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Exploring the Role of Trial Occlusion Test in Patent Ductus Arteriosus (PDA) Closure: A Serial Case

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Abstract:- Background: Patent ductus arteriosus occurs in around 1 in 2000 full-term live newborns, making up 10-15% of cases of congenital heart disease. Percutaneous device closure is largely regarded as the preferred therapy for people diagnosed with PDA. Nevertheless, the closure of the PDA with a device is still a difficult task in patients with severe PAH. The trial occlusion strategy is used to evaluate the response of patients with severe pulmonary hypertension after closing the defect by briefly blocking it. This test has the potential to serve as a reliable predictor of the future progression of pulmonary hypertension. Case Presentation: We provide a report on three instances that underwent percutaneous closure of patent ductus arteriosus (PDA) in the presence of pulmonary hypertension. A 24-year-old adult patient arrived at our hospital with symptoms of intermittent dyspnea, easy fatigability, and occasional non-productive cough. The examination detected a persistent murmur located at the upper left sternal boundary. The patient received a diagnosis of patent ductus aorta and pulmonary hypertension. Discussion: The condition was successfully treated with transcatheter closure, which included a trial occlusion test. In the second instance, a toddler aged 2 was diagnosed with patent ductus aorta and pulmonary hypertension. The procedure of percutaneous PDA closure was scheduled. Following the trial occlusion test using the device occluder, we observed an elevation in pulmonary pressure which persisted and led to a pulmonary crisis. Conclusion: The trial occlusion test aids in identifying individuals who are prone to experiencing long-term regression of pulmonary hypertension and those who may deteriorate due to progressive pulmonary vascular disease (PVD) and right heart failure, therefore determining their suitability for PDA closure.

Keywords:- *Patent Ductus Arteriosus, Trial Occlusion Test, Pulmonary Hypertension.*

I. INTRODUCTION

Patent ductus arteriosus (PDA) constitutes about 8% of cases of congenital cardiac disease. It may be asymptomatic, meaning that there are no noticeable symptoms, and hence it is often not detected until maturity. Diagnosis may occur accidentally when a murmur is heard during a routine examination, or sometimes when symptoms that are connected to the condition are identified. An extended improper connection between the aorta and pulmonary artery may lead to gradually developing high blood pressure and impaired function of the left ventricle. Since the first percutaneous closure of Patent Ductus Arteriosus (PDA) carried out by Porstmann in 1968, other occluder devices and coils have been put into clinical practice. Transcatheter occlusion of the patent ductus arteriosus (PDA) using different devices and coils is a widely accepted alternative to surgical closure. Although it has shown promising results in treating PDA patients overall, its use in cases of PDA with severe pulmonary hypertension (PH) has been restricted. Only a limited number of publications have been published on the use of transcatheter closure for patent ductus arteriosus (PDA) with severe pulmonary hypertension (PH) in adults (Ji et al., 2008). Patent ductus arteriosus (PDA), which is linked to significant pulmonary arterial hypertension in both adults and children, is a formidable challenge for cardiologists. Due to advancements in methods and occluders, the use of transcatheter closure for Patent Ductus Arteriosus (PDA) is becoming more common in children and adolescents. Temporary blockage of the duct during catheterization is a strategy that may be used to assess the extent to which pulmonary arterial hypertension can be reversed before surgery. One reliable method for preoperatively assessing the degree of reversibility is temporary occlusion of the duct. This method allows for the evaluation of the consequences of duct closure and indirectly determines the reversibility of severe pulmonary arterial hypertension, particularly in borderline cases. According to Yan et al. (2007), using an occluder to temporarily block something for testing purposes is a straightforward and easy procedure. The trial occlusion test in device closure is an essential step in evaluating the likelihood of persistent pulmonary arterial hypertension (PP-PAH) after transcatheter closure of the patent ductus arteriosus (PDA) in patients with borderline pulmonary hypertension. The test requires temporarily blocking the defect to assess the reaction of polycyclic aromatic hydrocarbons (PAH) after closure. The length of trial occlusion varies based on the dimensions of the PDA and the specific occlusion device used. Typically, tiny PDAs may be charged in 5 to 6 minutes, however larger PDAs may take 10 to 15 minutes to charge. Trials assessing the occlusion of devices in closure procedures have shown a high success rate, with most studies reporting full closure rates of around 90% to 95%. Nevertheless, the test has several restrictions, including the need for ongoing monitoring of hemodynamic parameters and the possibility of consequences such as device embolization and bradycardia (Zhang et al., 2014).

