Original Article

# IMMEDIATE RESULTS OF DEVICE CLOSURE IN HYPERTENSIVE PATENT DUCTUS ARTERIOSUS

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## Author's Contribution

AB:Conducted the study and wrote the article. MY:Helped in review the article. TA:Re-arranged data and corrected article.

**All authors declare no conflict of interest.**

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ABSTRACT:
OBJECTIVE: To assess the mean pulmonary artery pressures (PAP) in patients with large Patent Ductus Arteriosus (PDA) with 'reversible pulmonary hypertension' before and after the device closure.
MATERIAL AND METHODS: This descriptive retrospective study was carried out at CPE Institute of Cardiology, Multan, Pakistan from 2007 to June 2019. Patients who had large PDA with pulmonary hypertension (PH) were included. Mean PAP > 50% of mean aortic pressure was considered as pulmonary hypertension. Reversibility of PH was based on clinical criterial. Patients with room air saturation > 93%, cardiomegaly on chest x-ray with lung arterial vascularity extending into the lateral one third of the lung fields were considered to have reversible pulmonary hypertension. Patients with weight <8 kg and age < 2 year were excluded.
Out of 556 patients who had undergone PDA device closure, 98 had fulfilled our inclusion criteria. Mean age was 8 ±7.5 (2.5 – 45) years. Mean weight was 19.5 ±13.2 (7-66) kg. Mean diameter of PDA was 6 mm. Mean PAP decreased from 59 ± 12 mmHg to 30 ± 7 ($p < .001$) after device closure. Duct occluders were used in 92 patients (97.9%), while 2 had muscular VSD device (2.1%). In 2 patients, there was underestimation of the size of PDA so the device was retrieved and replaced with large one. Procedure remained unsuccessful in 2 patients because the device dropped into main pulmonary artery before it was released. Larger size device was not available at that time so the patients were referred for surgery. The duct occluder devices embolized partially in 2 patients which were referred for the surgical removal of the devices and PDA ligation.
Post procedure echocardiogram performed next day showed no residual PDA in all out patients. There was pulse loss in lower limb in 8 patients which was treated successfully with heparin infusion with no residual damage. Mild obstruction is documented in left pulmonary artery (n=6). None of our patients had high PAP after the device closure.
CONCLUSION: Device closure is safe and effective in closure of hypertensive PDA using clinical criteria. Follow up study is required to reinforce these findings.
KEY WORDS: Congenital heart disease, PDA, device closure, pulmonary hypertension, hypertensive PDA

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## INTRODUCTION:

In developing countries, patient ductus arteriosus (PDA) still remains a cause of irreversible pulmonary hypertension. The late diagnosis and appropriate treatment is due to poverty, poor education and few cardiac centers.[1] If left untreated, it can lead towards irreversible pulmonary hypertension by 2 years of age. This study will highlight the role of interventional pediatric cardiologist to reduce progression towards 'irreversible pulmonary hypertension.[2]

Due to large PDA, pulmonary circulation is exposed to high pressures. This lead to progressive morphological changes in pulmonary vascular bed resulting in rise of pulmonary vascular resistance (PVR). When PVR is increased to extent of systemic vascular resistance (SVR), initial left to right shunt progress to shunt reversal i.e. irreversible pulmonary hypertension or Eisenmenger complex is developed.[3]

It is crucial to decide whether the pulmonary hypertension is reversible or not. Pulmonary vasodilators including inhaled nitric oxide may be used to document reversibility. Balloon occlusion and trial occlusion by device are other methods to decide for reversibility.

## MATERIAL AND METHODS:

This descriptive retrospective study was carried out at CPE Institute of Cardiology, Multan, Pakistan from 2007 to June 2019. Ethical committee of hospital approved the study. During this period, 556 patients underwent device closure of patent ductus arteriosus (PDA). After informed consent, patients were evaluated initially by history, clinical examination, chest x-ray, ECG, oxygen saturation in room air and transthoracic echocardiogram (TTE). Patients who had evidence of irreversible pulmonary hypertension as evident by oxygen saturation less than 93%, normal cardiothoracic ratio and prunning on x-ray were excluded. Patients with age $<$ 2 year and weight $<$ 8 kg were also excluded. Ideally they should have their PVR assessed but due to resource limitation, this was decided on clinical grounds in our study.Patients who had undergone device closure had reversible hypertensive PDA. Reversible severe pulmonary hypertension decided on clinical ground (room air saturation$>$93%, cardiomegaly on x-ray chest with lung arterial vascularity extending into the lateral one third of the lung fields). Echocardiography was performed before and after the procedure. Pulmonary artery pressures were measured in the catheterization laboratory before and after the device closure.

