Long-term Follow-up Results of Lung Perfusion Studies after Transcatheter Closure of Patent Ductus Arteriosus

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ABSTRACT

Objective. This study presents the long-term follow-up of patients who developed left lung perfusion (LLP) abnormalities following patent ductus arteriosus (PDA) closure with various device types.

Design. The study includes 23 adult and pediatric patients who had undergone transcatheter PDA closure and were shown to have decreased LLP (<40%) by the first scintigraphy performed within the average follow-up period of 14.0 ± 8.12 months (2.0–30 months). For PDA closure, the Amplatzer duct occluder was used in 12 patients, and coils were used in 11. Within the average period of 58.91 ± 12.93 months (37–85 months) after transcatheter PDA closure, a second lung perfusion scintigraphy was performed.

Results. In 13 out of 23 patients (56.5%), LLP improved by the time of the second scintigraphy. Improved and unimproved patients did not differ with regard to age, weight, body surface area, PDA diameter, ampulla diameter, and PDA length at the time of PDA closure and the second scintigraphy. There was no significant difference with regard to the percent of improved patients between the different device types ($P = .88$). The left pulmonary artery indexes were also insignificantly different ($P = .446$). Patients with persistent LLP abnormality had significantly higher average Doppler velocity index [(LPA blood flow velocity—RPA blood flow velocity) / MPA blood flow velocity] × 100 ($P = .007$) and PDA diameter/length. If Doppler velocity index ≥50% is taken as the cutoff value, it is possible to predict persisting LLP abnormality with 80% sensitivity and 76% specificity. Left lung perfusion abnormality was found to persist in patients with PDA diameter/length ≥0.5 with 80% sensitivity and 92.3% specificity.

Conclusions. The LLP abnormalities seen after PDA closure with various devices eventually improve to normal in the majority of patients during long-term follow-up. Patients whose PDA length is shorter than its diameter are at risk of developing LLP abnormalities that persist long-term.

Key Words. Transcatheter Closure; Patent Ductus Arteriosus; Lung Perfusion; Scintigraphy

Introduction

Transcatheter closure of patent ductus arteriosus (PDA) was first described by Porstmann et al. and has since found wide use due to its efficacy, reliability, and variety of available devices.[1] However, despite this method being considered safe and effective, complications such as residual shunt, device embolization, hemolysis, and decreased pulmonary blood flow distribution have been reported.[2] Given the anatomical proximity of PDA and the left pulmonary artery (LPA), it has been thought that transcatheter PDA closure may lead to a decrease in LPA blood flow, and research has been done on the issue.[3,4] It has been reported that the use of practically any existing PDA closure device results in varying degrees of decreased LPA blood flow.[5,6] As far as our knowledge goes, there have been few studies focusing on the long-term follow-up of left lung perfusion (LLP), while the number of studies researching the effects of the Amplatzer duct occluder (ADO; AGA Medical Corporation, Golden Valley, MN, USA) use is particularly limited. In this study, we present the follow-up of patients in whom ADO and coils had been used for PDA closure and whose LLP decreased after the procedure.

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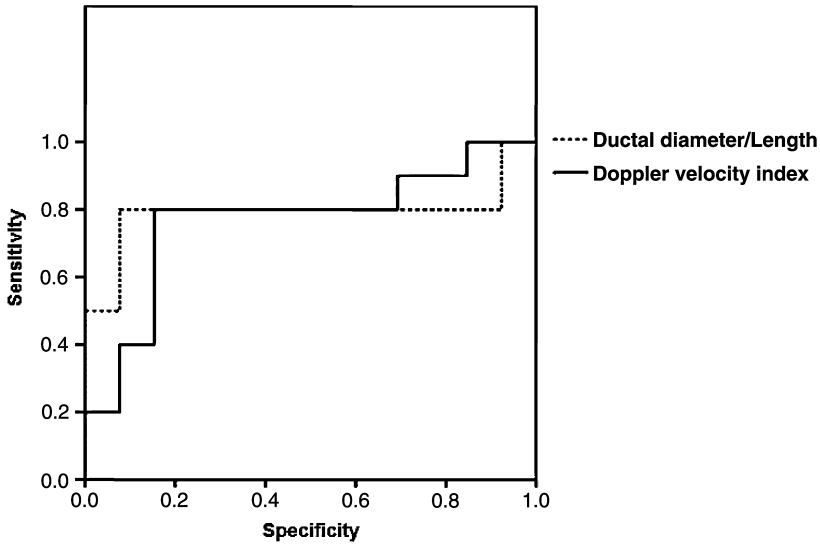