Transcatheter closure of patent ductus arteriosus (PDA) has evolved significantly over the years, with various techniques and devices being utilized to achieve successful

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closure (Baruteau et al., 2014). The role of trial occlusion tests in PDA closure has been highlighted as a valuable tool in assessing the feasibility and safety of permanent closure, especially in challenging cases such as those with severe pulmonary hypertension (Ji et al., 2008; Niu et al., 2013). These tests involve temporary occlusion of the ductus to evaluate the consequences of closure and determine the reactivity of severe pulmonary arterial hypertension, aiding in decision-making regarding the closure procedure (Vijayalakshmi et al., 2014). Studies have shown that transcatheter closure of PDA is an effective and preferred method due to high occlusion rates and low complication rates associated with newer devices like embolization coils and the Amplatzer Duct Occluder (Gu et al., 2016; Güvenç, 2016). Additionally, the use of trial balloon occlusion for permanent closure in adults with severe pulmonary hypertension has been investigated, demonstrating the applicability of such tests in challenging cases (Ji et al., 2008). Furthermore, the long-term follow-up results of device occlusion of PDA have been assessed, emphasizing the importance of monitoring patients post-closure to evaluate outcomes and ensure the success of the procedure (Amoozgar et al., 2016). In conclusion, the exploration of the role of trial occlusion tests in PDA closure is crucial in determining the feasibility and safety of permanent closure, particularly in complex cases. These tests, along with advancements in transcatheter techniques and devices, contribute to the overall success and effectiveness of PDA closure procedures.

II. LITERATURE REVIEW

The role of the trial occlusion test in the closure of Patent Ductus Arteriosus (PDA) has been a subject of interest in recent medical literature. Transcatheter techniques, such as coil occlusion and device closure, have become widely used for PDA closure, offering less invasive alternatives to surgical ligation (Schneider & Moore, 2006; Sathanandam et al., 2021). Platelets play a crucial role in PDA closure, with studies highlighting their involvement in the closure process (Sallmon et al., 2021). Additionally, studies have explored the use of trial balloon occlusion for permanent PDA closure, especially in cases with severe pulmonary hypertension (Ji et al., 2008). Research has also focused on factors influencing the success of PDA closure, such as platelet counts and hemoglobin levels (Sallmon et al., 2018; Kishi et al., 2022). Furthermore, studies have investigated the outcomes and complications associated with different closure methods, including device occlusion and coil embolization (Amoozgar et al., 2016; Hildebrandt et al., 2010). The use of novel occlusive devices has expanded the options for PDA closure, particularly benefiting underweight infants (Wang et al., 2023; Hoyer, 2005). In the context of preterm infants, managing PDA poses unique challenges, with guidelines emphasizing the importance of timely closure due to associated complications (Terek et al., 2018; Köksal et al., 2019). Studies have explored various approaches to PDA closure, including transcatheter techniques and the use of different occluders (Ng et al., 2011; Atik-Ugan & Saltik, 2018). Moreover, the impact of PDA closure on other cardiac conditions, such as mitral regurgitation, has been investigated, highlighting the interconnected nature of cardiac pathologies (Kheiwa et al., 2016). In conclusion, the literature review on the role of trial occlusion tests in PDA closure underscores the significance of transcatheter techniques, platelet involvement, and factors influencing closure outcomes. Understanding these aspects is crucial for optimizing PDA management and improving patient outcomes.

III. CASE PRESENTATION

During the last 3 months from January 2024 since March 2024, There were 3 patients undergoing trial occlusion test before percutaneous PDA closure procedure.

