Transcatheter Closure of a Patent Ductus Arteriosus with

Healed Vegetation: A Case Report

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ABSTRACT

Background: The unrepaired patent ductus arteriosus (PDA) is at risk for congestive heart failure (CHF) and/or infective endocarditis (IE). Transcatheter closure of PDAs with occluder devices has been advanced to be the strategy of choice for managing anatomically feasible PDAs. Despite it is not clear if the closure of a small PDA is beneficial, routine closure of any PDA in children and young adults appeared reasonable to decrease the risk of IE.

Case report: We report on a one-year and two-month-old boy who had a small Krichenko type D PDA, with two constrictions at its middle part and its pulmonary end, and with healed vegetation within the PDA. We successfully closed the PDA percutaneously using a 5x4 mm Nit-Occlud® PDA coil (PFM medical, Köln, Germany) with an unremarkable 2-year follow-up. To our knowledge, this is the first reported case of a PDA with a previous IE and healed vegetation within the duct that was closed by the transcatheter approach.

Conclusion: Closure of PDAs is indicated in patients with left ventricular overload or with continuous murmurs. Despite it is not clear if the closure of small PDAs is beneficial, routine closure of any PDA in children and young adults appeared reasonable to decrease the risk of IE. A history of previous IE makes PDA closure mandatory. Differentiation between IE recurrence and the persistence of healed vegetation from a previously treated IE can be difficult.

Keywords: Patent Ductus Arteriosus, Transcatheter Closure, Infective Endocarditis, Congestive Heart Failure, Tanta University, Healed Vegetation.

INTRODUCTION

The unrepaired patent ductus arteriosus (PDA) is at risk for congestive heart failure (CHF) and/or infective endocarditis (IE). Since transcatheter closure of PDA with occluder devices started in 1967^[1], the technique and the devices used have been advanced to make transcatheter PDA closure the strategy of choice for managing anatomically feasible PDA. In pediatrics, although transcatheter closure of PDA is generally a simple procedure, some challenges are still facing the interventionist; low body weights, venous anomalies, complex duct configurations, and complicated ducts with IE ^[1].

Here we report on a one-year and two-month-old boy who had a small Krichenko type D PDA with healed vegetation within the PDA from a previous attack of IE. We successfully closed the PDA percutaneously using a 5x4 mm Nit-Occlud® PDA coil (PFM medical, Köln, Germany). To our knowledge, this is the first reported case of a PDA with a previous IE and healed vegetation within the duct that was closed by the transcatheter approach.

CASE REPORT

Case description:

Here we report on a one-year and two-month-old boy, weighing 8 Kg, with a body surface area (BSA) of 0.74, who presented with dyspnea grade II-III, repeated attacks of chest infection, and with delayed milestones. At the age of 10 months, the patient's history denoted a small PDA with an attack of infective endocarditis with positive blood cultures for Streptococcus Viridans, which was managed by hospitalization and administration of high doses of intravenous (IV) antibiotics [Ceftriaxone 200 mg/kg/day for 4 weeks and gentamycin 5 mg/kg/day for two weeks]. Then, the patient was discharged after control of the fever and resolving of the blood cultures.

The patient's cardiac examination revealed a grade 3/6 continuous murmur on the upper left sternal border. His Oxygen saturation (SaO₂) was 99%. He had mild hypochromic microcytic anemia with a hemoglobin (Hb) level of 10.2 gm/dl. All his other laboratory findings were within normal ranges. His chest X-ray showed a mildly increased cardiothoracic ratio with mildly increased pulmonary vascular markings. His electroencephalogram (EEG), brain magnetic resonance imaging (MRI), electromyography (EMG), and bilateral lower limbs' motor nerve conduction study were unremarkable.

Transthoracic echocardiography revealed persistence of the small PDA that measured 1.5 mm at its pulmonary end leaving left to right shunt with a peak systolic pressure gradient of 76 mmHg and a diastolic one of 45 mmHg, figure 1. The left ventricle (LV) was mildly dilated with a good systolic function [Ejection fraction (EF) of 68%]. Also, there was mild mitral valve regurgitation (MR).

