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ORIGINAL ARTICLE

Effect on Pulmonary Atery Presssres after Transcatheter Occlusion of Patent Ductus Arteriosus with Severe Pulmonary Artery Hypertension

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ABSTRACT

Objective: The purpose of this study was to ascertain how the patent ductus arteriosus will respond to trans catheter closure on the pulmonary artery pressure.

Methods: All patients having clinical and echocardiographic evidence of hemodynamically significant isolated PDA and patients with PASP > 20 mmHg will be labelled as PDA with PAH, while patients with mean PASP > 60 mmHg will be labelled as PDA with severe PAH. These patients were all included in the research. Both pulmonary artery pressure measurements and echocardiography were done. Once the patient has been assessed, the viability of a transcatheter closure PDA has been confirmed.

Results: The Mean age of the patients were 10.6±8.18 years. Out 40 participants 35% were males and 65% were females. A significant variation (p=0.003) in MPA pressure was observed in patients after balloon occlusion. A significant variation in RV higher (0.007) and lower (0.003) pressure was observed in patients after balloon occlusion. A significant variation (p=0.005) in LV Higher pressure was observed in patients after balloon occlusion. A significant variation (p=0.003) in higher Aorta pressure was observed in patients after balloon occlusion.

Conclusion: After PDA closure, some individuals with borderline hemodynamic data with PDA and PAH may get worse or continue to have PAH. Care must be taken while providing these individuals with permanent closure.

INTRODUCTION

Patient outcomes after defect closure in those with congenital heart defects (CHDs) associated with pulmonary arterial hypertension (PAH) rely on the reversibility of pulmonary vascular alterations. Closing the shunt will normalize the shunt-induced PAH in the early, flow-dependent stage (1). High pulmonary pressure and higher flow for an extended period of time may produce permanent vascular alterations in the lungs if the blood supply is diverted from the system to the lungs. When PAH progresses to a bidirectional shunt, deciding whether or not to shut the shunt might be challenging (2). The defect may act as a safety valve, emptying the right ventricle in the event that the pulmonary vascular alterations become permanent (3, 4). The prognosis and death rate of these individuals after defect closure are comparable to those of those with idiopathic PAH. Patients with PAH caused by coronary heart disease have a substantially better chance of surviving than those with idiopathic PAH (5). This highlights the need for precise determination of operability in individuals with CHDs and elevated pulmonary vascular resistance (PVR) (6). There is currently no reliable way to predict which patients may benefit from defect closure. Accordingly, doctors evaluate potential candidates for defect closure based on a variety of parameters and their personal experiences. Persistent pulmonary hypertension after surgery, however, is not uncommon (7). Histological alterations in PAH-related small pulmonary arteries have traditionally been assessed by pulmonary biopsy, which is considered the gold standard for this purpose. The predictive usefulness of lung biopsies in assessing operability is low, despite studies suggesting a significant association between biopsy findings and postoperative hemodynamic measures (8). Uneven distribution of lesions within the lungs might lead to an incorrect assessment of pulmonary vascular disease. Percutaneous transcatheter closure of the PDA has become the first-choice treatment at many institutions because to advances in technology and the enhanced performance of interventions. Percutaneous closure of the PDA provides several benefits over traditional surgery for patients with a big PDA and severe PAH (9). For instance, this approach eliminates the negative effects of thoracotomy and general anesthesia in patients aged 13 and above. More crucially, hemodynamic changes can be tracked in real-time. The device may be removed quickly if there is a decline in hemodynamic parameters due to closure, avoiding any unfavorable consequences. The post closure PAH response may be evaluated in individuals with severe PAH by a trial occlusion technique, in which the defect is temporarily closed (10). In Pakistan, 8.6% of babies born with cardiac defects have patent ductus arteriosus. The range of clinical presentations is wide, from asymptomatic heart murmur to congestive heart failure or eisenmenger's syndrome. The size of the PDA, the size of the shunt, and the pulmonary vascular resistances all play important roles in determining the PDA's natural history (11). Those who have a substantial shunt from the left to the right of their brain may not experience any symptoms for quite some time. Atrial arrhythmias, congestive heart failure, endarteritis, irreversible hypertensive pulmonary vascular disease, and very infrequently ductus aneurysm or abrupt aortic dissection have all been linked to chronic volume overload in retrospective studies (12). In patients with severe pulmonary artery hypertension, the aim of this study was to determine the impact of transcatheter closure of the patent ductus arteriosus on pulmonary artery pressure.