A. Case 1

The first patient was a 24-year-old guy who was taken to the hospital because of sporadic difficulty in breathing and a tendency to get easily fatigued since his time in high school. Additionally, he had a past medical record of frequent upper respiratory tract infections. The existence of congenital cardiac disease in the parents and relatives has been negated. During the patient's assessment, a constant heart murmur was detected on physical examination. The chest X-ray revealed an enlarged heart along with indications of pulmonary hypertension. A transthoracic echocardiogram (TTE) was conducted. An 8.7 mm diameter patent ductus was detected using two-dimensional echocardiography. The morphology of CW Doppler with continuous flow was also assessed. A systolic pressure gradient of 36 mmHg and a diastolic pressure gradient of 6 mmHg were also observed throughout the ductus. The patient was scheduled for diagnostic and interventional cardiac catheterization. Based on the evaluation, the patient was deemed potentially eligible for percutaneous closure. The pressures of the left and right heart were measured and the results were as follows: PARi 6.6 WU x m2, FR 3.1 PAR/SVR 0.13. The aortic pressure was 127/82 (101) mmHg, while the pulmonary artery pressure (PAP) was recorded at 82/56 (69) mmHg. Aortography was conducted using the RAO 200 and true lateral views. The diameter of the ductus ostium was measured at 10.5 mm, while the ampulla measured 24.2 mm, as determined by aortography. The possibility of implanting a Mediinterv PDA occluder measuring 20-22 mm was taken into consideration. The subsequent stage included the process of 'trial occlusion'. We selected an occlusion device with a diameter of 20–22 mm. Once the occlude was securely attached to the delivery cable, it was dragged into the loader and inserted into the guiding sheath. Subsequently, the remaining part of the occluder was inserted into the Patent Ductus Arteriosus (PDA). Hemodynamic and clinical data were documented throughout the device's continued attachment to the cable. Following a 10-minute period of temporarily blocking blood flow, we reevaluated the pressures on the left and right sides of the heart. The aortic pressure was recorded as 141/84 (106) mmHg, while the pulmonary artery pressure (PAP) was measured at 61/27 (41) mmHg. Based on the provided information and after confirming the correct location, the device was released by spinning the delivery cord in a counterclockwise direction. A follow-up descending aortogram was performed 10 minutes following the discharge to assess for any remaining shunting. Prior to the completion of the treatment, pullback pressure measures were taken,

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revealing an aortic pressure of 127/82 (99) mmHg and a pulmonary artery pressure (PAP) of 47/26 (34) mmHg.

Table 1. Pre and Post Occlussion Test Pressure Case 1

	Pre Device Closure	10 menute Occlusion Test	Post Device Closure
	Pressure (mmHg)		
AoD	127/82 (101)	141/84 (106)	127/82 (99)
PA	82/56 (69)	61/27 (41)	47/26 (34)

B. Case 2

In the second instance, the patient was a 2-year-old kid who presented with a recurring respiratory tract infection that had been ongoing for 1 year. Additionally, the child had intermittent episodes of shortness of breath. The individual was diagnosed with Congenital Heart Disease at the age of 4 months. During the physical examination, the thorax was auscultated and vesicular breath sounds were detected, with no presence of rales or wheezing. Furthermore, a persistent heart murmur was detected during the physical examination. A transthoracic echocardiogram (TTE) was conducted, revealing the presence of a patent ductus with a diameter of around 6.4-7 mm as shown on the two-dimensional echocardiography. The morphology of CW Doppler with continuous flow was also assessed. The ductus was also found to have a systolic pressure gradient of 42 mmHg and a diastolic pressure gradient of 10 mmHg. The patient was scheduled for diagnostic and interventional cardiac catheterization. The results showed a PARi of 6.6 WU x m2, FR of 1.7 PAR/SVR of 0.37. Additionally, the aortic pressure was measured at 96/57 (76) mmHg and the pulmonary artery pressure (PAP) at 74/45 (61) mmHg. Aortography was conducted using the right anterior oblique (RAO) 200 and true lateral views. The diameter of the ductus ostium was measured to be between 7.5 mm and 10.3 mm. Subsequently, the possibility of implanting a Lifetech Heart PDA Occluder measuring 16-18 mm was taken into account. A "trial occlusion test" was conducted using the device for a duration of 10 minutes, without removing the device. Next, we conducted measurements of the aortic pressure, which yielded a reading of 64/35 (50) mmHg, and the pulmonary artery pressure (PAP), which registered at 88/54 (71) mmHg. The pulmonary artery pressure (PAP) exceeded the systemic pressure (oversystemic pressure). As a result of these changes in blood flow or an elevation in pulmonary artery pressure, the trial occlusion test was unsuccessful, leading us to decide against proceeding with the percutaneous closure of the patent ductus arteriosus (PDA). The devices were promptly removed and the treatments were halted.