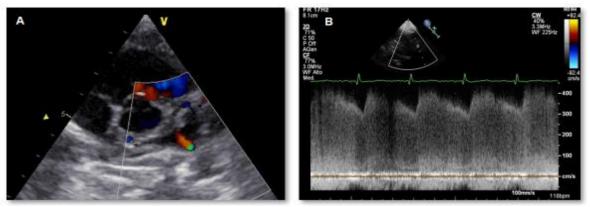


Figure (1): A: Transthoracic echocardiography in a short axis view showed a small patent ductus arteriosus (PDA) that measured 1.5 mm at its pulmonary end leaving left to right shunt. B: Continuous wave Doppler across the PDA showed a peak systolic pressure gradient of 76 mmHg and a diastolic one of 45 mmHg.

Firstly, the patient was admitted and underwent an invasive hemodynamic assessment that revealed normal rightand left-heart pressures; the aortic pressure was 82/51 mmHg, the systolic and mean pulmonary artery pressures (PAPs) were 21/11 mmHg, the pulmonary flow to the systemic flow ratio (QP/QS) was 1.63, and the pulmonary vascular resistance (PVR) was 1.3 Woods unit. There were no sudden alterations in SaO₂ levels at the main, right, or left pulmonary arteries (PAs). Aortography showed a small Krichenko type D duct, with an ampulla of 3 mm, a pulmonary end of 1.5 mm, and a total length of 6 mm. There were two constrictions in the PDA one at its middle part and the other at its pulmonary end with a filling defect/mass at the middle body part of the PDA in-between the 2 constrictions, figure 2.

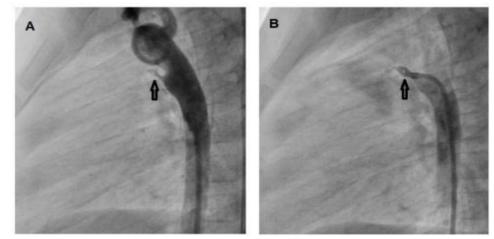


Figure (2): A: Aortography in the lateral projection using 5F Pigtail (PG) catheter and B: Selective aortography in the lateral projection using 5F Judkin right (JR) catheter. A & B showed a small Krichenko type D patent ductus arteriosus (PDA), with an ampulla of 3 mm, pulmonary end of 1.5 mm, and a total length of 6 mm. There were two constrictions in the PDA one at its middle part and the other at its pulmonary end with a filling defect/mass at the middle body part of the PDA in-between the 2 constrictions (Black arrows).

Three differential diagnoses regarding this filling defect/mass were suggested and included; vegetation of an actively recurred IE; a thrombus within the duct; or a healed vegetation of the previous attack of IE. After exclusion of recurrence of IE by the absence of fever, the insufficient Duke's criteria, and the negative aerobic and anaerobic blood cultures, a decision was taken to close this PDA with an occluder. The patient underwent successful transcatheter closure of this PDA with the included filling defect/mass under the cover of antibiotics therapy. Two 5F sheaths were introduced into the right common femoral vein and artery. The patient was given 800 Units of intravenous unfractionated heparin. A 5F Judkin right (JR) catheter was introduced retrogradely from the aorta on 0.018-inch/260 Terumo guidewire (Somerset, NJ, USA) to cross the duct and then to the PA. The wire was snared in the PA through a snare catheter using a 15-mm AmplatzerTM Gooseneck Snare (Abbott Vascular, IL, USA). After looping, a 5F implantation catheter was introduced antegrade from the venous aspect to cross the PDA to the aorta. As the duct was small, a 5x4 mm Nit-Occlud® PDA coil (PFM medical, Köln, Germany) was implanted to avoid any protrusion of the occluder in the descending thoracic aorta or the PA, to completely occlude the duct, and to include the healed vegetation within the coils of the occluder, figure (3). The procedure passed uneventfully and repeat aortography at the proximal descending thoracic aorta revealed no residual left-to-right ductal shunt or device encroachment on any of the cardiac structures.