METHODOLOGY

After the ethical approval from institutional review board, this crossectional study was conducted at Department of Pediatric Cardiology, NICVD, from 01/July/2022 to 30/Nov/2022. Through non-probability purposive sampling all the patients with clinical and echocardiographic evidence of hemodynamically significant isolated PDA and the patients with PASP > 20 mmHg will be labelled as PDA with PAH, while patients with mean PASP >60 mmHg will be labelled as PDA with severe PAH were included in the study. Patients with silent PDA, complex heart disease, with unsuitable PDA size with interventional closure, and patients having saturation <92% on pulse oximetry in lower limb were excluded from the present study. Patients who met the inclusion criteria and went to the pediatric outpatient department at the National Institute of Cardiovascular diseases in Karachi was recruited in the research. Each patient was have their information entered into a data collecting tool once verbal agreement from their parents has been obtained for participation and publishing in the research. Both echocardiography and measurements of pulmonary artery pressure was carried out. The viability of a transcatheter closure PDA was determined when the patient has been evaluated. The patient was admitted in advance of transcatheter closure of PDA. Patients having the surgery will be sedated with midazolam, and after the femoral vein and femoral artery have been punctured, a dosage of heparin (75-100 unit/kg) was

administered. Before closing the proximal aortic arch (PDA), an aortogram in left lateral projection was taken. PAH was evaluated both before and after a PDA was closed transcatheterally. Once the PDA was occluded with the occluding balloon, the aortic, pulmonary, and left ventricular pressures was monitored immediately, after 5 minutes, and after 10 minutes. An antegrade approach from the pulmonary artery was used to conduct transcatheter closure of PDA if this was a viable option. After the operation, an extra aortogram was done to ensure the aortic valve was completely closed. PDA was not blocked if either PA pressures do not fall or LV pressures do not decrease following the blockage. Data was expressed as mean ± standard deviation. Changes in echocardiographic parameters was analyzed with paired t-test. Mean p value ≤ 0.05 was considered statistically significant. All of the statistical analyses were done using SPSS version 26.

RESULTS

40 participants demographic and clinical data is represented in Table 1. The Mean age of the patients were 10.6±8.18 years. Out 40 participants 35% were males and 65% were females. About 33% of the patients presenting complained was failure to thrive, while 30% of the recruited patients were experiencing shortness of breath. The Mean heart rate of the patients were 95.3±13.4 B/min. The Mean respiratory rate of the patients was 25.32±7.2 B/min. The Mean systolic pressure of the patients was 102.85±17.7 mmHG. The Mean diastolic pressure of the patients was 52.2±10.6 mmHG.

Table 1: Clinical and demographic parameters of the study participants

Parameters	N=40
Age	
Mean	10.6
S. D	8.18
Gender	
Male	14 (35%)
Female	26 (65%)
Presenting Complaints	
Failure to thrive	13 (33%)
shortness of Breath	12 (30%)
Palpitation	8 (20%)
recurrent chest infection	7 (17%)
Heart Rate (B/MIN)	
Mean	95.3
S. D	13.4
Respiratory Rate (B/MIN)	
Mean	25.325
S. D	7.223138
Systolic BP	
Mean	102.85
D. D	17.7353
Diastolic BP	
Mean	52.2
D. D	10.66314
Pluses	
Good Volume	23
Low Volume	17

Echocardiography Parameters of the study patients were presented in Table 2. About 65% of the patients had moderate PR, while average of peak PR was 48.6±10.6mmHg. About 73% of the patients had moderate PR, while average of peak TR was 66.9±13.1mmHg. Mean RV TAPSE, LVIDD, LIVDS and LVEF was 17.85±4.7mm, 41.25±10.6mm, 16.225±4.67mm, and 61.6±2.6%.