Table 2. Pre and Post Occlussion Test Pressure Case 2

	Location	Pressure (mmHg)
Pre Device Closure	PA	74/45 (61)
	AO	96/57 (76)
Intra Occlusion Test	PA	64/35 (50)
	AO	88/54 (71)
Post Occlusion Test	PA	84/57(71)
	AO	81/55(68)
Post Canceled Closure	PA	89/61 (76)
	AO	94/58 (72)

C. Case 3

The third example included a 22-year-old girl who presented with a main complaint of shortness of breath that had been ongoing for 2 years prior to admission to the hospital. During the patient's physical examination, we also detected a constant heart murmur. To further investigate, we conducted a transthoracic echocardiography (TTE). A patent ductus with a diameter of 5 mm was detected using two-dimensional echocardiography. Furthermore, a systolic pressure gradient of 65 mm Hg and a diastolic pressure gradient of 9 mm Hg were observed across the ductus. The patient, who was deemed a potentially good candidate for percutaneous closure, was scheduled for interventional cardiac catheterization. Initially, the pressures of the left and right heart were assessed, yielding the following measurements: PARi 18.9 WU x m2, FR 1.6, and PAR/SVR 0.48. The aortic pressure was 119/61 (84) mmHg, whereas the pulmonary artery pressure (PAP) was also 119/61 (84) mmHg. Aortography was conducted using the RAO 200 and true lateral views. The diameter of the ductus ostium was measured to be between 7.5 and 8.2 mm, while the ampulla measured 19.1 mm, as determined by aortography. The possibility of implanting a Lifetech PDA Occlude measuring 14-16 mm was taken into consideration. The subsequent procedure included doing a "trial occlusion test". We selected an occlusion device with a diameter of 14-16 mm. Once the occlude was securely attached to the delivery cable, it was dragged into the loader and then inserted into the guiding sheath. The occluder was carefully positioned and secured against the aortic ampulla using fluoroscopic assistance. Subsequently, the remaining portion of the occluder was inserted into the Patent Ductus Arteriosus (PDA). Following a 10-minute period of trial occlusion, we conducted further measurements of the pressures in the left and right heart. The aortic pressure was found to be 124/53 (78) mmHg, while the pulmonary artery pressure (PAP) was recorded at 119/61 (84) mmHg. These findings suggest that the pulmonary artery pressure remains elevated and is not suited for percutaneous device placement. Following the shutdown, the devices were promptly removed and the processes were halted.

Table 3. Pre and Post Occlussion Test Pressure Case 3

	Location	Preassure
Pre Device Closure	PA	121/59 (82)
	AO	119/61(84)
Intra Occlusion Test	PA	124/53(78)
	AO	119/61 (84)
Post Occlusion Test	PA	124/53 (78)
	AO	108/70 (85)

IV. DISCUSSION

Patent ductus arteriosus (PDA) is a common congenital heart abnormality (CHD). If not corrected promptly, endothelial cell failure and vascular remodeling will progressively occur in the pulmonary arteries, resulting in elevated pulmonary vascular resistance (PVR) and severe pulmonary hypertension. (Xu et al., 2020). PDA may be linked to other congenital cardiac conditions, such as ASD or VSD, although in adulthood, it often exists as a solitary abnormality. The prevalence of Patent Ductus Arteriosus

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(PDA) is estimated to be about 1 in 2000 live births, making up 5-10% of all cases of congenital cardiac disorders. The annual death rate for people with untreated PDA is 1.8%. Furthermore, there are many other complexities to consider, including left ventricular overload, pulmonary hypertension, bacterial endocarditis, calcification, aneurysms, and, in rare cases, rupture. The citation is from Putra et al. (2016). Cardiologists are facing a problem in dealing with patent ductus arteriosus (PDA) that is accompanied by significant pulmonary hypertension in adults. The care and prognosis of these individuals depend on the reversibility of severe pulmonary arterial hypertension.