Figure 1. Receiver operating characteristic curve analysis for left lung perfusion abnormality.

Materials and Methods

Patients

Enrolled in the study were 28 adult and pediatric patients who had undergone transcatheter PDA closure at our clinic between 2003 and 2006 and who were found to have decreased pulmonary blood flow using the radionuclide method after a follow-up of 14 ± 8.1 months (range 2–30 months). Out of 28 patients with abnormal LLP (<40%), as shown by the first scintigraphy, 1 was excluded due to pregnancy and 4 due to failing to attend follow-up. The remaining 23 patients underwent a second scintigraphy after an average period of 44.9 ± 10.5 months (range 29–62 months) after the first scintigraphy and 58.9 ± 12.9 months (range 37–85 months) after transcatheter PDA closure, and these 23 adult and pediatric patients with abnormal LLP (<40%) were prospectively chosen for this study.

For PDA closure in these 23 cases, ADO was used in 12 patients and coils were used in 11—a Cook detachable coil (William Cook Europe, Bjaeverskov, Denmark) in 8 and a Gianturco coil (Cook Inc., Bloomington, IN, USA) in 3. The patients' age, weight, body surface area (BSA), and ductus size at the time of PDA closure as well as the results of the first scintigraphy were obtained from the patients' records.

Echocardiographic Examination

Before the patients with decreased artery blood flow had radionuclide imaging performed, their height and weight were taken and the main pulmonary artery (MPA), right pulmonary artery (RPA), and LPA diameters, and blood flow velocity were measured echocardiographically. In the suprasternal view, the end-systolic RPA and LPA diameters were measured five times, and the average diameter was calculated (Figure 1). The RPA and LPA indexes were calculated by dividing the RPA and LPA diameters by the BSA. Pulmonary blood flow velocities were measured (five times for each artery) using pulsed wave Doppler in the parasternal short-axis view and directly above the pulmonary valve for the MPA, in the upper parasternal short-axis view (and if device protrusion was present, at the distal end of the protrusion) for the LPA, and at the proximal end of the RPA. The Doppler velocity index (DVI) was calculated using the formula DVI = [(LPA blood flow velocity – RPA blood flow velocity) / MPA blood flow velocity] $\times 100^3$.

All examinations were performed by a researcher blinded to the results of the first scintigraphy.

Lung Perfusion Scintigraphy

Lung perfusion scintigraphy was performed using technetium-99 m macroaggregated human albumin and a gamma camera system. Technetium-99 m macroaggregated human albumin was administered through the peripheral vein in doses no less than 10 MBq at the rate of 1.2 MBq/kg (100 000–150 000 particles). Images consisting of 400 000 counts were recorded in the

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anterior and posterior projections. The LLP percentage was defined as the ratio of the average anterior and posterior radioactive count to the total lung count. Total lung perfusion values that were below 40% were considered decreased.[3,4,7,8]

In patients whose LLP improved, we evaluated the correlation between the improvement and the patients' age, weight, BSA, minimal ductus diameter (D1), ampulla diameter (D2), ductus length (L), ductus type, type of device used, LPA blood flow velocity, DVI, and the LPA diameter.

