Original Article

Transcatheter Closure of Large Tubular Hypertensive Ductus Arteriosus: Challenges and Solutions

Hussein Abdulzahraa Alsalkhi[1], Mohanad K. Shukur Al Ghanimi[2], Dena R. Alhillawy[3]

[1]Department of Pediatric Cardiology, College of Medicine, Kufa University, Najaf, Iraq, [2]Department of Pediatric Cardiology, College of Medicine, Babylon University, Babylon, Iraq, [3]Department of Family Medicine, Najaf Health Directorate, Najaf, Iraq

Abstract

Background: While intravascular hemolysis is rare after transcatheter closure of small and moderate patent ductus arteriosus (PDA), large and tubular hypertensive ducts have a distinct morphological and physiological entity that makes complete closure challenging. **Objectives:** We aimed to evaluate the advantages of using a double disk device in the closure of a large tubular hypertensive PDA. **Materials and Methods:** During the period from January 1, 2018, to January 1, 2023, 25 (5.9%) of 420 patients who underwent transcatheter closure of PDA at Al-Najaf Cardiac Center, Najaf, Iraq, were found to have large nonrestrictive tubular PDA with severe pulmonary arterial (PA) pressure and thus enrolled in this study. **Results:** The mean of patients' ages and weights were 14.6 years and 36 kg, respectively. Pre-closure mean and peak PA pressures were 50 mmHg and 95 mmHg, respectively. Post-closure mean and peak PA pressure were 30 mmHg and 55 mmHg, respectively. Immediate successful closure was achieved in 24 patients (96%). Amplatzer Muscular Occluder and Amplatzer Septal Occluder were successfully used in 17 patients with no, trivial, or mild residual shunt and without development of intravascular hemolysis during the follow-up period. **Conclusion:** The risk of intravascular hemolysis can be avoided when double-disk devices are used to close large tubular hypertensive PDA.

Keywords: Double-disk devices, ductus arteriosus, intravascular hemolysis, severe pulmonary hypertension

Introduction

Patent ductus arteriosus (PDA) is one of the most common congenital heart defects. It accounts for 6%–11% of congenital defects in children and is twice as common in female compared to male patients.[1,2] Most PDA cases are diagnosed and managed in childhood, and the first presentation in adulthood is infrequent.[3,4]

Although small-sized PDA may not cause significant symptoms until discovered accidentally, larger-sized PDA often causes shortness of breath, exercise intolerance, and palpitations. Untreated large PDA in late adolescence and adulthood carries a high risk of congestive heart failure, pulmonary hypertension, pulmonary vascular obstructive disease, and even death. PDA can be complicated by bacterial endocarditis, aneurysm formation, and ductal wall calcification.[3-6]

The most popular and accepted classification of PDA was described by Krichenko *et al.* Figure 1: Type A: conical ductus with a prominent aortic ampulla and constriction at the pulmonary artery end. Type B: window-like (wide and short) ductus. Type C: tubular ductus without constrictions. Type D: complex ductus with multiple constrictions. Type E: bizarre configuration with an elongated, conical ductus with narrowing as far from the anterior border of the trachea as possible (via lateral angiography).[7,8]

With significant advances in the device's technology, transcatheter closure of virtually all types of PDA has

Address for correspondence: Dr. Hussein A. Alsalkhi, Department of Pediatric Cardiology, College of Medicine, Kufa University, Najaf, Iraq.
E-mail: husseina.abdulridha@uokufa.edu.iq

Submission: 18-Mar-2024 **Accepted:** 22-Oct-2024 **Published:** 23-Jul-2025

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

How to cite this article: Alsalkhi HA, Al Ghanimi MKS, Alhillawy DR. Transcatheter closure of large tubular hypertensive ductus arteriosus: Challenges and solutions. Med J Babylon 2025;22:S8-13.