Given the advancements in medical technology and the improved effectiveness of treatments, percutaneous transcatheter closure of the patent ductus arteriosus (PDA) has become the preferred treatment option for many medical facilities when dealing with this patient. Percutaneous closure of the PDA offers some benefits compared to traditional surgery in patients with a large PDA and significant pulmonary hypertension. Specifically, this method eliminates the harmful effects of thoracotomy and general anesthesia in adolescent and adult patients. Crucially, the method allows for the monitoring of hemodynamic changes in real-time. If the hemodynamic parameters worsen due to device closure, the device may be promptly removed to minimize any possible negative consequences. (Zhang et al., 2014). The transcatheter closure of patent ductus arteriosus (PDA), which was first reported in 1967, has now become the established therapeutic approach for infants beyond the newborn stage. The method of coil occlusion for closure of patent ductus arteriosus (PDA), first developed in 1992, has now gained recognition as a well-established transcatheter therapy. Multiple occlusion systems have been created. At now, coils are the predominant choice for closing small-sized PDA. The amplatzer duct occluders (ADO) are often used for closing moderate to large-sized PDAs. Severe problems resulting with transcatheter PDA closures are infrequent (Amoozgar et al., 2016).

The use of methods and occluders has led to a rise in the frequency of transcatheter closure of PDA in children and adolescents. Temporary blockage of the duct during catheterization is a method used to assess the extent to which pulmonary arterial hypertension can be reversed before surgery. In children aged 5-12 years with severe pulmonary arterial hypertension, attempts have been made to close the PDA using transcatheter techniques, and these attempts have been successful (Yan et al., 2007). Nevertheless, there have been reports indicating that the transcatheter closure of the Patent Ductus Arteriosus (PDA) in adults might be both safe and successful (Bilkis et al., 2001). The prevalence of isolated patent ductus arteriosus (PDA) accounts for around 6% to 11% of all occurrences of congenital heart disease (CHD). Due to its lack of noticeable symptoms, isolated PDA is generally not detected by doctors until maturity, when pulmonary arterial hypertension (PAH) or congestive heart failure occurs. Consequently, a significant number of patients delay PDA closure until they have advanced pulmonary hypertension (Cassidy et al., 2009). The trial occlusion test is conducted to evaluate the hemodynamic reaction to the closure of a patent ductus arteriosus (PDA), especially in individuals with borderline pulmonary hypertension. The procedure entails the temporary obstruction of the PDA by means of a balloon or a self-expandable occluder, in order to mimic the effect of permanent closure. This test aids in the identification of individuals who are prone to developing pulmonary hypertension after PDA closure, enabling better informed decision-making about the closure surgery (Ji et al., 2008).

One reliable method for preoperatively determining the degree of reversibility is the temporary occlusion test of the duct. This test allows us to assess the consequences of duct closure and indirectly evaluate the reversibility of severe pulmonary arterial hypertension, particularly for borderline cases. While the calculation of pulmonary vascular resistance is significant for evaluating the reversibility of pulmonary arterial hypertension, it is less precise compared to temporarily blocking the duct. This is due to the inherent limitations of obtaining a pulmonary arterial sample in these individuals. In contrast to the temporary blockage of the duct using a balloon as described in prior studies (Yan et al., 2007). In relation to this patient, we believe that doing a trial occlusion using an occluder is a straightforward and beneficial procedure. Temporary blocking of the PDA using a balloon was similarly effective in predicting the changes in blood flow following closing the PDA using a catheter in individuals with mild pulmonary hypertension. Yan et al. proposed a more efficient and easy way to evaluate the response of pulmonary hypertension. Instead of using a balloon obstruction-based strategy, they substituted the balloon with a self-expandable occluder for trial occlusion (Yan et al., 2007; Ji et al., 2008).

Various aspects of the trial occlusion strategy need to be taken into account. Begin by selecting the dimensions of the gadget. In individuals with borderline pulmonary arterial hypertension (PAH), the size of the patent ductus arteriosus (PDA) is likely to be inaccurately assessed by an echocardiography or aortic angiography. This is because the flow of blood from the systemic circulation to the pulmonary circulation via the ductus reduces as the pulmonary artery pressure (PAP) increases. Furthermore, it is essential to continually evaluate the hemodynamic parameters, such as pulmonary artery pressure (PAP), arterial pressure (AP), and heart rate, during the occlusion experiment. After the closure of the PDA, some patients may have a significant drop in both arterial pressure (AP) and heart rate. Furthermore, the recording of the PAP should occur only after the PDA has been fully closed or closed with a little remaining flow. A significant correlation exists between severe pulmonary hypertension and a sizable patent ductus arteriosus (PDA) (Zhang et al., 2014).