Statistical Analysis

The SPSS statistical program for Windows, version 15 (SPSS Inc., Chicago, IL, USA) was used to perform data analysis. The Shapiro-Wilk test was used for the analysis of compliance with normal distribution. Normally distributed continuous data is presented as mean ± standard deviation and nominal variables are presented as counts and/or percentages. Nonnormally distributed continuous data is presented as mean ± standard deviation with the median parameters in brackets. For statistical comparison of group data, Student's *t*-test was used for normally distributed continuous variables and Mann–Whitney *U*–test for nonnormally distributed continuous variables. Incidences in groups were tested for significance using the chi-square test and Fisher's exact test.

The correlations between parameters were analyzed using the Spearman rho correlation coefficient. In order to determine the optimal cutoff value for variables predicting considerably decreased lung perfusion, a receiver operating characteristic (ROC) curve was used. All statistical tests were two-sided. A P value of <.05 was considered statistically significant.

Results

Patient Characteristics

After transcatheter PDA closure, the first scintigraphy was performed in 130 patients. The first scintigraphy showed LLP to be decreased in 15 out of 41 patients (37%) using the ADO, 10 out of 49 (20%) using a Cook detachable coil, and in 3 out of 40 (7.5%) using a Gianturco coil.

Out of the 23 patients who had a second scintigraphy performed, 17 (74%) were female and 6 (26%) were male. The average age was 10.61 ± 11.89 years (range 4.75–60 years), average weight 30.5 ± 19.0 kg (range 12.4–82 kg), BSA 1.01 ± 0.39 m^2 (range 0.55–1.92 m^2). The ADO was used in 12 patients (mean device size 10.5 ± 1.5 mm, median 10 mm, range 8–14 mm), a Cook detachable coil in 8 and a Gianturco coil in 3. An 8 × 5 Cook detachable coil was used in three patients, a 6.5 × 5 one in two, and a 5 × 5, 5 × 4, and 5 × 3 coil in one patient each. Two patients in whom an 8 × 5 Cook detachable coil was used had residual shunt. During the same session, one of them was additionally implanted with a 5 × 3 and a 3 × 4 coil, while the other was implanted with a 6.5 × 5 coil. The patient who was first implanted with a 6.5 × 5 Cook detachable coil and had residual shunt then had a 5 × 5 coil implanted during the same session. All Gianturco coils were 4 × 4.

In patients who had the Cook detachable and/or Gianturco coils implanted, the total number of loops that protruded into the pulmonary artery was 1.5 loops in five patients (two patients had two coils implanted), 1 loop in four patients, 0.5 in one patient, and 2.75 loops in one patient (three coils were implanted).

The average period between the first and the second scintigraphy was 44.9 ± 10.5 months (range 29–62 months), and the average period between PDA closure and the second scintigraphy was 58.9 ± 12.9 months (range 37–85 months). The patients' demographic characteristics and PDA parameters are listed in Table 1. Patent ductus arteriosus morphology types were as follows: conical in 17 patients (73.9%), window type in 1 (4.3%), tubular in 4 (17.4%), and atypical in 1 (4.3%).

Lung Perfusion Scan

Out of the 23 patients with LLP below 40% at the time of the first scintigraphy, in 13 (56.5%), the LLP percentage improved to normal by the time of the second scintigraphy (from 36 ± 3.4% to 43.5 ± 2.8%, $P < .0001$).

While there were three patients with significantly decreased LLP (<30%) during the first scintigraphy (27.8%, 29.5%, and 28.1%), there were no patients with LLP < 30% during the second scintigraphy (34.94%, 37.67%, and 30.50%, respectively).

Taking all patients into account, the average LLP percentage after the first scintigraphy was 34.95 ± 4.02% (range 27.85–39.88%), and 40.64 ± 4.33% (range 30.5–49.9%) after the second one.