Access this article online

Quick Response Code:

Website: https://journals.lww.com/mjby

DOI: 10.4103/MJBL.MJBL_44_24

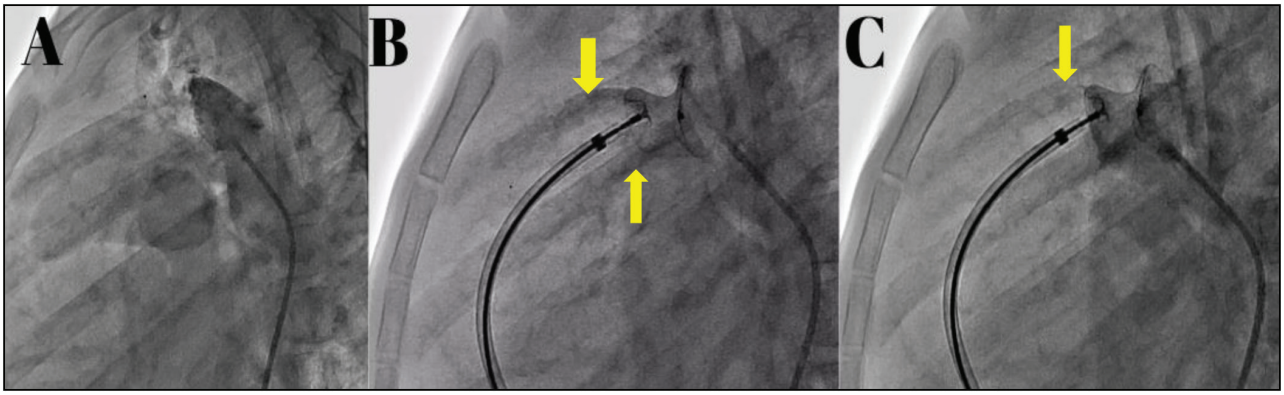

Figure 1: (A) Aortogram in lateral (90°) projection of a 10-year-old female patient demonstrates a large patent ductus arteriosus (PDA) without constriction. (B) Aortogram in lateral (90°) projection immediately after closure of PDA with Amplatzer Ductal Occluder (16:14 mm) demonstrates significant residual shunt between the device and the ductal walls (arrows). (C) Aortogram in lateral (90°) projection, 30 min after closure demonstrates good device position with significant decrement of the residual shunt (arrow). This patient developed severe intravascular hemolysis 18 h post-closure, which failed to respond to medical treatment, and the patient was referred for surgery

become the treatment of choice in most cardiac care centers. However, transcatheter closure of large tubular PDA in all age groups remains a technical challenge.[9,10] PDA with severe pulmonary arterial (PA) hypertension presents multiple challenges for cardiologists. Evaluation of pulmonary vascular bed reversibility and achievement of complete transcatheter device closure without residual shunt are definitive goals. Surgical closure of large non-constricting PDA is not free of risk due to known pathophysiological and hemodynamic complications.[11-13]

Intravascular hemolysis caused by residual shunt post transcatheter closure is rare, but persistent severe intravascular hemolysis may be associated with significant hemodynamic challenges such as coagulopathy and renal failure.[5,14] In this article, we report our experience with transcatheter closure of large tubular PDA with severe PA hypertension using single- versus double-disk occluders.

We evaluated the potential advantages of using double-disk devices in order to avoid intravascular hemolysis after transcatheter closure of large tubular PDA with severe PA pressure in older children, adolescents, and adults.

Materials and Methods

Patients and study design

From January 1, 2018, to January 1, 2023, 25 (5.9%) of 420 patients who underwent transcatheter closure of PDA at Al-Najaf Cardiac Center, Najaf, Iraq, were found to have large tubular PDA with severe PA pressure and enrolled in this study.

Pre-catheterization data like age, sex, weight, upper and lower limb oxygen saturation at rest and after exercise, chest X-ray, 12-lead ECG, and detailed transthoracic echocardiographic (TTE) study were recorded for each patient. Patients' demographic and hemodynamic data are shown in [Table 1].

TTE evaluation included the following: ventricular function and dimension, PA pressure (from tricuspid regurgitation velocity), size of the ductus, and associated cardiac lesions.

Patients included in this study were older children, adolescents, and adults with large tubular PDA (predominant left to right shunt, LV volume overload) and reactive pulmonary vascular bed (reversible pulmonary hypertension). Patients with irreversible pulmonary hypertension, constricted non-tubular PDA, and those requiring surgical interventions for associated cardiac lesions were excluded. Patients with pulmonary to systemic arterial pressure >60% 30 min post-closure (balloon or device) of the PDA were excluded, the device was removed, and they were referred for further evaluation. Procedures were performed under local anesthesia or deep sedation and TTE guidance.