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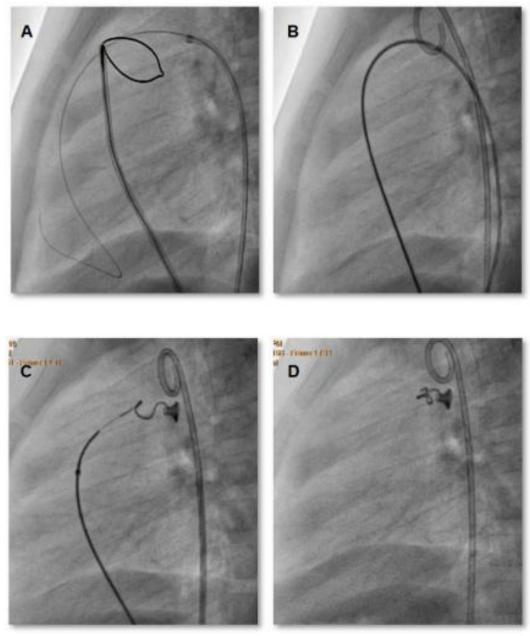


Figure (3): Transcatheter closure of the patent ductus arteriosus (PDA) with the healed vegetation A: A 5F Judkin right (JR) catheter was introduced retrogradely from the aorta on 0.018-inch/260 Terumo guidewire (Somerset, NJ, USA) to cross the duct and then to the pulmonary artery (PA). The wire was snared in the PA through a snare catheter using a 15-mm AmplatzerTM Gooseneck Snare (Abbott Vascular, IL, USA). B: Arterio-arterial looping. C: Antegrade implantation of a 5x4 mm Nit-Occlud® PDA coil (PFM medical, Köln, Germany) through a 5F implantation catheter. D: Release the coil to completely occlude the duct, and to include the healed vegetation within the coils of the occluder without any protrusion of the occluder in the descending thoracic aorta or the PA.

The patient was discharged on the next day of the procedure. Post-procedure, acetylsalicylic acid was prescribed at a dose of 5 mg/kg/day orally for 6 months. Follow-up of the patient at 3-month, 6-month, 1-year, and 2-year displayed no residual shunt. The parents recorded no other attacks of fever or chest infection.

Informed written consent was obtained from the guardians of the patient. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

DISCUSSION

Patent ductus arteriosus (PDA) accounts for 5-10% of all congenital heart disease (CHD), and was recorded in 0.3% to 0.8% of full-term infants with twice female to male ratio ^[2]. The clinical presentation is based on its size, the pulmonary to systemic flow ratio, and the systemic and pulmonary vascular resistances. The PDA shunt ranges from a small silent hemodynamically insignificant one that is not auscultated to a large hemodynamically significant shunt that causes congestive HF and pulmonary hypertension ^[3].

Here we report on a one-year and two-monthold boy who had a small PDA with an auscultated continuous murmur and who underwent transcatheter PDA closure with a Nit-Occlud® PDA coil. This PDA showed a significant hemodynamic effect; lung congestion, repeated chest infections, mild LV dilatation, and mild MR. According to the European Society of Cardiology guidelines 2020, closure should be considered in small PDAs with a continuous murmur, even with normal LV dimensions and normal PAP (IIa, C) and the device closure is the method of choice when technically suitable (I, C)^[4]. Even though it is still not clear to close a small PDA, routine closure of any PDA in children and young adults appeared reasonable. The reasons for this recommendation included the risk of infective endocarditis and the low morbidity from the transcatheter occlusion ^[5,6]. But, it is still debatable to close the isolated small PDA in childhood for the prevention of infective endarteritis alone^[7].