During trial occlusion in PDA closure, the following hemodynamic data are measured:

- A 20% drop in the baseline systolic PA pressures or at least no increase in systolic pulmonary artery pressure (PAP);
- No drop in systemic pressure;
- No drop in oxygen saturation or clinical signs of pulmonary hypertensive crisis.

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The hemodynamic parameters are continually monitored throughout the trial occlusion to assess the reaction of post-closure pulmonary arterial hypertension (PAH) and forecast the probability of residual pulmonary hypertension following the transcatheter closure of the patent ductus arteriosus (PDA) (Amoozgar et al., 2016). During our second and third cases, we attempted percutaneous closure of a patent ductus arteriosus (PDA) in a patient with pulmonary hypertension. However, after conducting a trial occlusion test, we discovered that the pulmonary pressure increased and was persistently causing a pulmonary crisis. As a result, we contemplated discontinuing the percutaneous PDA closure procedure. Failed occlusion test leading to incomplete PDA closure is a potential issue that may arise in some instances. This is especially accurate for individuals who have a significant patent ductus arteriosus and pulmonary hypertension. During such instances, the occlusion test may detect the deterioration of symptoms or an elevation in pulmonary artery pressures, suggesting that the closure procedure may not be appropriate for the patient. This phenomenon is often seen in individuals who have borderline hemodynamic data, where the presence of patent ductus arteriosus (PDA) and pulmonary arterial hypertension (PAH) are strongly interconnected. In such instances, the closure of the pulmonary ductus arteriosus (PDA) might prevent the regression of pulmonary hypertension and the progression of pulmonary vascular disease (PVD), ultimately leading to right heart failure. The sources do not specifically describe the specific mechanism responsible for the rise in pulmonary artery (PA) pressure after a trial occlusion test in the closure of patent ductus arteriosus (PDA). However, it may be deduced that a rise in PA pressure after trial occlusion may suggest the presence of irreversible pulmonary arterial hypertension (PAH). The progression of these individuals' condition is comparable to that of primary or idiopathic pulmonary arterial hypertension (PAH). Hence, it is important to thoroughly assess patients before to undertaking PDA closure, taking into account variables such as the dimensions of the PDA, the extent of pulmonary arterial hypertension (PAH), and the patient's general health condition (Amoozgar et al., 2016). In a large-scale investigation, the success rate of PDA closure using the trial occlusion test was found to be 97.2%, indicating a high level of success. The trial occlusion test is crucial in assuring the effectiveness of PDA closure, particularly in patients with severe pulmonary hypertension (Amoozgar et al., 2016). Although the trial occlusion test is useful in assessing the likelihood of chronic pulmonary hypertension, it does have several drawbacks. The sample size of the research is often limited, and the findings could not accurately represent the extended development of pulmonary hypertension, which might manifest much later than 1 to 10 years after the surgery. (Zhang et al., 2014). In addition, it is important to note that the test has some dangers, as patients may develop tachycardia and arrhythmia as a result of reduced cardiac function or nervous stress (Ji et al., 2008).

V. CONCLUSION

In summary, the trial occlusion test is a crucial step in the transcatheter closure of PDA in patients with pulmonary hypertension. The test is designed to assess the hemodynamic response to PDA closure and predict the risk of pulmonary hypertension after the procedure. The test involves preparing the patient, measuring the morphology and diameter of the PDA, performing trial occlusion, monitoring hemodynamic parameters, the occluder is released and evaluating the patient after the procedure. The occluder is released when all the criteria are satisfied after trial occlusion.

REFERENCES

[1]. Amoozgar, H., Salehi, S., Farhadi, P., Edraki, M. R., Borzoee, M., Ajami, G., Cheriki, S., & Mohammadi, H. (2016). Follow-Up Results of Device Occlusion of Patent Ductus Arteriosus. Iranian journal of pediatrics, 26(3), e3621. https://doi.org/10.5812/ijp.3621

[2]. Amoozgar, H., Salehi, S., Farhadi, P., Edraki, M., Borzoee, M., Ajami, G., & Mohammadi, H. (2016). Follow-up results of device occlusion of patent ductus arteriosus. Iranian Journal of Pediatrics, In Press(In Press). https://doi.org/10.5812/ijp.3621

[3]. Amoozgar, H., Salehi, S., Farhadi, P., Edraki, M., Borzoee, M., Ajami, G., & Mohammadi, H. (2016). Follow-up results of device occlusion of patent ductus arteriosus. Iranian Journal of Pediatrics, In Press(In Press). https://doi.org/10.5812/ijp.3621