Prophylactic antibiotics with ceftriaxone (50 mg/kg) were administered intravenously at the time of vascular access, followed by cefixime (10 mg/kg/day) orally for 3 days. All patients were kept on oral sildenafil citrate and/or bosentan for at least 6 months after the procedure.

A study of right and left heart catheterization was performed to evaluate pulmonary and systemic pressures, the magnitude of the left to right shunt, and pulmonary vascular resistance.

Aortograms in the right anterior oblique (30°) and lateral (90°) views were obtained, and measurements of length, aortic end, and pulmonary end of the PDA were done using the catheter diameter as the reference.

The same protocol for transcatheter PDA closure, as described by other authors, was followed. The devices

Table 1: Patient's demographic and hemodynamic data

Pt.	Age	Sex	Wt.	SPO2%		Systolic PAP		Systolic AOP		Qp/Qs	PVR	PAP at Follow-up*
				Pre	Post	Pre	Post	Pre	Post			
1	10	F	22	97	98	75	45	115	115	2.1	3.5	22
2	12	F	34	96	97	68	37	120	115	3	2	27
3	11	F	29	99	99	70	35	110	120	2.2	1.8	21
4	15	M	58	96	98	65	33	95	105	2.5	2	25
5	13	M	60	91	96	83	55	125	130	1.9	3	40
6	18	F	72	97	100	76	42	105	110	2	2.4	20
7	11	F	50	95	99	70	40	115	128	2.3	2.8	23
8	13	F	59	96	100	78	45	120	129	2	3.7	29
9	29	F	77	94	97	89	52	144	152	1.6	4.7	50
10	13	M	48	95	100	67	31	119	128	2.6	1.9	19
11	13	M	40	95	98	73	44	115	123	3.1	2	17
12	20	F	71	94	99	80	35	125	120	3	2.8	30
13	21	F	61	100	100	62	29	123	131	2.1	2.9	21
14	16	F	59	99	99	74	30	125	135	1.8	3.1	16
15	14	F	63	98	100	60	25	109	127	2.2	1.4	15
16	11	M	52	94	96	77	38	94	100	2.1	3.8	19
17	19	M	53	99	100	70	30	128	133	2	1	27
18	27	M	62	96	97	79	41	138	146	2.2	3.9	40
19	17	F	67	92	98	90	55	140	143	1.9	5.1	55
20	25	M	51	98	98	65	30	130	128	3.7	1.6	18
21	10	F	35	96	100	72	38	128	132	2.7	2.9	20
22	12	F	30	97	100	78	35	121	129	2.6	2.4	17
23	14	F	52	99	100	73	30	128	140	3.4	2.3	22
24	11	M	43	95	98	91	43	123	127	1.8	4.6	32
25	26	F	70	95	97	85	45	125	125	1.9	3.2	15
Mean	16		52.7	96	98.5	74.8	38.5	120	127	2.3	2.8	25.6
±SD	5.7		14.7	2.2	1.4	8.4	8.2	12.1	12	0.5	1	10.5

Pt. = patient, Wt. = weight, PPAP = peak pulmonary arterial pressure, PAOP = peak aortic pressure, PVR = pulmonary vascular resistance, pre, post = pre and post-PDA closure, * = peak pulmonary arterial pressure as measured by TTE from tricuspid regurgitation velocity

used for closure of the PDA were Amplatzer Ductal Occluder Type I (AGA Medical Corp., Plymouth, MN, USA), Amplatzer Muscular Occluder (AGA Medical Corp.), and Amplatzer Septal Occluder (ASO; AGA Medical Corp.).

The device chosen must be at least 5 mm larger than the diameter of the narrowest PDA, as long as it resulted in closure of PDA with no more than a mild residual shunt (successful closure).

All patients were admitted to the cardiac follow-up unit for overnight observation. Admission for more than 24 h was not required, except for those complicated by intravascular hemolysis. Each patient was evaluated clinically and by TTE at the time of hospital discharge and 1, 3, and 6 months post-closure and then annually for evidence of intravascular hemolysis, device position, residual shunt, ventricular function, and PA pressure.