Cardiac catheterization parameters were presented in the table 3. 53% of the patients had type A PDA, while 35% had type B PDA. Mean MPA Pressures (mmHG) of the patients after cardiac catheterization was 46.025±11.2. Mean RV Pressures (mmHG) of the patients after cardiac catheterization 68.8±17.8/7.35±1.21. Mean LV Pressures (mmHG) of the patients after cardiac catheterization was 105±18.23/7.95±1.6. Mean aortic pressures (mmHG) of the patients after cardiac catheterization was 106.8±17.8/51.2±9.3.

Table 2: Echocardiography Parameters of the study participants

Echocardiography Parameters	N=40
PR	
Mild	11 (28%)
Moderate	26 (65%)
Severe	3 (7%)
PEAK PR (MMHG)	48.6±10.6
TR	
Mild	3 (7%)
Moderate	29 (73%)
Severe	8 (20%)
PG TR (MMHG)	66.9±13.1
RV TAPSE (MM)	17.85±4.7
LVIDD (MM)	41.25±10.6
LVIDS (MM)	16.225±4.67
LVEF (%)	61.6±2.6

Table 3: Arterial pressure of the study patients after Cardiac catheterization

Cardiac catheterization parameters	N=40
Type of PDA	
A	21(53%)
B	14 (35%)
C	5(13%)
MPA Pressures (mmHG)	46.025±11.2
RV Pressures (mmHG)	
Upper	68.8±17.8
Lower	7.35±1.21
LV Pressures (mmHG)	
Upper	105±18.23
Lower	7.95±1.6
Aortic Pressures (mmHG)	
Upper	106.8±17.8
Lower	51.2±9.3
HR	71.6±10.8

Out 40 patients Balloon occlusion was done in 13 patients (Table 4). Mean ON MPA Pressures (mmHG) of the patients immediately after occlusion was 56.1±5.4. Mean ON RV Pressures (mmHG) of the patients immediately after occlusion 88.2±9.9/8.53±1.1. Mean ON LV Pressures (mmHG) of the patients immediately after occlusion was 123.9±13.4/ 8.84±1.1. Mean ON aortic pressures (mmHG) of the patients immediately after occlusion was 126.3±5.4/60.3±7.0. Mean ON MPA Pressures (mmHG) of the patients 5 minutes after occlusion was 51.23±6.7. Mean ON RV Pressures (mmHG) of the patients 5 minutes after occlusion 83.8±13.0/ 8.23±1.1. Mean ON LV Pressures (mmHG) of the patients 5 minutes after occlusion was 123.2±12.9/8.9±1.43. Mean ON aortic pressures (mmHG) of the patients 5 minutes after occlusion was 127.6±14.9/60.6±6.8. Mean ON MPA Pressures (mmHG) of the patients 10 minutes after occlusion was 46.3±8.2. Mean ON RV Pressures (mmHG) of the patients 10 minutes after occlusion 79.9±14.9/7.9±1.4. Mean ON LV Pressures (mmHG) of the patients 10 minutes after occlusion was 123.8±13.3/8.5±1.9. Mean ON aortic pressures (mmHG) of the patients 10 minutes after occlusion was 123.1±12.2/60.4±7.4.

Table 4: Arterial Pressure of the study patients after Balloon Occlusion

Balloon Occlusion Parameters		N=13
Not Done		27
Done		13
Effect immediately after occlusion	ON MPA Pressures (mmHg)	56.1±5.4
	ON RV Pressures (mmHG	
	Upper	88.2±9.9
	Lower	8.53±1.1
	ON LV Pressures (mmHG	
	Upper	123.9±13.4
	Lower	8.84±1.1
	ON Aortic Pressures (mmHG)	
	Upper	126.3±5.4
	Lower	60.3±7.0
	HR	80.5±9.9
Effect after 5 minutes of occluding	ON MPA Pressures (mmHg)	51.23±6.7
	ON RV Pressures (mmHG	
	Upper	83.8±13.0
	Lower	8.23±1.1
	ON LV Pressures (mmHG	
	Upper	123.2±12.9