[4]. Atik-Ugan, S. and Saltik, İ. (2018). Transcatheter closure of ventricular septal defect with occlutech duct occluder. Cardiology in the Young, 28(4), 598-601. https://doi.org/10.1017/s1047951117002463

[5]. Baruteau, A., Hascoët, S., Baruteau, J., Boudjemline, Y., Lambert, V., Angel, C., & Pass, R. (2014). Transcatheter closure of patent ductus arteriosus: past, present and future. Archives of Cardiovascular Diseases, 107(2), 122-132. https://doi.org/10.1016/j.acvd.2014.01.008

[6]. Bilkis, A. A., Alwi, M., Hasri, S., Haifa, A. L., Geetha, K., Rehman, M. A., & Hasanah, I. (2001). The Amplatzer duct occluder: experience in 209 patients. Journal of the American College of Cardiology, 37(1), 258–261. https://doi.org/10.1016/s0735-1097(00)01094-9

[7]. Cassidy, Harvey & Cassidy, Lynsey & Blackshear, Joseph. (2009). Incidental Discovery of a Patent Ductus Arteriosus in Adults. Journal of the American Board of Family Medicine : JABFM. 22. 214-8. 10.3122/jabfm.2009.02.070230.

[8]. Gu, X., Zhang, Q., Sun, H., Fei, J., Zhang, X., & Kutryk, M. (2016). Transcatheter closure of calcified patent ductus arteriosus in older adult patients: immediate and 12-month follow-up results. Congenital Heart Disease, 12(3), 289-293. https://doi.org/10.1111/chd.12437