The mean of the follow-up period was 29.52 months (12–52 months). At each visit, detailed clinical history was obtained and thorough physical examination done, including monitoring of oxygen saturation at upper and lower limbs and at rest and exercise with transthoracic echocardiographic study looking for ventricular function, device position, residual shunt, and degree of PA pressure.

Ethical approval

This study was approved by the Najaf Health directorate. Patients were informed about the procedures in the study, and verbal consent was obtained from all patients for enrollment. According to document number 598, a local ethics commission reviewed and gave its approval to the study protocol, subject information, and permission form on August 16, 2022.

Statistical analysis

Statistical analysis was carried out using SPSS version 27. Categorical variables were presented as frequencies and percentages. Continuous variables were presented as (means ± SD). Pearson's chi-square test was used to find the association between categorical variables. A P value of ≤ 0.05 was considered significant.

Results

Twenty-five (5.9%) of 420 patients with PDA met the inclusion criteria and were included in this study. In terms of patient's age and weight, the mean and range were 14.6 years (10–29 years) and 36 kg (26–74 kg), respectively. Pre-closure mean and peak PA pressures were 50 mmHg and 95 mmHg, respectively. PDA pulmonary end diameters (the narrowest parts) ranged from 9 to 15 mm, and their lengths ranged from 12 to 27 mm.

Post-closure mean and peak PA pressure were 30 mmHg and 55 mmHg, respectively. The means of fluoroscopic time and procedure duration were 17.8 (12–22) min and 73.1 (110–45) min, respectively. Immediate successful closure was achieved in 24 patients (96%), while immediate complete closure was found in 40% of patients.

Amplatzer Ductal Occluder (size 16:14 mm) was used in seven patients, Figure 1. In two patients, the devices embolized into pulmonary arteries were retrieved and replaced by Amplatzer Muscular occluders with no residual shunt. Of those with successful closure, four patients had a mild residual shunt post-closure. Intravascular hemolysis that developed in four patients with residual shunt within the first 24 h was resolved, with conservative management (intravenous hydration and alkalinization of the urine) in two patients and required surgical intervention and ligation of the PDA, due to persistent severe intravascular hemolysis, severe anemia, and increased renal function indices in the other two patients.

Amplatzer Muscular Occluder (sizes 16, 18, and 20 mm), Figures 2 and 3 was used in 14 patients with no residual shunt in 10 patients and trivial or mild residual shunt in four patients, which disappeared completely, without the development of intravascular hemolysis during the follow-up period.

ASO (two devices, each sized 24 and 26 mm) were used in three patients, with a trivial–mild residual shunt in two of them that resolved spontaneously a few days post-closure without the development of clinically detectable hemolysis.

Failure of the closure of the PDA was observed in a 25-year-old male patient with a large tubular PDA and mild coarctation of the aorta due to a persistent moderate residual shunt 30 min after deployment of an ASO, size 26 mm, so the device was retrieved and the patient referred for surgery. No device embolization, pulmonary branch stenosis, or aortic coarctation was reported during the postoperative and follow-up periods. Mild left ventricular dysfunction developed in two patients post-PDA closure and was treated conservatively.

At 6-month follow-up, only two patients were found to have moderate pulmonary hypertension with peak PA pressure of 55 and 50 mmHg, as estimated from TTE measurement of tricuspid valve regurgitation velocity. At the last follow-up, all patients showed a reduction in PA pressure, improvement in left ventricular dilation and function, and complete closure with no evidence of residual shunt or clinically detectable hemolysis.

Discussion

Transcatheter closure of PDA is a well-established procedure with favorable outcomes and few adverse effects.[3,6,15] Large non-constricting PDA and simultaneous severe pulmonary hypertension is an anatomical and physiological entity that presents substantial challenges in transcatheter closure.[3,4,14,15]