Left lung perfusion remained decreased up to the time of the second scintigraphy in 5 out of 11 (45.4%) patients using coils, and 5 out of 12 (41.6%) patients using ADOs. The second scintigraphy showed the LLP percentages to be similar

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Table 1. Demographic Data and Echocardiographic, Angiographic and Scintigraphic Findings for All Patients and Patients with Normal and Abnormal Left Lung Perfusion

Variable	All Patients (n = 23)	Abnormal Perfusion (n = 10)	Normal Perfusion (n = 13)	*P*
Age, years*	6.7 ± 12.2	6.5 ± 8.5 (1.5)	6.9 ± 14.8 (2.3)	.563
Age, years†	10.6 ± 11.8	9.0 ± 7.4 (5.5)	11.8 ± 14.6	.148
Weight, kg*	17.9 ± 18.6	19.1 ± 21.7 (9.1)	17.0 ± 16.8	.522
Weight, kg†	30.51 ± 19.02	27.4 ± 19.4	32.8 ± 19.1	.208
BSA, m^2*	0.66 ± 0.44	0.67 ± 0.51 (0.43)	0.64 ± 0.39 (0.59)	.522
BSA, m^2†	1.01 ± 0.39	0.93 ± 0.42 (0.84)	1.05 ± 0.38 (0.86)	.208
D1, mm	4.73 ± 2.38	5.37 ± 3.31 (4.60)	4.23 ± 1.26 (4.20)	.748
D2, mm	11.45 ± 3.81	9.93 ± 3.17 (10.20)	12.51 ± 3.99 (11.00)	.313
L, mm	10.03 ± 3.28	8.62 ± 3.39 (8.75)	11.13 ± 2.84 (10.90)	.088
D1/D2	0.47 ± 0.31	0.40 ± 0.23 (0.36)	0.35 ± 0.12 (0.33)	.780
D1/L	0.54 ± 0.37	0.74 ± 0.49 (0.55)	0.38 ± 0.11 (0.39)	.018
D2/L	1.23 ± 0.43	1.28 ± 0.48 (1.12)	1.18 ± 0.40 (1.15)	.648
D1/BSA	10.42 ± 8.37	11.96 ± 10.47 (11.96)	8.68 ± 5.34 (8.80)	.693
Peak LPA velocity (m/s)	1.72 ± 0.45	1.92 ± 0.42 (1.87)	1.56 ± 0.42 (1.68)	.077
DVI (%)	50.76 ± 37.86	71.4 ± 42.6	34.8 ± 25.2	.007
LPA index (mm/m^2)	11.71 ± 2.29	11.95 ± 2.62	11.52 ± 2.14	.446
1st LLP (%)	34.95 ± 4.02	33.46 ± 4.39 (33.68)	36.08 ± 3.46 (37.5)	.148
2nd LLP (%)	40.64 ± 4.33	36.91 ± 0.75 (37.60)	43.49 ± 2.86 (43.57)	.000
1st LPS time, months	14.08 ± 8.12	11.10 ± 9.02 (8.50)	16.23 ± 6.88 (18.0)	.115
2nd LPS time, months	58.91 ± 12.93	54.10 ± 9.25 (54.5)	62.61 ± 14.43 (56.0)	.446

*During PDA closure.
†During the second scintigraphy.
BSA, body surface area; D1, minimal ductal diameter; D2, ductal ampulla diameter; L, ductal length; LPA, left pulmonary artery; DVI, Doppler velocity index; LLP, left lung perfusion; LPS, lung perfusion scintigraphy.

for both device types (40.3 ± 4.8 vs. 40.9 ± 3.8%, $P = .88$). No statistically significant difference was found between device types in terms of the number of patients whose LLP improved by the time of the second scintigraphy, (six cases in the coil group and seven cases in the duct occluder group, $P = .88$; Table 2).

Improved and unimproved (at the time of the second scintigraphy) patients did not differ significantly with regard to age, weight, and BSA ($P > .05$, $P > .05$, $P > .05$, respectively). However, in 7 out of 11 (63.6%) patients who weighed under 10 kg at the time of PDA closure, LLP abnormality persisted. Out of all ductus parameters, the ratio of D1 to L was lower in improved patients as opposed to the unimproved ones ($P = .018$; Table 1).