Detailed echocardiography was crucial to document size of the PDA, suitability for device closure, pressure gradients, and estimation of the pulmonary artery pressure (PAP) and diastolic equalization of the pressures across the duct according to the guidelines as given by American Society of Echocardiography.[4] Colour flow mapping was used to assess the direction of the shunt. Tricuspid regurgitation (TR) jet is used to assess systolic pulmonary artery pressures. Other factors (additional shunt lesion, left ventricular dysfunction, mitral valve abnormality, pulmonary venous stenosis) were ruled out. Pulmonary hypertension was defined as mean pulmonary artery pressure be$>$ 50% of mean aortic pressures. 98 patients with age $>$2years and weight $>$ 8 kg with evidence of large PDA with reversible pulmonary hypertension were included in the study.

Informed consent taken from the family. After sedation, arterial and venous access were taken under local anesthesia. Aortograms were done in lateral and $30^0$ RAO projection to delineate the size, shape and suitability for device closure. Various hemodynamic parameters were recorded in room air and post 20 min of oxygen inhalation using venture mask (60%). Balloon occlusion was done where-ever appropriate size balloon was available using Osypka balloon VACS II /III balloon (Osypka AG, Rheinfelden, Germany) via 2nd femoral venous access. (Figure 1) Pulmonary hypertension was defined as reversible ($>$ 20 % fall in mean PA pressure). In the patients with reversible pulmonary hypertension, device closure was done using anterograde approach. The devices used were ADO I (AGA Medical Corporation, Plymouth, MN), Shasma duct occluder, AGA (AGA Medical Corporation, Plymouth, MN ) muscular VSD device. (Figure 2,3) Others who had irreversible pulmonary hypertension were managed medically subsequently.

Continuous variables measured were mean pulmonary artery pressures, residual PDA, aortic or left pulmonary artery obstruction. TTE was performed next day of the procedure to document any residual PDA, LV functions, pulmonary artery pressures, LPA/ aortic obstruction.

Complication noted down including embolization, bleeding, pulse loss. Patients were discharged on oral antibiotics and analgesics for 3 days. Patients who had evidence of residual pulmonary hypertension on day 1 post procedure were discharged on pulmonary vasodilators (oral sildenafil) for three months.

## RESULTS:

Out of 556 patients who had undergone PDA device closure, 98 had fulfilled our inclusion criteria. Mean age was 8 +7.5 (2.5 – 45) years. Mean weight was 19.5 +13.2 (7-66) kg. (Table 1) Successful device closure done in 94 patients (95.9%). Mean diameter of PDA was 6 mm. Mean PAP decreased from 59 + 12 mmHg to 30 +7 mmHg ($p < .001$). Duct occluder was used in 92 patients (97.8%), while 2 had muscular VSD device. (Table 2) In 2 patients, there was under estimation of the size of PDA so the device was retrieved and replaced with another larger one successfully. Two patients had the device fully dropped into main pulmonary artery before it was released. Larger size device was not available at that time so the patients were referred for surgery. The duct occluder device embolized partially in 2 patient on very next day which was referred for the surgical removal and PDA ligation. (Table 3)

Post procedure echocardiogram performed next day to procedure showed no residual PDA in all out patients. None of our patient had residual shunt on echo performed next morning of the procedure. There was pulse loss in 8 patients which was treated successfully with heparin infusion with no residual damage.