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAY987

[9]. Güvenç, O. (2016). Closure of wide patent ductus arteriosus using a fenestrated muscular vsd occluder device in a pediatric patient with down syndrome and pulmonary hypertension. Turk Kardiyoloji Dernegi Arsivi-Archives of the Turkish Society of Cardiology. https://doi.org/10.5543/tkda.2016.38283
[10]. Hildebrandt, N., Schneider, C., Schweigl, T., & Schneider, M. (2010). Long-term follow-up after transvenous single coil embolization of patent ductus arteriosus in dogs. Journal of Veterinary Internal Medicine, 24(6), 1400-1406. https://doi.org/10.1111/j.1939-1676.2010.0605.x
[11]. Hoyer, M. (2005). Novel use of the amplatzer plug for closure of a patent ductus arteriosus. Catheterization and Cardiovascular Interventions, 65(4), 577-580. https://doi.org/10.1002/ccd.20347
[12]. Ji, Q., Feng, J., Mei, Y., Wang, X., Cai, J., Sun, Y., & Yong-wu, W. (2008). Transcatheter closure of adult patent ductus arteriosus with severe pulmonary hypertension. Hypertension Research, 31(11), 1997-2002. https://doi.org/10.1291/hypres.31.1997
[13]. Ji, Q., Feng, J., Mei, Y., Wang, X., Cai, J., Sun, Y., & Yong-wu, W. (2008). Transcatheter closure of adult patent ductus arteriosus with severe pulmonary hypertension. Hypertension Research, 31(11), 1997-2002. https://doi.org/10.1291/hypres.31.1997
[14]. Ji, Q., Feng, J., Mei, Y., Wang, X., Cai, J., Sun, Y., Zhou, Y., Li, D., & Wang, Y. (2008). Transcatheter closure of adult patent ductus arteriosus with severe pulmonary hypertension. Hypertension research : official journal of the Japanese Society of Hypertension, 31(11), 1997–2002. https://doi.org/10.1291/hypres.31.1997
[15]. K. Elissa, “Title of paper if known,” unpublished.
[16]. Kheiwa, A., Ross, R., & Kobayashi, D. (2016). Reversal of severe mitral regurgitation by device closure of a large patent ductus arteriosus in a premature infant. Cardiology in the Young, 27(1), 189-192. https://doi.org/10.1017/s1047951116000998
[17]. Kishi, K., Jarutach, J., Ooi, Y., Wong, N., Mood, M., Geetha, K., & Leong, M. (2022). High haemoglobin levels at the time of occlusion predict worse outcome for patients with patent ductus arteriosus and pulmonary hypertension. Cardiology in the Young, 33(8), 1359-1366. https://doi.org/10.1017/s104795112200244x
[18]. Köksal, N., Aygün, C., & Uraş, N. (2019). Turkish neonatal society guideline on the management of patent ductus arteriosus in preterm infants. Türk Pediatri Arşivi, 53(sup1), 76-87. https://doi.org/10.5152/turkpediatriars.2018.01808
[19]. Ng, B., Schneider, D., & Hokanson, J. (2011). Closure of tubular patent ductus arteriosus in infants and small children with the amplatzer vascular plug ii. Congenital Heart Disease, 6(1), 64-69. https://doi.org/10.1111/j.1747-0803.2010.00466.x
[20]. Niu, M., Mallory, G., Justino, H., Ruiz, F., & Petit, C. (2013). Treatment of severe pulmonary hypertension in the setting of the large patent ductus arteriosus. Pediatrics, 131(5), e1643-e1649. https://doi.org/10.1542/peds.2011-1417
[21]. Putra, S. T., Djer, M. M., Idris, N. S., & Sastroasmoro, S. (2016). Transcatheter Closure of Patent Ductus Arteriosus in Adolescents and Adults: A Case Series. Acta medica Indonesiana, 48(4), 314–319.
[22]. Sallmon, H., Timme, N., Atasay, B., Erdeve, Ö., Hansmann, G., Singh, Y., & Shelton, E. (2021). Current controversy on platelets and patent ductus arteriosus closure in preterm infants. Frontiers in Pediatrics, 9. https://doi.org/10.3389/fped.2021.612242
[23]. Sallmon, H., Weber, S., Dirks, J., Schiffer, T., Klippstein, T., Stein, A., & Koehne, P. (2018). Association between platelet counts before and during pharmacological therapy for patent ductus arteriosus and treatment failure in preterm infants. Frontiers in Pediatrics, 6. https://doi.org/10.3389/fped.2018.00041
[24]. Sathanandam, S., Gutfinger, D., Morray, B., Berman, D., Gillespie, M., Forbes, T., & Zahn, E. (2021). Consensus guidelines for the prevention and management of periprocedural complications of transcatheter patent ductus arteriosus closure with the amplatzer piccolo occluder in extremely low birth weight infants. Pediatric Cardiology, 42(6), 1258-1274. https://doi.org/10.1007/s00246-021-02665-3
[25]. Schneider, D. and Moore, J. (2006). Patent ductus arteriosus. Circulation, 114(17), 1873-1882. https://doi.org/10.1161/circulationaha.105.592063
[26]. Terek, D., Yalaz, M., Ülger, Z., Köroğlu, Ö., & Kültürsay, N. (2018). Patent ductus arteriosus in preterm infants born before 30 weeks’ gestation: high rate of spontaneous closure after hospital discharge. Cardiology in the Young, 28(8), 995-1000. https://doi.org/10.1017/s1047951118000641
[27]. Vijayalakshmi, I., Setty, N., Narasimhan, C., Singla, V., & Manjunath, C. (2014). Percutaneous device closure of patent ductus arteriosus with pulmonary artery hypertension: long-term results. Journal of Interventional Cardiology, 27(6), 563-569. https://doi.org/10.1111/joic.12156
[28]. Wang, Z., Wang, Q., & Xianyang, Z. (2023). Iatrogenic or predetermined aortic dysplasia?. Cardiology in the Young, 33(8), 1456-1458. https://doi.org/10.1017/s104795112200419x
[29]. Yan, C., Zhao, S., Jiang, S., Xu, Z., Huang, L., Zheng, H., Ling, J., Wang, C., Wu, W., Hu, H., Zhang, G., Ye, Z., & Wang, H. (2007). Transcatheter closure of patent ductus arteriosus with severe pulmonary arterial hypertension in adults. Heart (British Cardiac Society), 93(4), 514–518. https://doi.org/10.1136/hrt.2006.091215
[30]. Zhang, D. Z., Zhu, X. Y., Lv, B., Cui, C. S., Han, X. M., Sheng, X. T., Wang, Q. G., & Zhang, P. (2014). Trial occlusion to assess the risk of persistent pulmonary arterial hypertension after closure of a large patent ductus arteriosus in adolescents and adults with elevated pulmonary artery pressure. Circulation. Cardiovascular interventions, 7(4), 473–481. https://doi.org/10.1161/CIRCINTERVENTIONS.113.001135