Left Pulmonary Artery Velocity and DVI

Taking all patients into account, the average blood flow velocity was 1.72 ± 0.45 mps (range 0.58–2.56 mps) in the LPA, 1.12 ± 0.27 mps (range 0.42–1.60 mps) in the RPA, and 1.19 ± 0.23 mps (range 0.72–1.56 mps) in the MPA. Improved and unimproved patients did not differ significantly in terms of LPA blood flow velocity (respectively, 1.56 ± 0.42 vs. 1.92 ± 0.42, $P = .077$). Despite the lack of statistical difference between groups with improved and unimproved LLP, the LPA blood flow velocity was higher in the unimproved group ($P > .077$). No significant difference was found between the ADO and coil groups in terms of LPA blood flow velocity (1.83 ± 0.42 vs. 1.59 ± 0.46, respectively, $P = .379$; Table 2).

Table 2. Comparison of the ADO and the Coil Groups

Parameter	ADO Group	Coil Group	*P*
Age, years*	3.1 ± 5.1 (1.72)	10.7 ± 16.4 (4.0)	.051
Age, years†	7.1 ± 4.5 (5.6)	14.4 ± 16.1 (8.2)	.230
Weight, kg*	13.5 ± 16.5 (8.5)	37.9 ± 20.3 (35.3)	.027
Weight, kg†	23.6 ± 15.1 (20.1)	22.7 ± 20.4 (14.5)	.037
BSA, m^2*	0.53 ± 0.39 (0.41)	0.79 ± 0.46 (0.61)	.023
BSA, m^2†	0.85 ± 0.34 (0.81)	1.1 ± 0.39 (1.1)	.037
D1, mm	6.2 ± 2.2 (5.55)	3.0 ± 0.97 (3.1)	.000
D2, mm	11.3 ± 3.9 (9.9)	11.5 ± 3.8 (11.8)	.310
L, mm	11.4 ± 2.6 (10.8)	8.5 ± 3.3 (7.8)	.016
D1/D2	0.43 ± 0.11 (0.39)	0.31 ± 0.22 (0.2)	.004
D1/L	0.58 ± 0.31 (0.48)	0.49 ± 0.44 (0.4)	.379
D2/L	1.00 ± 0.27 (1.01)	1.4 ± 0.45 (1.5)	.016
D1/BSA	14.8 ± 8.3 (12.04)	4.9 ± 2.4 (4.4)	.000
Peak LPA velocity (m/s)	1.83 ± 0.42 (1.81)	1.59 ± 0.46 (1.77)	.379
DVI (%)	44.9 ± 32.1 (37.8)	57.1 ± 43.9 (60.5)	.487
LPA index (mm/m^2)	12.1 ± 2.1 (11.6)	11.2 ± 2.4 (10.7)	.379
1st LPS (%)	33.3 ± 4.1 (33.8)	36.6 ± 3.2 (37.9)	.059
2nd LPS (%)	40.3 ± 4.8 (40.6)	40.9 ± 3.8 (40.4)	.880
1st LPS time, months	9.9 ± 7.9 (5.0)	18.4 ± 5.7 (18.0)	.019
2nd LPS time, months	53.6 ± 6.8 (53.5)	64.6 ± 15.7 (61.0)	.151

*During PDA closure.
†During the second scintigraphy.
BSA, body surface area; D1, minimal ductal diameter; D2, ductal ampulla diameter; L, ductal length; LPA, left pulmonary artery; DVI, Doppler velocity index; LLP, left lung perfusion; LPS, lung perfusion scintigraphy.

Average DVI among all patients was 50.76% ± 37.86 (range –4.00–151%). Average DVI was

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significantly higher in patients with persisting LLP abnormalities as opposed to patients with improved LLP (71.42% ± 42.62 vs. 34.86% ± 25.20, $P = .007$). Doppler velocity index was >50% in 8 out of 10 (80%) patients with LLP <40% and in 3 out of 13 (23%) with LLP >40%. No significant difference in terms of DVI was found between the ADO and coil groups (44.9 ± 32.1 vs. 57.13 ± 43.94, $P = .487$).