In our patient, there was a previously documented attack of IE and aortography revealed a small Krichenko type D PDA, with two constrictions at its middle part and its pulmonary end and with a filling defect/mass in-between the 2 constrictions. Three differential diagnoses regarding this filling defect/mass were suggested and included: vegetation of an actively recurred IE, a thrombus within the duct, or a healed vegetation of the previous attack of IE. Firstly, the mass might be vegetation from an active newly arose IE that is seated within the PDA. This was the most critical scenario and was excluded by the absence of fever, the insufficient Duke's criteria to diagnose an active IE, and the negative aerobic and anaerobic blood cultures. Secondly, the mass could be a thrombus within the PDA. Despite, the patient's delayed milestones raised the possibility of cerebral embolization/stroke from a thrombus within the PDA, this suggestion was excluded by the negative patient's cerebral imaging to detect any stroke and the pediatric neurological clearance of any

cerebral issues. Also, any embolization from such a mass will pass from the high-pressure aorta to the low-pressure PA to induce pulmonary embolism rather than systemic cerebral embolization/stroke. Mostly, the patient's delayed milestones were related to recurrent chest infections from the duct itself. Consequently, this mass was diagnosed as healed vegetation from the previous attack of IE that was seated within the duct middle part. The differentiation between IE recurrence and the persistence of healed vegetation from a previously treated IE can be difficult ^[8].

Nowadays, despite IE being a rare complication in patients with PDA, it was the commonest cause of death before the surgical PDA closure. The estimated incidence of IE complicating PDAs was 1% per year with a decrease in recent years related to improved dental care, the early routine closure of PDAs, and the use of antibiotics for IE prophylaxis ^[3]. But the unrepaired PDAs still carry the risk for IE as the turbulent blood flow through the aorta and the PA causes endothelial injury with the subsequent placing of pathogenic vegetations ^[3,9]. The clinical picture of an infected PDA could be subtle, and the diagnosis is mostly delayed. Symptoms may be attributed to pneumonia, pleurisy, or pulmonary infarction from pulmonary embolism. In most case reports on PDAs complicated by IE, the vegetations involved the pulmonary artery or the pulmonary valve and were resolved by surgical resection or antibiotics ^[10-12]. In one case report of a young woman with PDA complicated by IE, the vegetations were in both the pulmonary and the aortic walls with mycotic aneurysms of the descending aorta that were resolved by surgery ^[13]. In our patient, the vegetation was seated inside the PDA, this rare position could be explained by the presence of a constriction in the distal end of the PDA at its entry into the pulmonary artery as a trial of duct closure, that prohibited the vegetation to pass to the pulmonary artery side.

Despite, there is no strong recommendation for the closure of small or silent PDAs to prevent the initial attack of IE, a history of previous IE makes PDA closure mandatory. Like this case, the persistence of a healed vegetation after curing of a previous IE was previously reported by Vuille et al who followed the vegetations and found 29/41 vegetations persisted after the medical management. They concluded that despite successful medical management, the persistence of vegetation is still confirmed echocardiographically, and resolving of IE should be clinically guided rather than echocardiographically-guided ^[14]. Also, this concept was established by the ESC recommendations for the practice of echocardiography in IE and by the ESC for the management of infective Guidelines endocarditis. They stated the difficulty of interpreting an unchanged or reduced vegetation size with antibiotic therapy and considered the growth of vegetation and the increasing valvular regurgitation risk factors for

embolic events and poor prognosis ^[15,16]. Therefore, a follow-up of IE after antibiotic treatment should be guided mainly by the clinical course and the response to therapy and not by the change of the echocardiographic morphology of vegetations.

In most previously recorded patients with PDAs complicated with IE, the vegetations did not resolve with antibiotics and required surgical excision of the vegetation with concomitant PDA closure ^[8,9,13]. To our knowledge, this is the first reported case of a PDA that was previously complicated with IE in which the healed vegetation was seated within the duct and was successfully occluded by transcatheter closure using a 5x4 mm Nit-Occlud® PDA coil (PFM medical, Köln, Germany).