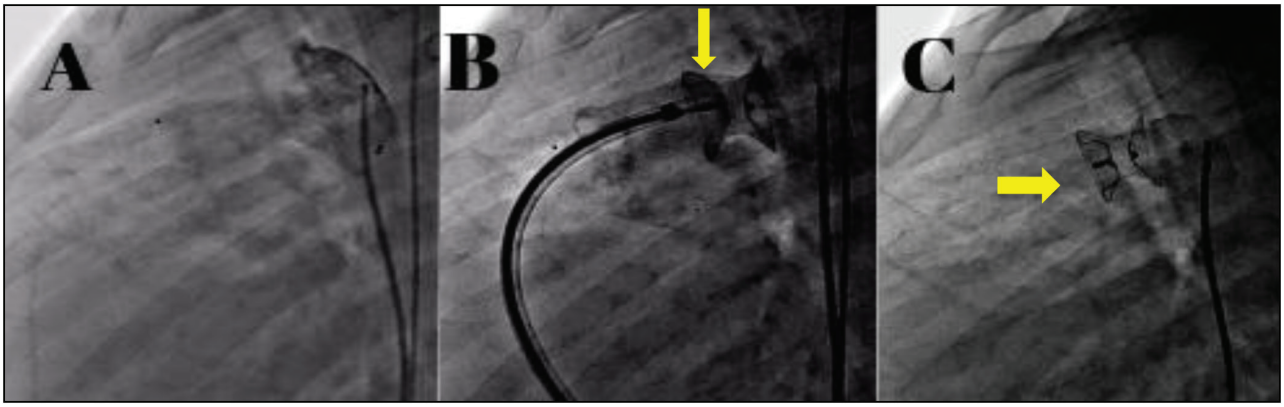

Figure 2: (A) Aortogram in lateral (90°) projection found in a 12-year-old female patient demonstrates a large tubular patent ductus arteriosus (PDA) without significant constriction. The peak and mean pulmonary arterial pressure were 80 and 50 mmHg, respectively. (B) Aortogram in lateral (90°) projection immediately after closure of large PDA with Amplatzer Muscular Occluder (18 mm) demonstrates significant residual shunt through the device and between the device and the ductal wall (arrow). (C) Aortogram in lateral (90°) projection, 20 min after closure, demonstrates good device position with mild residual shunt mostly through the device (arrow)

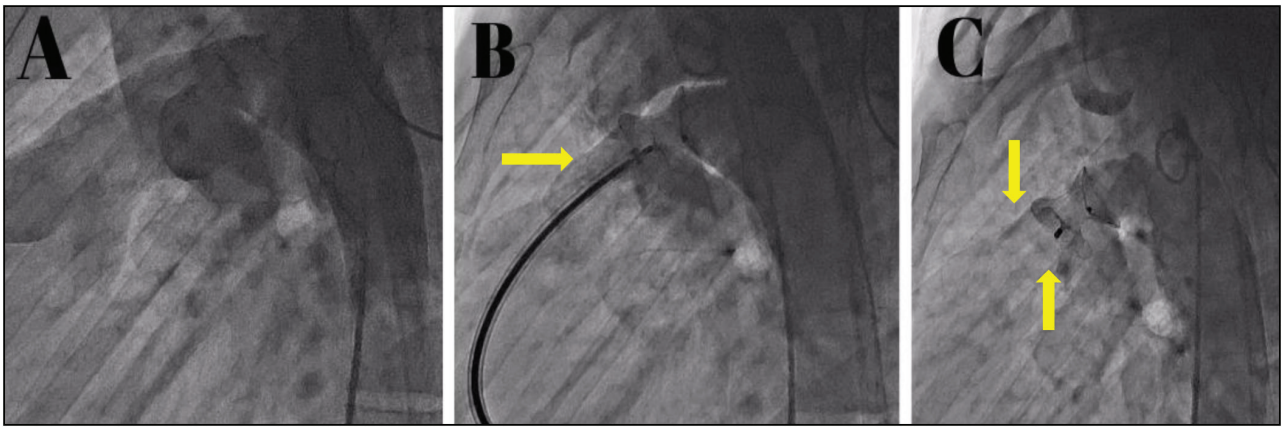

Figure 3: (A) Aortogram in lateral (90°) projection of an 18-year-old male patient demonstrates a large patent ductus arteriosus (PDA) without significant constriction. (B) Aortogram in lateral (90°) projection immediately after closure of large PDA with Amplatzer Muscular Occluder (20 mm) demonstrates good device position and significant residual shunt through the device (arrow). (C) Aortogram in lateral (90°) projection, 10 min after closure, demonstrates good device position with less than mild residual shunt (arrows) that disappeared on follow-up without clinically detectable hemolysis

We assume that longstanding large left to right ductal shunt and progressive PA hypertension change the anatomical and physiological features of the ductus.