**Table 1: Data**

| | |
|---|---|
| **Mean Age (years)** | 8 ± 7.5 (2.5 – 45) |
| **Mean weight (Kg)** | 19.5 + 13.2 (7-66) |
| **PDA device closure performed** | 556 |
| **Hypertensive Device closure attempted = n** | 98 |
| **Successful device closure = n** | 94/98 (95.9%) |
| **Hypertensive PDA failed / abandoned attempt = n** | 4 |

**Table 2: Hemodynamics**

| | |
|---|---|
| **Mean PA pressure (pre-device)** | 59 ± 12 mmHg |
| **Mean Ao pressure (pre-device)** | 84 ± 8 mmHg |
| **Mean PA pressure (post-device)** | 30 ± 7 mmHg |
| **Mean Ao pressure (post-device)** | 97 ± 7 mmHg |
| **PVR before / after device occlusion** | not done |
| **Trial by balloon occlusion = n** | 2 |

**Table 3: Results and Complications**

| | |
|---|---|
| **Residual PDA** | 0 |
| **Pulse loss** | 1 |
| **Referral for surgery after abandoned procedure** | 2 |
| **Embolized device with successful surgical retrieval** | 2 |
| **Duct Occluder** | 92 |
| **Muscular VSD device** | 2 |
| **LPA obstruction (mild)** | 6 |
| **Aortic obstruction** | nil |
| **Pre-device treatment with vasodilator** | not known |
| **Post-device treatment with vasodilator** | 2 |

Mild obstruction is documented in left pulmonary artery (n=6) and device protrusion into aortic (n=8) with no obstruction. 2 patients had their PAP still high after device closure though they were reduced significantly. These were put on oral pulmonary vasodilators (sildenafil) for 3 months

## DISCUSSION:

In developing countries, diagnosis of large PDA with pulmonary hypertension is often delayed. This is due to multiple reasons like poverty, ignorance, late recognition by the primary physician, paucity of pediatric cardiac services. Older children and adults develop calcification and aneurysmal dilation and calcification which are risk factors for surgical closure. Variety of devices are available now in Pakistan, though we have limited option in our catheterization lab.

The prognosis of these patients depend upon whether the pulmonary hypertension is reversible. Temporary occlusion of the defect i.e. balloon occlusion test is one way to assess the size of PDA and reversibility of the pulmonary hypertension. We used this test in 2 of our patients wherever appropriate size balloon was available. This is more accurate assessment than oxygen inhalation but it increased the cost of the procedure in resource limited setup. If ever, the balloon is not stable for 10 minutes, it could lead to false results. Venture mask provide oxygen inhalation up to 60%. However the standard protocol to test reversibility was inhalation of 100% oxygen which can be done only via endotracheal intubation. Paucity of anesthesia services in catheterization laboratory was another issue in our setup. 98 patients had reversible pulmonary hypertension;. We proceeded directly for the device closure to cut down the cost of the procedure. This was less cumbersome than balloon occlusion. But if ever, pulmonary artery pressures did not regresses after device closure, it had to be removed and wasted. We did not face this condition in any of our patient.

Regression of pulmonary artery pressure PAP after the device closure is main factor which determines long term prognosis. Sadiq et al reported success rate of 96 % with significant regression of mean PAP pressures ($p<.001$). However severe pulmonary hypertension was persistent in four (9.7%) patients at follow-up of 80(41–151) months.[5] Another study documented same phenomena in 4/43 patients at median follow up of 80 months (range 41-151) on long term follow up.[6] However study evaluated immediate results and did not include follow up. This is main limitation of our

study. Our study has 2 patients had high PAP after the device closure.

One strategy is pre-procedure treatment with pulmonary vasodilator. Successful PDA device closure reported in cases with PVR as high as 5-13 wood U.m$^2$.[7] Similar benefit of pre-medication has been documented by others as well.[8] Temporary occlusion of PDA with device proved to be safe and less time consuming for evaluation of vaso-reactivity/ reversibility in severe pulmonary hypertension. But the risk of device embolization in aorta is higher in cases with pulmonary hypertension.[9] Our decision for reversibility was based on clinical criteria which saved time and resources. Moreover, long term follow up is not indicated unless there is apparent immediate or early complication.[10] Success of the PDA device closure in hypertensive PDA has been documented in adults as well. [11]

## CONCLUSION:

Device closure is safe and effective in closure of hypertensive PDA using clinical criteria.Follow up study is required to reinforce these findings.

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