	Lower	8.9±1.43
	ON Aortic Pressures (mmHG)	
	Upper	127.6±14.9
	Lower	60.6±6.8
	HR	79.4±9.3
Effect after 10 minutes of occluding	ON MPA Pressures (mmHg)	46.3±8.2
	ON RV Pressures (mmHG	
	Upper	79.9±14.9
	Lower	7.9±1.4
	ON LV Pressures (mmHG	
	Upper	123.8±13.3
	Lower	8.5±1.9
	ON Aortic Pressures (mmHG)	
	Upper	123.1±12.2
	Lower	60.4±7.4
	HR	78.6±9.6
Occlusion Results		
Positive		10
Negative		3

Table 4 shows the comparison in arterial pressure of the patients in cardiac catheterization and balloon occlusion. A significant variation (p=0.003) in MPA pressure was observed in patients after balloon occlusion. A significant variation in RV higher (0.007) and lower (0.003) pressure was observed in patients after balloon occlusion. A significant variation (p=0.005) in LV Higher pressure was observed in patients after balloon occlusion. A significant variation (p=0.003) in higher Aorta pressure was observed in patients after balloon occlusion.

Table 5: Comparison of the arterial pressure of the patients after Balloon Occlusion and Cardiac catheterization

	Cardiac Catheterization	Balloon Occlusion	P Value
MPA Pressure	46.025±11.2	56.1±5.4	0.003
RV Pressure			
Higher	68.8±17.8	88.2±9.9	0.007
Lower	7.35±1.21	853±1.1	0.003
LV Pressure			
Higher	105±18.23	123.9±13.4	0.005
Lower	7.95±1.6	8.84±1.1	0.127
Aorta Pressure			
Higher	106.8±17.8	126.3±5.4	0.003
Lower	51.2±9.3	60.3±7.0	0.15
Heart Rate	71.6±10.8	80.5±9.9	0.8

DISCUSSION

The current standard of care for PDA therapy is TCC. With more practice, PDA patients with PAH can now be assessed for transcatheter device occlusion. The results of this trial showed that TCC of PDA in patients with PAH is possible, efficient, and safe—even in those who have significant symptoms (13, 14). Although rare, right heart failure, progressive pulmonary vascular disease, and non-regression of pulmonary hypertension might cause a small percentage of patients with borderline hemodynamic data to worsen following PDA repair. Their natural course is comparable to that of idiopathic PAH. In the present study, a significant difference in the arterial pressure was observed in the patients after balloon occlusion. A difficult clinical issue is whether to attempt TCC of PDA in patients with severe PAH. If the severe PAH is reversible, it will be the main factor in care and prognosis in this disease (15). A clinical examination is done to assess if severe PAH is reversible. However, if the clinical tests are ambiguous and there are certain restrictions for computing PVR in PDA, it might be challenging to decide whether to act (16). The use of vasodilators, including inhaled nitric oxide, has little usefulness in determining operability, according to multicenter research by Balzar et al (15). Contrary to widespread perception, a recent study revealed that postoperative outcome did not correspond with preoperative hemodynamic data for a variety of reasons. To determine the contribution of the left to right shunt and PVR to PAH, trial blockage of the PDA with a device has been used (17, 18). The standards we used were as follows: 1) a decrease in pulmonary artery pressure or no increase; 2) no drop in aortic pressure or SaO2; and 3) no worsening of symptoms. We regarded the PAH as reversible if all the requirements were met. Otherwise, occlusion was abandoned since it was thought to be irreversible PAH. This is a valid test to prevent device closure in patients with questionable hemodynamic data (19). Our study has two primary limitations. First, the study's small sample size, which reduced its power, was a key drawback. Second, only ultrasonography was used to measure pulmonary arterial pressure during the follow-up. Nobody underwent catheterization once again.

CONCLUSION

TCC is presently the recommended treatment for PDA with severe PAH. Although PAP would fall by >10% very away following trial occlusion, certain individuals with equivocal hemodynamic data with PDA and PAH might continue to have PAH or worsen after PDA closure. Treatment for permanent closure in these individuals must be administered with prudence.

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