Diameter of the Pulmonary Arteries

The average LPA index among all patients was 11.71 ± 2.29 mm/m^2 (7.14–16.92), while the average RPA index was 12.81 ± 2.04 mm/m^2 (9.23–18.15). No significant difference was found between the LPA and RPA indexes of all patients ($P = .09$). When compared, no significant difference was found between the LPA indexes of improved and unimproved patients (11.95 ± 2.62 vs. 11.52 ± 2.1 mm/m^2, $P = .446$), or between those using ADO and coils (12.11 ± 2.13 mm/m^2 vs. 11.27 ± 2.49 mm/m^2, $P = .379$).

No statistically significant correlations were found between the LPA indexes and LPA blood flow and DVI of all patients ($P = .455$, $P = .833$, respectively).

A statistically significant negative medium-degree correlation was found between the LLP values and the DVI and D1/L ratios at the time of the second scintigraphy ($r = –0.565$, $P = .005$ and $r = –0.492$, $P = .017$, respectively), while a statistically significant positive medium-degree correlation was found between the LLP and D2 values at the time of the first scintigraphy ($r = 0.480$, $P = .021$ and $r = 0.544$, $P = .007$, respectively).

The ROC curve analysis determined a D1/L average of ≥0.5 (sensitivity 80%, specificity 92.3%, area under the ROC curve = 0.792, $P = .018$) and DVI of ≥50% (80% sensitivity and 76% specificity, area under the ROC curve = 0.769, $P = .030$) as the most distinctive cutoff value for predicting LLP abnormality (Figure 1).

Discussion

Protrusion of the device into the LPA after percutaneous transcatheter PDA closure was first described in 1988 by Dyck et al.[5] In the following years, Stromberg et al.[9] reported a 90% protrusion rate for patients using Gianturco coils, while Soares et al.[3] reported a 94.7% rate, and Sreeram et al.[4]—a 40% protrusion rate for 32 patients using detachable coils. However, all these studies report a weak correlation between the protrusion of the device into the LPA and LLP. Additionally, the incidence of LPA gradients before PDA closure and the relationship of these gradients to late LPA stenosis after successful PDA occlusion had been evaluated by Gowda et al., who found that the incidence of LPA stenosis can be as high as 10–15% in patients with PDA prior to closure.[10]

Nevertheless, there are many reports about device-related LPA stenosis in medical literature. Left pulmonary artery obstruction after transcatheter PDA closure with ADO has been put forward as the main ADO-related morbidity cause.[11] In their study of 68 patients who underwent PDA closure with ADO and had a lung perfusion scan performed, Kharouf et al. report that while no LPA stenosis had been previously seen and no pressure drop had been detected in the LPA after the procedure, 17% of patients were found to have decreased LLP. Polat et al. report decreased LLP during short- and midterm follow-up in 17 out of 47 patients.[12,13] Kramoh et al. report a 31% rate of LLP abnormalities in all patients within 24 hours of PDA closure with Gianturco coils, ADO, and the Rashkind device,[6] while Soares et al. report a decrease in LLP within the average 81 ± 73.4-day period following PDA closure in 7 out of 70 patients.[3] In a previous study done at our clinic, LLP abnormalities were detected in 28 out of 130 (21.5%) patients who had undergone PDA closure with Gianturco coils, detachable coils, and ADO. In addition to protrusion and distortion caused by the device itself, ductal tissue retraction, fibrosis, or invasion into the LPA have all been suggested as the possible causes of LLP abnormalities following transcatheter PDA closure.