We report two observations during transcatheter closure of large non-constricting hypertensive ducts. The first is the expandable character of the ductus arteriosus; for example, a duct diameter that normally would call for an ADO-I of 12:10 mm could accept a device of 16:14 mm without creating a noticeable waist, which makes it a wonder that the recommendation for a proper device size in such a PDA should differ from that of the usual ones. The second observation is that such a large non-constricting hypertensive ductus takes a long time to properly grab the device, in other words, the device needs a long time to adhere to the ductal wall, which was identified by the dye opacification between the device and the ductal wall during the post-closure aortogram, and it was more noticeable with low-profile devices like ADO-I [Figures 1B and 2B].

Although complete closure (without residual shunt) is commonly achieved in constricting PDA,[16] this is not always possible or may require a longer time with large non-constricting PDA (needs up to 30 min in some cases).[9,11,17]

Elimination of the residual shunt is mandatory to avoid the risk of post-closure intravascular hemolysis, which may be severe and continuous, resulting in significant adverse events (such as coagulopathy and renal failure).[4,10,18] Mild hemolysis may be efficiently treated conservatively with hydration, alkaline diuresis, haptoglobin, antiplasmin, and correction of anemia (blood transfusion, iron, and folate replacement), but residual shunt should be eliminated invasively by catheterization or by surgery.[5,10,14,19,20] In

patients with large PDA and severe pulmonary hypertension, every effort should be made to achieve complete closure and avoid serious potential sequelae of the residual shunt.

In this study, we found that double-disk devices like the Amplatzer Muscular Occluder (the most frequently used device) are effective in closing large non-constricting (tubular) PDAs with complete closure or only a trivial residual shunt that vanishes completely without any reported intravascular hemolysis events during follow-up.

The harder profile of the Amplatzer Muscular Occluders (in comparison to the Amplatzer ductal occluders) and the presence of two discs are probably the reasons behind the absence of significant post-closure intravascular hemolysis.

Although two patients had undergone device embolization shortly after discharge, no device embolization was recorded at the post-catheterization follow-up period. Mild left ventricular dysfunction that developed in two patients post-PDA closure was resolved by conservative treatment. The same finding had been reported by other authors.[21,22]

In our study, we followed strict rules to avoid closure of large PDA with irreversible PA hypertension. Only two patients were found to have a moderate degree of pulmonary hypertension during the follow-up period, and both had positive treatment outcomes with target pulmonary hypertension treatment. Rebound or persistent pulmonary hypertension was reported even after initial apparently successful closing procedures.[23-25]

