5.5)	
Correct classification rate	(%)	78.6			

Table 5. Multivariable binary logistic regression analysis for predictors of the occurrence of significantly prolonged MPI (> 20%) from preprocedure value.

CI = confidence interval; GLS = global longitudinal strain; LVEDD = left ventricular end diastolic diameter; MPI = myocardial performance index; PDA = patent ductus arteriosus; ROC = receiver operating characteristics; SE = standard error.

predictors in a receiver operating characteristic curve showed an overall predicted probability of all the variables of >0.238 with sensitivity 84.6% (95% CI, 54.6–98.1) and specificity 58.6% (95% CI, 38.9–76.5), area under receiver operating characteristic curve of 0.78 (95% CI, 0.63–0.89), and a correct classification rate of 78.6%, denoting that the collective use of all the previous parameters can be helpful in determining the postclosure myocardial dysfunction and MPI prolongation (Table 5).

Left ventricular GLS

There was a significant decrease in GLS immediately after the closure compared with the preclosure measures (p < 0.001) with significant improvement at 1-month follow-up (p = 0.0026) (Fig. 3).

Discussion

The current study demonstrated an early deterioration of LV function following successful transcatheter ductal closure, which was subclinical but evident by TDI and speckle tracking echocardiographic parameters. Previously published studies have demonstrated acceptable accuracy



Fig. 3. Changes of global longitudinal strain of left ventricle at baseline and after PDA closure. PDA = patent ductus arteriosus.

and reliability of TDI and speckle tracking echocardiographic modalities in assessing regional myocardial function in children and adults [9,10]. As regards the LV dimensions following ductal closure, an immediate significant decrease of LVEDD and LA/AO ratio was observed at 48 hours, which continued to decrease further at 1 month and 6 months. At 6 months, LVEDD showed no significant difference from that of controls. These findings are secondary to the relief of LV volume overload following PDA closure. Both EF and FS showed a significant decline at 48 hours postclosure, which was increased at 1 month and continued up to 6 months, denoting considerable improvement of the myocardial function assessed by conventional echocardiography. These findings are in accordance with the previously published data on pediatric patients [11–14]. These changes could be explained by the fact that PDA is associated with the left to right shunt, which increases the LV preload. Based on the Frank-Starling's law, an increase in preload culminates in augmented contractility (FS and EF), while PDA closure results in a sudden drop in LV preload and thus a decrease in systolic performance. Another rationale for this observation is a sudden increase in the afterload, which is due to termination of blood flow through PDA and the low-resistance pulmonary circulation that contributes to systolic dysfunction [15]. By TDI, there was a significant decline of all tissue Doppler velocities as well as E'/A' ratio immediately after PDA closure, which was reverted at 1 month, denoting improvement of myocardial function. These results partially agreed with that of Amoogzar et al [16] who reported a significant decrease in E' and E'/A'ratio, but not in A' or S' wave, at 48 hours and improvement in these parameters at 1-month follow-up. Moreover, MPI became significantly prolonged at 48 hours but started to normalize at 1 month. These changes denote immediate and transient LV myocardial dysfunction following transcatheter ductal closure. These findings are in accordance with that of Noori et al [17]who studied 23 neonates before and after PDA ligation. As the MPI is inversely related to global myocardial function, an increase in the MPI indicated a deterioration of cardiac function. However, the LV MPI began to normalize even though the loading conditions remained unchanged. Therefore, although a sudden decrease in preload immediately after ligation likely contributed to the increase in the LV MPI, the data suggest that transient deterioration in global cardiac function also occurred, possibly due to the acute decrease in myocyte fiber length after the sudden decrease in LV loading volume [16]. In the current study, further analysis for MPI post-PDA closure was performed, and 20% change in the MPI was considered significant prolongation. In the current study, 31% (*n* = 13) had a significant change in the MPI in contrast to a nonsignificant change in 69% (*n* = 29). In our cohort, the younger age at the preclosure (1.9 years vs. 3.4 years, p = 0.041) and the longer preclosure MPI (p = 0.041) were the characteristic factors among the patients with a significant change in the MPI. None of the suggested variables (body surface area, age, PDA pulmonary end diameter, systolic pulmonary artery pressure, and LVEDD) showed a correlation with the prolonged MPI postclosure. The fact that patients with a significant change in the MPI had younger age and a prolonged baseline MPI could be interpreted as follows: the MPI shows progressive prolongation until the age of 3 years and shows no further change afterward. The agerelated changes in the MPI may reflect changes in myocardial maturation with changes in total collagen and proteins [18]. However, no significant correlation was found between MPI prolongation and the different variables chosen in our cohort. In contrast, Gupta et al [1] reported that the baseline LV EF and left ventricular end diastolic volume (LVEDV), and PDA diastolic gradient predicted postclosure LV dysfunction. However, the preclosure GLS in patients with a significantly prolonged MPI was significantly higher before PDA ligation and showed a significant drop at 48 hours postclosure as well. By multivariate analysis, GLS was proved to be a significant single predictor for postclosure prolongation in the MPI. Regarding GLS, a significant decrease at 48 hours postclosure has been shown, with marked improvement afterward at 1-month follow-up, suggesting a transient depression of systolic function at 48 hours. This agrees with other published studies on post-PDA surgical or percutaneous closure. They have assumed that a larger LV deforms less with lower strain values to generate the same LV output. Moreover, with a greater stroke volume and a higher output state, strain values were highest in preclosure time and decreased in early postclosure in parallel to deterioration of LV systolic function [12,15].

Study limitations

The small sample size of the current study could have hindered the possibility of eliciting small differences in the variables affecting the LV dysfunction. In addition, the parameters used for the evaluation of LV systolic function, although in concordance with previous studies, were preload dependent and could influence the interpretations in the postclosure status. In addition, closer intervals of follow-up of patients may have detected fine changes that change rapidly. Furthermore, we did not try any medical treatment for patients with an evident decrease in myocardial function post-PDA closure, which could have highlighted the possible changes in myocardial function and need for transient postclosure medical treatment.

Conclusion

Transcatheter PDA closure causes a significant transient decrease in LV performance, especially at younger age, which recovers completely at 6 months following the procedure. Preclosure GLS can be used as a predictor of this postprocedure LV myocardial dysfunction.

Conflict of interest

The authors declare that there is no conflict of interest.

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