Few studies focusing on long-term follow-up of LLP abnormalities are mentioned in medical literature. The majority of the existing ones aim to determine LLP frequency and the relation between LLP abnormality and pulmonary artery blood flow velocity.[3–5,9,14] As far as we are aware, in our study, patients with LLP abnormalities developed after transcatheter PDA closure are followed for the longest period of time. Out of the 23 patients shown to have LLP abnormalities by the first scintigraphy, 13 (56.5%) showed improvement in LLP during the second scintigraphy performed after an average period of 58.9 ± 12.9 months (55.0 months). With the exception of one patient, all patients showed an increase in LLP percentage as compared to the first scintigraphy. Kramoh et al. report improvement in 23 out of 35 (65.7%) of patients with previously decreased LLP within the average period of 20.6 ± 23.6 months.

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The same study reports that out of nine patients whose LLP remained decreased, five showed improvement within the average period of 48.4 ± 32.7 months.[6] Beside this study, no other reports of long-term follow-up of LLP abnormalities have been published. In a study by Carey et al., LLP gradually improved and the increased LPA flow receded within a 12- to 15-month period in four out of six patients with increased LPA flow and LLP abnormality.[15]

Several mechanisms that could explain the gradual improvement of LLP with time have been suggested. For instance, in patients with Gianturco coils the improvement of LLP is attributed to the eventual endothelialization and the protruding device becoming buried in the vessel wall.[15–17] In a study by Verma et al., an angiographic examination performed after occlusion of aortopulmonary collaterals with a coil showed that the coil spirals had prolapsed into the aorta, while the follow-up angiographies showed them to have disappeared, presumably due to endothelialization.[17] Additionally, the increase of pulmonary artery diameter as a result of physical growth has also been reported to reduce obstruction.[15] We believe that the eventual improvement of LLP in our patients can be attributed to the same mechanisms as described previously.

Left lung perfusion abnormalities that develop as a result of PDA closure using ADO devices have been linked to young age and low weight.[6,16] A ductus ampulla size smaller than 5.8 mm was reported to have 90% sensitivity and 81% specificity for predicting LLP abnormalities in a series of 47 cases that used ADO for PDA closure.[13] In our study, patients whose LLP returned to normal and those whose LLP abnormality persisted showed no difference in terms of age, sex, weight, BSA, D1, D1/BSA, or D2/L at the time of PDA closure or the second scintigraphy. Although no statistically significant difference was found between the improved and unimproved patients, the L was shorter in the latter. The D1/L ratio was higher in the unimproved patients ($P = .018$). This shows that perfusion abnormalities persist in PDA patients whose L is short and D1 is wider than its length. In terms of predicting cases with persistent LLP abnormality, when the multivariate regression analysis results and ductus parameters like the D2 and the D1/L ratio are taken into account, we see a high risk of persistent LLP abnormality for short, wide, and tubular ductus types. In the light of these data, the patients in our study whose D1/L ratio is over 0.5 are at risk of developing persisting LLP abnormalities with 80% sensitivity and 92.3% specificity. It can be thought that device protrusion will happen more frequently in patients with a short ductus and cause perfusion abnormalities. In our study, there was one patient with a short window-like (Krichenko type B) ductus. For this reason, to prevent LPA obstruction in all types of ductus except the window type, it may be wise to use devices with shorter body length in patients whose L is shorter than the D1 and whose D2 is small. The use of the shorter ADO 2 as opposed to ADO 1 in suitable cases may decrease the risk of perfusion abnormalities.