Conclusion

We conclude that the use of double-disk devices in the closure of large tubular PDA and severe pulmonary hypertension is associated with avoiding the risk of post-closure intravascular hemolysis and its adverse effects.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Schneider DJ. The patent ductus arteriosus in term infants, children, and adults. In: Seminars in Perinatology. Vol. 36, No. 2. WB Saunders; 2012. p. 146-153.
2. Forsey JT, Elmasry OA, Martin RP. Patent arterial duct. Orphanet J Rare Dis 2009;4:1-9.
3. Putra ST, Djer MM, Idris NS, Sastroasmoro S. Transcatheter closure of patent ductus arteriosus in adolescents and adults: A case series. Acta Med Indones 2016;48:314-9.
4. Khattak KK, Ullah M, Sheikh AM, Kanwal, A, Shah SA, *et al*. Managementof hypertensive PDA. Pak Armed Forces Med J 2021;70:S701-05.
5. Kanabar K, Bootla D, Kaur N, Pruthvi CR, Krishnappa D, Santosh K, *et al*. Outcomes of transcatheter closure of patent ductus arteriosus with the off-label use of large occluders (≥16 mm). Indian Heart J 2020;72:107-12.
6. Jang GY, Son CS, Lee JW, Lee JY, Kim SJ. Complications after transcatheter closure of patent ductus arteriosus. J Korean Med Sci 2007;22:484-90.
7. Krichenko A, Benson LN, Burrows P, Möes CA, McLaughlin P, Freedom RM. Angiographic classification of the isolated, persistently patent ductus arteriosus and implications for percutaneous catheter occlusion. Am J Cardiol 1989;63:877-80.
8. Habib NM, Shafie OM, Soliman NMS. Percutaneous closure of patent ductus arteriosus; implications of the Krichenko classification. Al-Azhar Journal of Ped 2023;26. doi:10.21608/azjp.2023.287919.
9. Yan C, Zhao S, Jiang S, Xu Z, Huang L, Zheng H, *et al*. Transcatheter closure of patent ductus arteriosus with severe pulmonary arterial hypertension in adults. Heart 2007;93:514-8.
10. Zabal C, García-Montes JA, Buendía-Hernández A, Calderón-Colmenero J, Patiño-Bahena E, Juanico-Enriquez A, *et al*. Percutaneous closure of hypertensive ductus arteriosus. Heart 2010;96:625-9.
11. Zhang DZ, Zhu XY, Lv B, Cui CS, Han XM, Sheng XT, *et al*. 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. Circ Cardiovasc Interv 2014;7:473-81.
12. Rigby ML. Closure of a large patent ductus arteriosus in adults: first do no harm. Heart 2007;93:417-8.
13. Kalavrouziotis G, Kourtesis A, Paphitis C, Azariades P. Closure of a large patent ductus arteriosus in children and adults with pulmonary hypertension. Hellenic J Cardiol 2010;51:15-8.
14. Anil SR, Sivakumar K, Philip AK, Francis E, Kumar RK. Clinical course and management strategies for hemolysis after transcatheter closure of patent arterial ducts. Catheter Cardiovasc interv 2003;59:538-43.
15. Al Ghanimi MKS, Alsalkhi HA, Alkhafaji MF. A transcatheter closure of large-size patent ductus arteriosus with severe degree of pulmonary hypertension by different types of devices. Iran J Neonatol 2023;14:21-7.
16. Bilkis AA, Alwi M, Hasri S, Haifa AL, Geetha K, Rehman MA, *et al*. The Amplatzer duct occluder: Experience in 209 patients. J Am Coll Cardiol 2001;37:258-61.
17. García-Montes JA, Camacho-Castro A, Sandoval-Jones JP, Buendía-Hernández A, Calderón-Colmenero J, Patiño-Bahena E, *et al*. Closure of large patent ductus arteriosus using the Amplatzer Septal Occluder. Cardiol Young 2015;25:491-5.
18. Ji Q, Feng J, Mei Y, Wang X, Cai J, Sun Y, *et al*. Transcatheter closure of adult patent ductus arteriosus with severe pulmonary hypertension. Hypertens Res 2008;31:1997-2002.
19. Rashid A, Lone A, Rather H, Hafeez I. Management of PDA device closure complicated by severe hemolysis by transcatheter retrieval and deployment of new device. Egypt Heart J 2023;75:17.
20. Joseph G, Mandalay A, Zacharias TU, George B. Severe intravascular hemolysis after transcatheter closure of a large patent ductus arteriosus using the Amplatzer duct occluder: Successful resolution by intradevice coil deployment. Catheter Cardiovasc Interv 2002;55:245-9.
21. Jeong YH, Yun TJ, Song JM, Park JJ, Seo DM, Koh JK, *et al*. Left ventricular remodeling and change of systolic function after closure of patent ductus arteriosus in adults: Device and surgical closure. Am Heart J 2007;154:436-40.
22. Zhang CJ, Huang YG, Huang XS, Huang T, Huang WH, Xia CL, *et al*. Transcatheter closure of large patent ductus arteriosus with severe pulmonary arterial hypertension in adults: Immediate and two-year follow-up results. Chin Med J (Engl) 2012;125:3844-50.
23. Feng J, Kong X, Sheng Y, Yang R. Patent ductus arteriosus with persistent pulmonary artery hypertension after transcatheter closure. The Clin Risk Manag 2016;12:1609-13.
24. Xu J, Wang L, Shen Y, Geng L, Chen F. Transcatheter closure for patent ductus arteriosus in patients with Eisenmenger syndrome: to do or not? BMC Cardiovasc Disord 2020;20:1-8.
25. Al-Murib AHA, Al-Jumaily HS, Hassan ZF, Al-Saad SF. Left atrial strain by 2D speckle tracking echocardiography in patients with systemic hypertension in Babylon province. Med J Babylon 2024;21:485-90.