CONCLUSION

Closure of PDAs is indicated in patients with LV overload or with continuous murmurs. Despite it is not clear if the closure of small PDAs is beneficial, routine closure of any PDA in children and young adults appeared reasonable to decrease the risk of IE. A history of previous IE makes PDA closure mandatory. Differentiation between IE recurrence and the persistence of healed vegetation from a previously treated IE can be difficult. In most previously recorded patients with PDA and IE, the vegetations did not resolve with antibiotics and required surgical excision of the vegetation with concomitant PDA closure. To our knowledge, this is the first reported case of a PDA with a previous IE and healed vegetation within the duct that was closed by the transcatheter approach.

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REFERENCES

- 1. Portsmann W, Wierny L, Warnke H (1967): Closure of persistent ductus arteriosus without thoracotomy. Ger Med Mon., 12:259-261.
- Hoffman J, Kaplan S (2002): The incidence of congenital heart disease. J Am Coll Cardiol., 39:1890-1900.
- **3.** Schneider D, Moore J (2006): Patent ductus arteriosus. Below Circulation, 114:1873-1882.
- Baumgartner H, De Backer J, Babu-Narayan S et al. (2021): 2020 ESC Guidelines for the management of adult congenital heart disease: The Task Force for the

management of adult congenital heart disease of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Adult Congenital Heart Disease (ISACHD). European Heart Journal, 42(6,7):563-645.

- 5. Gilbert H, Patriozo L, Antunes M et al. (2015): ESC Guidelines for the management of infective endocarditis: The TaskForce for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). Eur Heart J., 36(44):3075-3128.
- 6. Fortescue E, Lock J, Galvin T *et al.* (2010): To close or not to close the very small patent ductus arteriosus. Congenit Heart Dis., 5:354-365.
- 7. Hakim K, Tagorti M, Msaad H *et al.* (2021): Transcatheter closure of silent patent ductus arteriosus for prevention of endocarditis is justified. Archives of Cardiovascular Disease, 13:297-334.
- 8. Ramiro V, Taquiso J, Obillos S *et al.* (2019): Pulmonary Artery and Pulmonic Valve Vegetations in a Young Pregnant Filipino with Patent Ductus Arteriosus. Case Rep Cardiol., 9: 1-4.
- 9. Choi K, Yang T, Park B *et al.* (2008): A case with patent ductus arteriosus complicated by pulmonary artery endarteritis. J Cardiovasc Ultrasound, 16:90-92.
- **10.** Yanýk A, Yetkin E, Lleri M *et al.* (2000): Vegetation due to Streptococcus viridans in the pulmonary artery in a child with patent ductus arteriosus. International Journal of Cardiology, 72(2)189-191.
- **11.** Sugimura Y, Katoh M, Toyama M (2013): Patent ductus arteriosus with pulmonary endarteritis. Internal Medicine, 52(18):2157-2158.
- 12. Cuenza L, Adiong A, Rondilla L (2014): Pulmonary endarteritis in a patient with patent ductus arteriosus and a bicuspid aortic valve. Philippine Journal of Internal Medicine, 52(4):1-3.
- **13. Bouhdadi H, Rhissassi J, Wazaren H** *et al.* (2022): Pulmonary and aortic endarteritis revealing a patent ductus arteriosus in an adult. Journal of Surgical Case Reports, 1:1-3.
- 14. Nidorf M, Weyman A, Picard M (1994): Natural history of vegetations during successful medical treatment of endocarditis. American Heart Journal, 128(6):1200-1209.
- **15. Habib G, Badano L, Tribouilloy C** *et al.* (2010): Recommendations for the practice of echocardiography in infective endocarditis. European Journal of Echocardiography, 11(2):202-219.
- **16.** Habib G, Lancellotti P, Antunes M *et al.* (2015): 2015 ESC Guidelines for the management of infective endocarditis: The Task Force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC) Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). European Heart Journal, 36(44):3075-3128.