No statistically significant difference was found between the device types used in our study with regard to the rate of LLP improvement. Abnormal perfusion normalized in 7 out of 12 (58%) cases using ADO and in 6 out of 11 (54%) cases using a coil. Kramoh et al. reports LLP abnormalities in three out of nine (33%) patients using ADO, in all of whom LLP improved to an extent but never completely normalized. It has been suggested that ADO use increases the risk of LLP abnormality and accompanies persistent LLP abnormality.[6] Another study reports that LPA obstruction was echocardiographically detected after ADO use in 7 out of 46 infants under 12 months of age and that LLP improved in 4 of them within 3 months after the procedure.[18]

The information in medical literature regarding the link between LLP abnormality and LPA blood flow velocity is contradictory. While Dessy et al.[7] reports a weak correlation between increased LPA blood flow velocity and LLP abnormality, and Soares et al.[3]—a strong one, according to Sreeram et al.[4] there is no correlation between the two parameters. Pass et al. found that the LPA gradient was above 20 mm Hg in two patients out of 484, reported LLP abnormality in these patients, and suggested that using large and oversized devices in younger children was a risk factor.[11] In our study, despite the LPA blood flow velocity being higher in patients with persisting LPA abnormality, no significant difference was found between the two groups.

When measuring flow velocity at the stenosis using pulsed wave Doppler, the results may vary depending on the position of the sample volume. While the sample volume is not intended for device-related stenosis, it has been reported that placing it directly before the stenosis can yield more accurate measurements.[19] In their study, Soares et al. preferred to make the measurements at the distal end device if there was any protrusion,

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found the LPA flow velocity to be higher compared to MPA and RPA, and used DVI to determine which patients had LLP abnormalities. They suggested that DVI would give the most reliable results as LPA blood flow is affected by many factors such as heart rate, volume status, and ventricular function. The same study also found that average LPA blood flow velocity and DVI was higher in patients with LLP abnormalities as opposed to normal ones, and reported that DVI with a 50% cutoff value had 100% sensitivity and 98% specificity for showing LLP.[3]

If there was device protrusion in our study, measurements were made at the distal end and LLP was found to be under 40% in 8 out of 10 patients with DVI over 50% and only in 3 out of 13 patients with DVI under 50%. With 50% set as the cutoff value for DVI, it has 80% sensitivity and 76.92% specificity for showing LLP abnormality. This leads us to the thought that DVI may be used for predicting LPA obstruction in patients after transcatheter PDA closure.

In children, vascular growth depends on blood flow.[20] Changes in LPA blood flow are considered a possible influence on the growth of the pulmonary artery. Carey et al. reported that the LPA index stayed low as compared to RPA in 4 out of 25 patients who underwent PDA closure and that LPA blood flow velocity was higher in three out of these four patients.[15] A study that used coils for PDA closure reported that LPA diameter remained smaller than the RPA diameter and that the LPA index was lower in patients with DVI over 50%.[3] There are no studies that have reported on LPA diameter follow-up in patients using ADO. In our study, no difference with regard to RPA and LPA indexes was found between improved and unimproved patients, among all patients, and between all the device types used.

A major limitation of this study is our failure to evaluate patients for LPA stenosis before performing transcatheter PDA closure.

Conclusion

Left lung perfusion abnormalities are frequently seen after PDA closure performed using various device types. In fact, even when no significant gradient is detected with a catheter after transcatheter PDA closure, LLP may still be impaired. However, long-term follow-up shows that LLP eventually improves to normal in the majority of patients. The patients in whom it does not completely normalize still generally show some extent of improvement. On the other hand, LLP abnormalities persist in patients with L shorter than D1. The development and use of shorter devices may help to avoid LPA obstruction in patients with this type of ductus. Additionally, the DVI values may be used during post-PDA closure follow-up of patients who are thought to have LPA obstruction, although in order to reach a more certain opinion on this matter, this study must be conducted on a wider case series.

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Author Contributions

Fadli Demir: Data collector, organizer, and writer of this report.

Ahmet Celebi: Critical revision of article; chief of our department and one of the interventional cardiologists who performed all procedures.

Turkay Saritas: Organizer and statistician of this report.

Abdullah Erdem: Interpreter and designer of this report.

Halil Demir: Echocardiographer and junior cardiologist who attended some procedures.

Mehmet Fatih Firat: Specialist on nuclear medicine; interpreter of this report.

Tugcin Bora Polat: Interpreter and data collector of this report; one of the interventional cardiologists who performed some of the procedures.

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